BOSTON
MEDICAL
LIBRARY
6
G
77
VOL. 1

A

SYSTEM OF MEDICINE.

EDITED BY

J. RUSSELL REYNOLDS, M.D., F.R.S.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON;
FELLOW OF THE IMPERIAL LEOPOLD-CAROLINA ACADEMY OF GERMANY;
FELLOW OF UNIVERSITY COLLEGE, LOND.;
PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE IN UNIVERSITY COLLEGE;
PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL;
EXAMINER IN MEDICINE TO THE UNIVERSITY OF LONDON.

WITH NUMEROUS ADDITIONS AND ILLUSTRATIONS,

BY

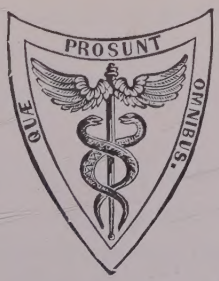
HENRY HARTSHORNE, A.M., M.D.,

FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; FORMERLY PROFESSOR OF PRACTICE OF MEDICINE IN MEDICAL DEPARTMENT OF PENNSYLVANIA COLLEGE, AND PHYSICIAN TO THE EPISCOPAL HOSPITAL OF PHILADELPHIA; LATELY PROFESSOR OF HYGIENE IN THE UNIVERSITY OF PENNSYLVANIA, AND PROFESSOR OF HYGIENE AND DISEASES OF CHILDREN IN THE WOMAN'S MEDICAL COLLEGE OF PENNSYLVANIA; ETC.

IN THREE VOLUMES.

VOL. I.

GENERAL DISEASES AND DISEASES OF THE NERVOUS SYSTEM.



PHILADELPHIA:
HENRY C. LEA'S SON & CO.
1880.

BOSTON MEDICAL LIBRARY
IN THE
FRANCIS A. COUNTWAY
LIBRARY OF MEDICINE

6
G
77

Entered according to Act of Congress, in the year 1879, by
HENRY C. LEA,
in the Office of the Librarian of Congress. All rights reserved.

COLLINS, PRINTER.

PREFACE TO THE AMERICAN EDITION.

Of the eminent authority of the contributors to this System of Medicine, and the excellence of their work, there can be but one opinion on either side of the Atlantic. If the present republication were made only in the interest of medical literature, in the historical sense, it would be an unwarrantable presumption to attempt any additions to it. But the purpose of such a work, cyclopædic in character, is, obviously, to render the greatest possible advantage to students and practitioners; for which end, it is desirable that the most important recent advances in clinical observation, pathology, diagnosis, and therapeutics should be supplied in connection with essays, some of which were written several years ago. This remark applies especially to the articles contained in the first Volume of the present edition, upon General Diseases and the Affections of the Nervous System. Moreover, the practical aspect of some subjects is different in this country from that which they present abroad, and the American physician may reasonably expect that the results of American experience should be concisely laid before him. So far as comments are made, and opinions occasionally expressed in this edition, more or less divergent from those of the authors of the work, it is, of course, at the option of every reader to estimate them at their proper value. If such comments appear at times obtrusive, the excuse of the American Editor is, his conscientious desire, sustained by the request of the Publisher, that he should omit nothing which seemed likely to prove serviceable to those concerned in, or preparing for, the responsible and arduous labors of medical practice.

The text of the articles in the English edition has nowhere been altered, except in correction of typographical or other oversights; and in the omission or abridgment of several extremely elaborate and complex Tables, which, it is believed, very few readers would follow in detail, as the inferences deduced from them are fully developed in the text. In two or three instances, the order of succession of articles has

been changed; not, however, without conformity to the general plan of arrangement.

The most considerable additions are upon the following subjects: the question of Blood-letting (Introduction); Rötheln, or German Measles; Apyretic measures in Typhoid Fever; Prophylaxis of Yellow Fever; recent history of Plague; Typho-malarial Fever; Pernicious Fever; Pathology and Treatment of Cholera; Chlorosis; Scrofula; Salicylic Acid in Rheumatism; Hysterical Hemianæsthesia; Hystero-epilepsy; Methomania; Athetosis; Puerperal Convulsions; Pseudo-hypertrophic Muscular Paralysis; Tubercular Meningitis; Ophthalmoscopic Appearances in Brain Disease; Cerebral Localization; Spinal and Cerebro-spinal Sclerosis; Symptomatology of Locomotor Ataxy; Reflex Paralysis; Croup; Communicability of Phthisis; Treatment of Pneumonia; Paracentesis Pericardii; Cardiac Exhaustion; Hæmophilia; Cholera Infantum; Trichina; Progressive Pernicious Anæmia.

All additions are marked by brackets, and signed [H.].

HENRY HARTSHORNE.

PHILADELPHIA, *November*, 1879.

ENGLISH PREFACES.

PREFACE TO THE FIRST EDITION OF VOL. I.

The object proposed to himself by the Editor of this *System of Medicine* is to present, within as small a compass as is consistent with its practical utility, such an account of all that constitutes both the natural history of disease and the science of pathology as shall be of service in either preventing the occurrence, or detecting the presence, and guiding the treatment of special forms of illness.

As the science and art of Medicine have within the last few years increased very greatly, in regard to both facts and principles, it is held to be desirable—and indeed almost imperative—in order to secure the ends that have been mentioned, to divide the large field of growing knowledge into such comparatively small sections as should be enriched by the results of individual and special culture. The Editor feels confident that these results have been obtained; and, in order to secure the like confidence of others, has simply to refer to the names of the many distinguished men who have kindly contributed to this volume.

The general scope of the *System of Medicine*, and the mode in which it has been carried out, render unnecessary any discussion of the so-called "Principles" of Medicine; and the Editor has preferred, by the omission of articles on "general" subjects, to incur the risk of occasional repetition, rather than that of such apparent contradiction as might arise from the treatment of some matters "in the general" by one contributor, and "in detail" by many others.

He has, further, only to express his most grateful thanks to those who have rendered him their invaluable aid; and to submit these results of their toil to the Profession of which they are the distinguished ornaments.

March 21st, 1866.

PREFACE TO THE SECOND EDITION OF VOL. I.

In this Second Edition, the articles have been submitted for revision to their respective authors. That on Purpura, by the late Dr. Thomas Hillier, has been revised by Dr. Tilbury Fox.

The Editor has introduced into this volume the article on Epidemic Cerebro-Spinal Meningitis, which in the First Edition was placed among the diseases of the Nervous System.

38 Grosvenor Street, Grosvenor Square,
January 18th, 1870.

PREFACE TO VOLUME III.

In dealing with so large a series of subjects as those which make up the section on "Diseases of the Respiratory System," some repetition of statement and occasional divergence of opinion have been found inevitable. The Editor has, however, thought it desirable to allow the occurrence of the former, in order to give completeness to separate articles, and has taken pleasure in the representation of the latter, inasmuch as, in his opinion, such divergence expresses, with the greatest faithfulness, the present state of scientific knowledge on many unsettled problems of pathology, and by so doing will prove more useful than would any attempt at enforced uniformity of teaching.

The general doctrine of Tubercle; the relation which that material bears to local and general diseases; the precise meaning of certain morbid conditions, the characters of which are matters of familiar recognition; and the inter-relations of many well-known words, are each and all of them susceptible of various interpretations: and the Editor is grateful and glad to be able to bring together in a connected form, under the notice of his professional brethren, the views that are severally entertained by those distinguished authors who have already shed much light upon these obscure regions, and have furnished many of the results of their finest labors in the present work.

J. RUSSELL REYNOLDS.

38 Grosvenor Street,
August 8th, 1871.

PREFACE TO VOLUME IV.

The Articles on Position and Malposition of the Heart, on Angina Pectoris, on Pericarditis, and Endocarditis, were begun by their respective authors some years ago, and several distinct portions of each of those articles were at once committed to the press. But both Dr. Gairdner and the late Dr. Sibson held that much new matter must be introduced into them; and by far the largest contributor to this volume, the late Dr. Sibson, found a mass of facts at his disposal, the analysis and representation of which occupied an amount of time and space that far exceeded his anticipation. The entire originality of his work, the subtlety of thought which it displayed, the carefulness of the observations upon which it was based, the catholicity of the views which it expressed, the honest, kind, although keen criticism that it contained of the opinions of other workers, and the intimate and important relations of all its parts, decided me not to reduce its magnitude beyond that which it now presents, and to wait for its completion. Those who know what it is to give a concise account of facts derived from their personal observation, and represented by the statistical method, will appreciate the years of labor that have been bestowed upon the articles, Position and Malposition of the Heart, upon Pericarditis and Endocarditis. Their Author, when he left England during this past autumn, had left one table uncorrected, and three pages on Carditis unwritten; I have endeavored to correct the table, and Dr. Gowers has written the article on Carditis.

We have, in this volume, the results of many years of Dr. Sibson's ardent toil, and the last, and, as I think, the best production of that earnest, industrious, enthusiastic worker, and most kind and genial friend.

Another of the contributors to this volume has also passed away since his papers were printed, and happily in the main corrected by himself; I refer to the late Dr. Warburton Begbie, whose work was as good as his heart was large, and who never spared any pains to carry to the highest point of his ability even the smallest fragment of labor that he undertook to perform.

J. RUSSELL REYNOLDS.

38 Grosvenor Street,
December, 1876.

PREFACE TO VOLUME V.

The fifth and concluding volume of this System of Medicine will, I believe, be as instructive and interesting as any of those which have preceded it. It contains information upon a very wide range of diseases, and the manner in which the authors of the respective articles which fill its pages have dealt with them throughout, has been such as to justify my belief. To those who have contributed to this volume, and to whom I can now address myself, I offer my heartiest thanks, both for the readiness with which they have done their work, and for the industry, learning, and critical faculty which they have displayed.

One of the contributors, Dr. Basham, was removed by death before he had completed his work, but I was most kindly and ably assisted by Dr. Frederick Roberts, in the anxious work of editing MSS., which in some cases proved to be but half completed. To Dr. Gowers, I am deeply grateful, not only for the very able papers which he has written, but also for his kindness in rendering me very great assistance in the preparation of this volume for the press.

It is not a matter for much surprise that a work, which has taken so long a time to produce, by men who had already become eminent in our profession—the longevity of which is less than that of most—should not be completed until many of its contributors had passed beyond the reach of either our praise or our blame, and that they should not have seen the work, which they had done so carefully, placed in its proper niche in the edifice that they had helped to raise. To two of these, Drs. Warburton Begbie and Sibson, I have made some allusion in my note of introduction to the fourth volume; of the others, from whom I had expected further help, I must say something here.

Dr. Anstie—whose loss the profession has never ceased to feel, and about whom, every one who had the privilege of his friendship can scarcely speak in terms that shall do justice to the memory of his earnest, loving soul, and of that devotion to duty and science which led to his early death—was one of my most helpful and considerate coadjutors in the production of the second volume, not only by contributing his papers to the *System of Medicine*, but by the great assistance which he rendered me in the preparation of the second volume for the press. From him, I had hoped for much more help, but, for him "there was nobler work to do."

Dr. Basham entered most heartily into the scheme of this book, but the publication of his papers was long delayed in consequence of circumstances which I explained in my note to the fourth volume. Those which he has left behind him, nearly completed, and which have been finished by Dr. Frederick Roberts, will show, as his other works have done, the simple and scientific character of his writing, and the thorough practicality of all his teaching.

The death of Dr. Warburton Begbie, to whom I have already alluded, occasioned another of the great losses which this *System* has sustained. There are few, if any, who have surpassed him in the scientific ability, scholarship, and high moral tone of his work.

Dr. Hughes Bennett, whose strong personal views on pathological questions gave an interest to the subjects with which he dealt, contributed the article on Phthisis, containing much original matter and thought, which will be useful to all interested in the history of tuberculosis.

The death of Dr. Thomas Hillier, the whole of whose published works afforded such rich promise of still more useful labors, occasioned another loss which the profession could ill sustain, a loss also to myself as a friend, colleague, and, as I hoped, future fellow-worker.

To Dr. Parkes, it is absolutely impossible for me to express my obligation. From the first day that I planned the construction of these volumes, until the last day but one before his death, he was my faithful counsellor and friend. His direct contribution to the *System of Medicine* was comparatively small, but the help that he gave me, indirectly, was immeasurably large. He was punctual to an hour, and precise to a three-place decimal. High above all his scientific work, great as it was, rose his grand moral character, for which all who have known him well must be profoundly grateful, in feeling that they are "better men, and are conscious that they may be better still."

Dr. Hyde Salter contributed a valuable paper on Asthma, condensing for this *System* the result of a great many years of good, scientific work. His well-known and recorded personal sufferings from the malady, upon which he wrote so ably, supply an interest and instruction that could perhaps have not been otherwise obtained.

To Dr. Sibson, the profession is much indebted for some most carefully elaborated papers, which appeared in the fourth volume, and to which in its introductory preface I have already alluded, so that I can here do no

more than reiterate my regret for the loss which medical science and literature have sustained in his unexpected death.

Dr. Edward Smith, a highly distinguished member of our profession, who had contributed much to the advance of medical science, and especially in some of its more practical bearings upon daily life, took a warm interest in the volumes which I have edited, and, until his work was prematurely arrested by death, he had hoped to render still further assistance in their production.

Dr. Squarey, a worker of great earnestness and promise, contributed several articles on Diseases of the Mouth and Throat to the third volume, but his life was cut short in the midst of his devoted labor.

The value of this work is greatly enhanced by the Indices to each separate volume, and also to the five volumes collectively. For the production of these, I have to thank, and I do so most heartily, Mr. Marcus Beck, the late Dr. Loy, and Dr. J. W. Langmore, the last of whom has furnished the Indices to the fourth and to the fifth volume.

To the dead and to the living, I again express my thanks, and feel most grateful that I have been able to put into the hands of the medical profession a series of admirable original contributions to the medical literature of our country, of which I think any country might be proud.

J. RUSSELL REYNOLDS.

38 Grosvenor Street,
November, 1878.

CONTENTS OF VOL. I.

INTRODUCTION, by the EDITOR.

	PAGE
Definition of Disease; and Names of Diseases	17
Structural and Functional Disease	19
Natural History of Disease	21
Causes of Disease	21
Predisposing Causes	21
Exciting Causes	23
Symptoms of Disease	23
Symptoms and Signs	23
Objective and Subjective Symptoms	24
Natural History of Disease—	
Course of Disease	25
Duration of Disease	25
Termination of Disease	26
Diagnosis of Disease	26
Pathology	27
Pathological Anatomy	28
Prognosis	29
Therapeutics and Hygienics	30
Classification of Diseases	32

PART I.

GENERAL DISEASES, OR AFFECTIONS OF THE WHOLE SYSTEM.

§ I. *Those determined by Agents operating from without.*

INFLUENZA, by EDMUND A. PARKES, M.D., F.R.S.

Definition	33
Synonyms	33
History of Influenza	34
Spread of the Disease	34
Etiology	34
Symptoms	41
Consideration of the Special Symptoms	41
Varieties	43
Mortality	43
Diagnosis	43
Pathology	43
Morbid Anatomy	44
Prognosis	44
Treatment	45

HOOPING-COUGH, by EDWARD SMITH, M.D., F.R.S.

Definition of Hooping-Cough	48
History	48
Causes	48
Symptoms	49
Diagnosis	52
Pathology	52
Morbid Anatomy	52
Prognosis	53
Treatment	53

DIPHTHERIA, by WILLIAM SQUIRE, L.R.C.P., LOND.

Definition	57
Synonyms	57
Name	58
History	58
Etiology	61
Symptoms	65
Diagnosis	70
Pathology	72
Morbid Anatomy	74
Prognosis	77
Therapeutics	78

SCARLET FEVER, by Samuel Jones Gee, M.D., Lond.

	PAGE		PAGE
Definition	83	Sequelæ	89
Causes	84	Diagnosis	93
Symptoms	84	Morbid Anatomy	94
Symptoms of Ordinary Scarlet Fever	85	Prognosis	94
Symptoms of Malignant Scarlet Fever	88	Prophylaxis	95
Symptoms of Latent Scarlet Fever	89	Treatment	95

DENGUE, or DANDY FEVER, by William Aitken, M.D.

Definition	98	Etiology and Propagation	103
Synonyms	98	Diagnosis	103
History	98	Treatment	103
Symptoms	99		

ROSEOLA, by Hermann Beigel, M.D.

Definition	104	Diagnosis	106
Cause	105	Prognosis	106
Course	105	Treatment	106

MEASLES, by Sydney Ringer, M.D.

Definition	106	Complications and Sequelæ	111
Synonyms	106	Diagnosis	113
Symptoms	106	Prognosis	114
Varieties	107	Treatment	114

[RÖTHELN (German Measles), by Henry Hartshorne, M.D.

Symptoms	117	Treatment	117]
Diagnosis	117		

PAROTITIS, by Sydney Ringer, M.D.

Definition	118	Pathology	119
Synonyms	118	Diagnosis	120
Symptoms	118	Treatment	120

SUDAMINA and MILIARIA, by Sydney Ringer, M.D.

Sudamina	122	Treatment	124
Miliaria	123		

VARICELLA, by Samuel Jones Gee, M.D.

Definition	124	Prognosis	127
Causes	125	Treatment	127
Description of the Disease	125	Varieties and Synonyms	127
Diagnosis	126		

SMALLPOX, by J. F. Marson.

Definition	127	Varieties	129
Synonyms	127	Primary Fever	132
History	127	Secondary Fever	132
Description of Smallpox	128	Diagnosis	135

	PAGE
Prognosis	136
Susceptibility to Smallpox	138
Infectious Nature of Smallpox	138
Treatment	140
Mortality from Smallpox	145
Morbid Appearances	146
Anatomical Characters of the Variolous Pock	147
Smallpox after Vaccination.—Varicelloid, and modified Smallpox	149
Epidemic Diffusion of Smallpox	156
Inoculation from Smallpox	156

VACCINATION, by Edward Cator Seaton, M.D.

Phenomena of Cow-pox in the Human Subject	159
Phenomena of Revaccination	161
Method of Vaccinating	161
Protection afforded by Vaccination against Smallpox	166
Revaccination	174
Relations of Variola and Vaccinia	176
Alleged Dangers of Vaccination	177

GLANDERS—EQUINIA, by Arthur Gamgee, M.D., and John Gamgee.

Definition	182
Nomenclature and History	182
Glanders and its Varieties in the Horse, Ass, and Mule	183
Chronic Glanders	183
Acute Glanders	183
Chronic Farcy	184
Acute Farcy	184
History of the Disease in Man	185
Etiology of the Disease in Man	188
Semeiology	189
Diagnosis	191
Morbid Anatomy	191
Prognosis	192
Therapeutics	192

HYDROPHOBIA, by John Gamgee and Arthur Gamgee, M.D.

Definition	192
Synonyms	192
History	192
Causes	195
Hydrophobia in Man	197
Symptoms of Hydrophobia in man	197
Diagnosis	199
Morbid Anatomy	199
Prognosis	200
Therapeutics	200

ENTERIC or TYPHOID FEVER, by John Harvey, M.D., F.L.S.

Definitive Description	201
Synonyms	201
Preliminary Observations	202
Clinical History of the Disease	202
Morbid Anatomy	209
Pathology	218
Associated Pathology of Enteric Fever	221
Varieties	232
Distribution	234
Causes	235
Diagnosis	244
Prognosis	246
Treatment	247

TYPHUS FEVER, by George Buchanan, M.D.

Definition	251
Etiology	252
Symptomatology	254
Duration	261
Termination and Sequelæ	261
Diagnosis	262
Pathology	263
Morbid Anatomy	264
Prognosis and Mortality	265
Therapeutics	266
Varieties	269

RELAPSING FEVER, by J. Warburton Begbie, M.D.

Definition	269
History, Nomenclature, and Bibliography	269
Geographical Distribution	273
Etiology	274
Symptomatology	277
Therapeutics	280

YELLOW FEVER, by J. Denis Macdonald, M.D., F.R.S.

	PAGE		PAGE
Definition	281	Pathology	286
Synonyms	281	Morbid Anatomy	290
History	281	Prognosis	292
Altitudinal and Horizontal Ranges	284	Therapeutics	293
Symptoms	284	Varieties and their Classification	294
Diagnosis	285		

EPIDEMIC CEREBRO-SPINAL MENINGITIS, by John Netten Radcliffe.

Definition	296	Prognosis	305
Synonyms	296	Morbid Anatomy	305
Description of the Disease	297	History and Geographical Distribution	306
Special Symptoms	299	Etiology	308
Complications	303	Nature	311
Duration	303	Treatment	312
Termination	304	Bibliography	314
Mode of Death	304		
Diagnosis	304		

THE PLAGUE, by Gavin Milroy, M.D.

Definition	314	Curative Treatment	317
Synonyms	314	Natural History	317
Symptoms	314	Causation	317
Diagnosis	315	Prophylaxis, &c.	317
Morbid Anatomy	316	[Recent History	320]

ERYSIPELAS, by J. Russell Reynolds, M.D., F.R.S.

Definition	321	Pathology	325
Synonyms	321	Morbid Anatomy	325
Natural History	321	Prognosis	326
Causes	321	Treatment	327
Symptoms	322	Varieties	329
Diagnosis	325		

PYÆMIA, by John Syer Bristowe, M.D.

Etymology	330	Symptoms—	
Definition	330	Considered further in regard to Pyæmia	347
Pathology	330	Diagnosis	347
Morbid Anatomy	331	Treatment	348
General Pathology	338	Prophylactic	348
Symptoms	344	Medical	350
Considered collectively	344		
Considered in relation to the various Organs	345		

MALARIAL FEVERS, by W. C. Maclean, M.D.

Intermittent Fever	352	Remittent Fever	365
Definition	354	Definition	365
Synonyms	354	Synonyms	365
History and modes of Commencement	354	History	365
Causes	356	Symptoms	366
Symptoms	356	Diagnosis	368
Diagnosis	358	Pathology	368
Morbid Anatomy	358	Prognosis	368
Prognosis	359	Treatment	369
Treatment	360		

DYSENTERY, by W. C. Maclean, M.D.

	PAGE		PAGE
Definition	372	Symptoms—	
Terminology	372	Malignant	377
History	372	Scorbutic	377
Modes of Commencement	373	Diagnosis	378
Causes	373	Pathology	378
Symptoms	375	Morbid Anatomy	378
Acute	376	Prognosis	380
Chronic	376	Treatment	380
Malarious	377		

EPIDEMIC CHOLERA, by Edward Goodeve, M.B.

Definition	384	Diagnosis	409
Synonyms	384	Pathology	410
History	385	Morbid Anatomy	410
Etiology	386	Considered during life	412
Symptoms	398	Prognosis	415
Varieties	406	Treatment	415
Duration	408	Treatment of Choleraic Diarrhœa	420
Mortality	408	Prophylaxis	421

CONSTITUTIONAL SYPHILIS, by Jonathan Hutchinson, F.R.C.S.

Stages of Syphilis	424	Diagnosis	438
Conditions interfering with their Evolution	426	Diagnosis when consequent upon inherited Taint	440
Modes of Communication	429	Contrast between acquired and inherited Syphilis	443
Tertiary Symptoms	431		
Treatment	435		

§ II. *General Diseases determined by Conditions existing within the Human Body.*

SCORBUTUS, or SCURVY, by Thomas Buzzard, M.D.

Definition	445	Pathology	455
Synonyms	445	Morbid Anatomy	456
Etiology	445	Prognosis	457
Symptoms	451	Therapeutics	458
Diagnosis	454		

PURPURA, by Thomas Hillier, M.D., revised by Tilbury Fox, M.D.

Description of different Forms	460	Pathological Anatomy	463
Anatomical Characters	460	Nature	465
Symptoms	460	Prognosis	466
Varieties	461	Diagnosis	466
Causes	463	Treatment	467

[CHLOROSIS, by Henry Hartshorne, M.D.

History	468	Pathology	469
Symptoms	468	Prognosis	470
Causation	469	Treatment	470]

RICKETS, by W. Aitken, M.D.

	PAGE		PAGE
Definition	472	Pathology	485
Synonyms	472	Morbid Anatomy	487
Causes	473	Prognosis	494
Symptoms	475	Therapeutics	495
Diagnosis	484		

[SCROFULA, by Henry Hartshorne, M.D.

History	497	Pathology	503
Symptomatology	498	Causation	507
Anatomy	503	Treatment	509]

GOUT, by Alfred Baring Garrod, M.D., F.R.S.

Definition	512	Morbid Anatomy	523
Synonyms	512	Causes dependent on the Individual	526
History	512	Causes independent of the Individual	527
Description of an attack of Acute Gout, and progress of the Disease	512	Pathology	531
Gout in a Chronic form	516	Diagnosis	536
Irregular Gout; effects on different forms of Gout	518	Prognosis	537
State of the Urine in different forms of Gout	522	Treatment	538
		Diet and Regimen in different forms of Gout	548

RHEUMATOID ARTHRITIS, by A. B. Garrod, M.D., F.R.S.

Definition	550	Pathology	554
Synonyms	550	Diagnosis	555
History	550	Prognosis	556
Description of Rheumatoid Arthritis	551	Treatment	556
Morbid Anatomy	553	Diet and Regimen	558
Causes	554		

RHEUMATISM, by A. B. Garrod, M.D., F.R.S.

Articular Rheumatism	559	Articular Rheumatism— Effects of Climate, Seasons, and Weather	565
Definition	559	Pathology	565
Synonyms	559	Diagnosis	566
History	559	Prognosis and Treatment	567
Description of Acute and Subacute Rheumatism	559	Muscular Rheumatism	573
Consideration of the different Phenomena	561	Definition	573
Condition of the Blood in Acute Articular Rheumatism	562	Synonyms	573
Urine in Acute Articular Rheumatism	562	History	573
Cardiac and other Inflammatory Affections	563	Description	573
Morbid Anatomy	563	Causes	574
Causes	564	Pathology	575
		Diagnosis	575
		Prognosis	575
		Treatment	575

GONORRHŒAL RHEUMATISM, by B. E. Brodhurst, F.R.C.S.

History	576	Treatment	579
Symptoms	576		

PART II.

LOCAL DISEASES.

INTRODUCTION, by The Editor . . . . PAGE 581

GENERAL DISEASES OF THE NERVOUS SYSTEM, List of . . . 584

INSANITY, by Henry Maudsley, M.D., F.R.C.P.

Synonyms 584
Definition 584
Classification 585
Causes 587
Forms of Insanity and their Symptomatology 591
Melancholia 592
Mania 595
Monomania 599
Dementia 600
Forms of Insanity and their Symptomatology—
Moral Insanity 601
Idiocy 603
General Paralysis 605
Diagnosis 607
Pathology 610
Morbid Anatomy 614
Prognosis 616
Therapeutics 618

HYPOCHONDRIASIS, by Sir William Withey Gull, Bart., M.D., D.C.L., F.R.S., and Francis Edmund Anstie, M.D., F.R.C.P.

Definition 623
Nomenclature 623
History 624
Symptoms 624
Diagnosis 626
Prognosis 628
Etiology 628
Treatment 629

HYSTERIA, by J. Russell Reynolds, M.D., F.R.S.

Natural History 631
Causes 631
Symptoms 634
Pathology 640
Diagnosis 642
Prognosis 643
Treatment 644

ECSTASY, by Thomas King Chambers, M.D., F.R.C.P. . 646

[HYSTERO-EPILEPSY, by Henry Hartshorne, M.D. . 649]

CATALEPSY, by Thomas King Chambers, M.D., F.R.C.P.

Name 652
Definition 652
Description 652
Causes 654
Treatment 658

SOMNAMBULISM AND ALLIED STATES, by Thomas King Chambers, M.D., F.R.C.P.

Description 658
Treatment 660

SUN-STROKE, by W. C. Maclean, M.D.

Definition 661
Synonyms 661
History 661
Etiology 664
Symptoms 666
Diagnosis 667
Pathology 667
Morbid Anatomy 668
Mortality 668
Prognosis 668
Prophylaxis 668
Treatment 669
Treatment of the Sequelæ . . 670

ALCOHOLISM, by Francis Edmund Anstie, M.D., F.R.C.P.

	PAGE		PAGE
Definition	670	Diagnosis	681
Synonyms	670	Prognosis	682
History	670	Complications	683
Etiology	671	Pathology	684
Symptoms	675	Treatment	684
Chronic Alcoholism	675		
Acute Alcoholism	678		

VERTIGO, by J. Spence Ramskill, M.D.

Definition	690	Stomachal Vertigo	691
Description	690	Vertigo of the Aged	693
Prognosis	691	Essential Vertigo	693
Etiology	691	Vertigo from Overwork	694
Varieties	691	Treatment	695

CHOREA, by C. B. Radcliffe, M.D., F.R.C.P.

Symptoms	696	Diagnosis	710
Exceptional Forms of Chorea	700	Prognosis	711
Pathology	704	Treatment	711
Causes	709		

PARALYSIS AGITANS, by W. Rutherford Sanders, M.D., F.R.C.P.

Synonyms	718	Diagnosis	726
Definition	718	Complications	727
Historical Notice	719	Pathology and Morbid Anatomy	727
Description	720	Treatment	729
Causes	725	Bibiography	730

[ATHETOSIS, by Henry Hartshorne, M.D. . . 731]

WRITER'S CRAMP, by J. Russell Reynolds, M.D., F.R.S.

Definition	732	Diagnosis	734
Synonyms	733	Prognosis	735
Symptoms	733	Pathology	735
Etiology	734	Treatment	737

CONVULSIONS, by J. Hughlings Jackson, M.D., F.R.C.P.

Convulsions in Children	738	Convulsions in Children—	
The Paroxysm	740	Treatment	749
Premonitory Symptoms	740	Convulsions in Adults	752
Sequelæ	744	The Convulsion begins unilaterally	752
The Causes of Convulsions	745	Causation	754
Prognosis	749	Death	761
		Treatment	761

EPILEPSY, by J. Russell Reynolds, M.D., F.R.S.

Definition	762	Natural History—	
Synonyms	763	Complications	776
Natural History	763	Pathology	777
General Prevalence of the Disease	763	Diagnosis	778
Causes	763	Prognosis	779
Symptoms	767	Treatment	780
Relations between the Symptoms	775		

MUSCULAR ANÆSTHESIA, by J. Russell Reynolds, M.D., F.R.S.

	PAGE		PAGE
Definition	783	Diagnosis	784
Nomenclature	783	Pathology	785
Symptoms	783	Prognosis	785
Causes	784	Treatment	786

WASTING PALSY, by William Roberts, M.D., F.R.C.P.

Definition	786	Diagnosis	790
Synonyms	786	Morbid Anatomy	790
History	786	Pathology	793
Etiology	786	Prognosis	796
Symptoms	788	Therapeutics	798
Course and Duration	789		

METALLIC TREMOR, TREMBLEMENT MÉTALLIQUE, by W. Rutherford Sanders, M.D., F.R.C.P.

Synonyms	801	Lead Tremors	806
Definition	801	Symptoms	807
Mercurial Tremor or Shaking Palsy	801	Causes	807
Causes	801	Course	807
Description	802	Prognosis	807
Symptoms	802	Pathology and Morbid Anatomy	807
Course and Prognosis	804	Diagnosis	807
Diagnosis	805	Treatment	807
Pathology and Morbid Anatomy	805		

PARTIAL DISEASES OF THE NERVOUS SYSTEM, List of . . . 808

SIMPLE MENINGITIS, by J. Spence Ramskill, M.D.

Definition	808	Acute Meningitis—	
Acute Meningitis	808	Etiology	814
Symptoms	808	Diagnosis	815
Inflammation of the Dura Mater	813	Treatment	815
Progress, Duration, and Termination	813	Chronic Meningitis	816
Pathological Anatomy	814	History and Description	816
		Treatment	817

TUBERCULAR MENINGITIS, by Samuel Jones Gee, M.D., F.R.C.P.

Causes	817	Primary Tubercular Meningitis in the Child—	
Symptoms	818	(*b*) Meningitis of the Convexity	827
Primary Tubercular Meningitis in the Child	818	Secondary Tubercular Meningitis in the Child	828
Symptoms	818	Tubercular Meningitis in the Adult	829
Invasion	819	Diagnosis	829
The Established Disease	820	Morbid Anatomy	832
(*a*) Meningitis of the Base	820	Prognosis and Treatment	835

CHRONIC HYDROCEPHALUS, by J. Spence Ramskill, M.D.

Anatomical Characters	836	Diagnosis	839
Symptoms	837	Treatment	839

MENINGEAL HEMORRHAGE, by J. Spence Ramskill, M.D.

	PAGE		PAGE
Symptoms	841	Congenital Malformations of the Meninges	844
Treatment	842		
Adventitious Products in the Meninges	843		

CONGESTION OF THE BRAIN, by J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.

Symptoms	845	Morbid Anatomy	851
Causes	848	Prognosis	853
Diagnosis	848	Treatment	853
Pathology	849		

CEREBRITIS, by J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.

Causes	855	Pathology	855
Symptoms	855	Prognosis	856
Diagnosis	855	Treatment	856

SOFTENING OF THE BRAIN, by J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.

Definition	856	Pathology	865
Causes	857	Morbid Anatomy	873
Symptoms	857	Diagnosis	880
Acute Softening of the Brain	857	Prognosis	882
Chronic Softening of the Brain	864	Treatment	882

ADVENTITIOUS PRODUCTS OF THE BRAIN, by J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.

Symptoms	883	Prognosis	901
Diagnosis	886	Treatment	901
Morbid Anatomy	889		

CEREBRAL HEMORRHAGE AND APOPLEXY, by J. Hughlings Jackson, M.D., F.R.C.P.

Morbid Anatomy	902	Localization of Lesions—	
Etiology and Pathology	905	Pons Variolii	914
Constitutional State prior to Cerebral Hemorrhage	906	Medulla Oblongata	915
Hemorrhage from Aneurism of the larger Cerebral Vessels	910	Cerebellum	915
Localization of Lesions	910	The Apoplectic Condition	920
Cerebral Hemisphere	910	Diagnosis	922
Lateral Ventricles	911	Premonitory Symptoms	923
Corpus Striatum	911	Mode of Onset of Cerebral Hemorrhage	925
Thalamus Opticus	915	Special Diagnosis	927
Crus Cerebri	913	Prognosis	931
		Treatment	932

ABSCESS OF THE BRAIN, by Sir William W. Gull, Bart., M.D., F.R.S., and Henry G. Sutton, M.B., F.R.C.P.

PAGE
History 934
Morbid Anatomy 936
Symptoms 937
Pathology 938
Diagnosis 940
Treatment 941

DISEASES OF THE SPINAL COLUMN, List of 942

DISEASES OF THE SPINAL CORD, by C. B. Radcliffe, M.D., F.R.C.P.

Preliminary Remarks . . . 942
Meningitis 951
Symptoms 951
Post-mortem Appearances . . 955
Causes 955
Diagnosis 955
Prognosis 955
Treatment 956
Myelitis 956
Symptoms 956
Post-mortem Appearances . . 962
Causes 963
Diagnosis 963
Prognosis 964
Treatment 964
Congestion 965
Symptoms 965
Post-mortem Appearances . . 968
Causes 968
Diagnosis 968
Prognosis 968
Treatment 968
Tetanus 968
Symptoms 969
Post-mortem Appearances . . 976
Causes 977
Diagnosis 978
Prognosis 979
Treatment 979
Locomotor Ataxy 980
Symptoms 981
Causes 989
Locomotor Ataxy—
Prognosis 989
Diagnosis 989
Treatment 990
Spinal Irritation 991
Symptoms 991
Post-mortem Appearances . . 997
Causes 997
Diagnosis 997
Prognosis 998
Treatment 998
General Spinal Paralysis . 999
Hysterical Paraplegia . . 1000
Reflex Paraplegia . . . 1001
Infantile Paralysis . . . 1004
Spinal Hemorrhage . . . 1007
Non-inflammatory Spinal Softening 1008
Induration of the Spinal Cord 1008
Atrophy and Hypertrophy of the Spinal Cord . . . 1015
Tumor of the Spinal Cord . 1016
Concussion of the Spine . . 1016
Compression of the Spinal Cord 1017
Caries of the Vertebral Column 1017
Spina Bifida 1018

DISEASES OF THE NERVES, List of 1020

NEURITIS AND NEUROMA, by J. Warburton Begbie, M.D., F.R.C.P.E.

Neuroma 1022
Traumatic Neuroma . . . 1025

NEURALGIA, by Francis E. Anstie, M.D., F.R.C.P.

Definition 1026
Synonyms 1026
Clinical History and Symptoms . 1027
Varieties 1027
Complications 1037
Diagnosis 1040
Prognosis 1040
Pathology and Etiology . . . 1041
Treatment 1042

LOCAL PARALYSIS FROM NERVE DISEASE, by J. Warburton Begbie, M.D., F.R.C.P.E.

PAGE

General History 1048
Varieties 1050
Prognosis 1053

LOCAL SPASMS, by J. Warburton Begbie, M.D., F.R.C.P.E. 1055

TORTICOLLIS, by J. Russell Reynolds, M.D., F.R.S.

Definition 1060
Synonyms 1060
Causes 1061
Symptoms 1061
Diagnosis 1062
Pathology 1062
Prognosis 1063
Treatment 1063

LOCAL ANÆSTHESIÆ, by J. Warburton Begbie, M.D., F.R.C.P.E. 1064

Index 1067

List of Chief Authors referred to in Each Article 1109

LIST OF CONTRIBUTORS TO VOL. I.

William Aitken, M.D., L.R.C.S. Edinburgh; Professor of Pathology in the Army Medical School, Netley.

Francis Edmund Anstie, M.D., F.R.C.P.; Senior Assistant Physician to the Westminster Hospital, and Lecturer on Medicine in the Westminster Hospital Medical School.

Henry Charlton Bastian, M.A., M.D., F.R.S., F.L.S.; Professor of Pathological Anatomy in University College; Physician to University College Hospital.

J. Warburton Begbie, M.D., F.R.C.P. Edinburgh; Physician to the Royal Infirmary of Edinburgh.

Hermann Beigel, M.D. Berlin; M.R.C.P. Lond.; Fellow of the Imperial Leopold-Carolina Academy of Germany; Physician to the Metropolitan Free Hospital.

John Syer Bristowe, M.D., F.R.C.P. Lond.; Physician to St. Thomas's Hospital.

Bernard Edward Brodhurst, F.R.C.S., F.L.S., Surgeon to, and Lecturer on Orthopædic Surgery at, St. George's Hospital.

George Buchanan, M.D., F.R.C.P. Lond.; Fellow of University College; formerly Physician to the Lond. Fever Hospital, and to the Hospital for Sick Children; Inspector of Public Health in Medical Department of H.M. Privy Council.

Thomas Buzzard, M.D., M.R.C.P. Lond., University Medical Scholar; Physician to the National Hospital for the Paralyzed and Epileptic; formerly on the Staff of H.H. Omar Pacha.

Thomas King Chambers, M.D. Oxon., F.R.C.P. Lond.; Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician and Lecturer on Medicine in St. Mary's Hospital.

Tilbury Fox, M.D. Lond., M.R.C.P.; University Medical Scholar; Physician to the Department for Skin Diseases in University College Hospital.

Arthur Gamgee, M.D. Edinburgh, late Resident Physician to the Royal Infirmary of Edinburgh.

John Gamgee, Principal of the Albert Veterinary College.

Alfred Baring Garrod, M.D., F.R.S., F.R.C.P. Lond.; Physician to King's College Hospital, and Professor of Materia Medica and Therapeutics in King's College.

Samuel Jones Gee, M.D., F.R.C.P. Lond., University Medical Scholar, Assistant Physician to St. Bartholomew's Hospital and to the Hospital for Sick Children.

Edward Goodeve, M.B., Deputy Inspector-General of Hospitals H.M. Bengal Army; Hon. Physician to the Queen; late Professor of Medicine in the Medical College, and First Physician to the Medical College Hospital, Calcutta; Member of the Senate of the University of Calcutta.

Sir William Withey Gull, Bart., M.D., F.R.S., F.R.C.P. Lond., D.C.L. Oxon.; late Physician to Guy's Hospital.

JOHN HARLEY, M.D., F.R.C.P. Lond., F.L.S.; Hon. Fellow of King's College, London; Assistant Physician to the London Fever Hospital.

HENRY HARTSHORNE, M.D., lately Professor of Hygiene in the University of Pennsylvania, &c.

THOMAS HILLIER, M.D., F.R.C.P. Lond.; Fellow of University College; late Physician to the Hospital for Sick Children, and to the Department for Skin Diseases in University College Hospital.

JONATHAN HUTCHINSON, F.R.C.S. Lond.; Surgeon to the London Hospital, and to the Metropolitan Free Hospital.

J. HUGHLINGS JACKSON, M.D., F.R.C.P., Physician to the National Hospital for the Paralyzed and Epileptic; Physician to the London Hospital.

JOHN DENIS MACDONALD, R.N., M.D., F.R.S., Staff-Surgeon to H.M.S. *Victory*.

WILLIAM CAMPBELL MACLEAN, M.D. Edin.; Member of the Senate of the University of Madras; Deputy Inspector-General of Hospitals; Professor of Clinical and Military Medicine in the Army Medical School, Netley.

JAMES FURNESS MARSON, F.R.C.S. Lond.; Corresponding Fellow of the Royal Academy of Surgeons of Madrid; Resident Surgeon to the Smallpox and Vaccination Hospital.

HENRY MAUDSLEY, M.D. Lond., F.R.C.P., Physician to the West of London Hospital; Professor of Medical Jurisprudence in University College, London.

GAVIN MILROY, M.D. Edinburgh, F.R.C.P. Lond.; Superintendent Medical Inspector to the General Board of Health.

EDMUND A. PARKES, M.D., F.R.S., F.R.C.P. Lond.; Emeritus Professor of Clinical Medicine in University College; Member of the General Council of Medical Education; Professor of Hygiene in the Army Medical School, Netley.

CHARLES BLAND RADCLIFFE, M.D., F.R.C.P. Lond.; Physician to the Westminster Hospital, and to the National Hospital for the Paralyzed and Epileptic.

JOHN NETTEN RADCLIFFE, Hon. Secretary of the Epidemiological Society; Inspector of Public Health in Medical Department of H.M. Privy Council.

J. SPENCE RAMSKILL, M.D. Lond.; Physician to the London Hospital, and to the National Hospital for the Paralyzed and Epileptic.

J. RUSSELL REYNOLDS, M.D., F.R.S., F.R.C.P. Lond., Examiner in Medicine to the University of London; Professor of the Principles and Practice of Medicine in University College; Physician to University College Hospital.

SYDNEY RINGER, M.D., M.R.C.P. Lond.; Professor of Materia Medica and Therapeutics in University College; Physician to University College Hospital, and to the Hospital for Sick Children.

WILLIAM ROBERTS, M.D., F.R.C.P. Lond.; Physician to the Manchester Royal Infirmary; Lecturer on Medicine in the Manchester School of Medicine.

WILLIAM RUTHERFORD SANDERS, M.D., F.R.C.P. Edinburgh; Physician to the Royal Infirmary, and Lecturer on the Institutes of Medicine, Edinburgh.

EDWARD CATOR SEATON, M.D. Edin., M.R.C.P. Lond., Medical Department of H.M. Privy Council.

EDWARD SMITH, M.D., F.R.S., F.R.C.P. Lond.; Inspector to the Poor Law Board; late Assistant-Physician to the Hospital for Consumption, Brompton.

WILLIAM SQUIRE, I.R.C.P., M.R.C.S. Lond.; late Senior Surgeon to the St. Marylebone Infirmary.

HENRY G. SUTTON, M.B. Lond., F.R.C.P., Assistant Physician to the London Hospital and to the City of London Hospital for Diseases of the Chest.

A SYSTEM OF MEDICINE.

INTRODUCTION.

BY THE EDITOR.

DEFINITION OF DISEASE; AND NAMES OF DISEASE.—The attempt to define "disease" must be a failure until we are possessed of a satisfactory definition of "health;" and we are not likely to arrive at this possession until we are able to define the idea that we entertain of the still more fundamental fact of "life." Yet some attempt at definition is not only important, but even essential, for the work set before us in these volumes; inasmuch as the general ideas entertained about disease vary as the years pass on, and the position of "medicine" in the "system of the sciences" is not only expressed by the approximative definition that we frame of disease, but is actually determined by the principle or idea which such definition is constructed to convey.

If we regard disease in the "abstract," we have to deal with that which changes, fetters, renders painful, shortens, or puts an end to life; and, from this point of view, disease may be defined to be any condition of the organism which limits life in either its powers, enjoyments, or duration. We need not stop to discuss the many futile essays that have been made to define that which transcends definition, but which we all, more or less accurately, understand by "life." We accept it as a fact, of which we all know much, but of which we are all assured, by what we do know, that there is much more that we do not know; for it goes beyond our observation, not only at its beginning, and at its end, but in its middle term, when it is the most—but even then only partially—exposed to both our senses and our consciousness.

Disease is a condition of the individual man; it is always something more than the changes that we yet can recognize and describe in any particular organ or its function. It is the man who is ill; and, under all circumstances of illness, he has a diminished life. Some organs may be overactive; but their excess of work is needed either because work elsewhere has been left undone, or because it has been done so roughly that parts of organs have been killed before their time, and their wasted materials have to be changed and got rid of with all haste; or because that which held their activity in check has been damaged or destroyed. At all times such overwork is fatiguing and hazardous; often it is directly dangerous, and sometimes it is destructive. Disease is that which limits life—in its usefulness, enjoyments, or duration; and although the body is so constructed that it can often regain its balance when the disturbance has been slight, yet disease, under all circumstances and to all degrees, is the lowering of life, and, even in its most trivial forms, is the "shadow of death."

This mode of regarding disease furnishes us with the *measures* of its importance. A man is ill, or diseased, in degree exactly corresponding with this limitation of his life. A morbid idea may make life miserable and useless; a fatty tumor may be an unfelt excrescence. Many lives are spoiled by diseases that the anatomist cannot name; many lives are but little altered by growths or lesions which may be weighed and demonstrated before a crowd.

If we consider disease from another point, and deal with it as a "fact" of daily experience, we come to regard it as any departure from the structure or functions of the body as these are shown to us in health; and thus we may define it to be an abnormal condition of function, or structure, or both.

But if we contemplate disease in its relation to the many "names" by which its various forms are recognized, we have a complicated problem with which to deal, and can only solve it by endeavoring to separate that which is common to all phases of ill-health, from that which is pe-

culiar to the various names by which those phases may be known, but by which they are only imperfectly expressed. And in order to do this we must recall some typical examples of these names. A patient may be described as suffering from, or he may be said to be an example of, the disease called inflammation of the lung, hooping-cough, tuberculosis, anæmia, typhoid fever, hysteria, or of some other malady, which would or would not readily fall into one or the other of these categories. And be it observed that by this term "inflammation of the lung," is expressed a particular kind of change in one organ of the body; by "hooping-cough" is meant a special and characteristic variety of a symptom common to many very different affections; by "tuberculosis" is intended some general change in the whole body, distinguished from other general changes by its association with the appearance in one, two, or many organs of a particular material known as tubercle; by "anæmia" is understood literally only an absence or deficiency of blood, but generally an alteration of quality rather than of quantity; by "typhoid fever" is conveyed the idea of a change, of a particular type, in the whole organism, and one which is produced by the introduction into the body of a poison from without; while by "hysteria," and similar phrases, is conveyed some meaning or none at all, and, when the former, a meaning as various in character as are the individuals who use the word. There are other principles upon which disease has been named, and by which it is now described, but these examples are sufficient to show by their very existence the varying prevalence, at different periods, of diverse theories about disease; about the relation of the one organ to the whole system of organs; the nature of the changes which different organs may undergo; the value of particular functional alterations, and of special symptoms; the relation of the blood to life, and to the tissues of the body; the lien between certain materials we can see, and some general conditions we can appreciate by their effect on life; the position in which life-functions stand to the various poisonous agencies around them, and the concealment, by patent facts of little moment, of important conditions which may be inferred to be their cause. Thus the history of a science might be shown to be written in the names by which the objects about which it is concerned have been described and recognized: but such is not the end now in view; it is to show that the principles upon which diseases have been named have varied widely, not only at different periods, but at the same; and that so great is the diversity among them, and so strangely aberrant are the forms which disease sometimes assumes, that, hitherto, no self-consistent and at the same time practical nosology has been devised. English physicians have therefore thought it better to retain old names that were well understood, although based upon doubtful, if not erroneous pathology, rather than to invent new terms which could not possess the advantages of their predecessors, although they might very probably share their faults. We have therefore gradually accepted a nosology of most complex composition with the tacit or expressed admission on all hands, that by "pneumonia" is intended much beyond the particular condition of the lung; that it implies changes antecedent to itself in the general nutrition of the body, and alterations in all the tissues and in their processes when the disease itself appears; that the words "hooping-cough" convey more than is included in a particular variety of cough which is characterized by a hooping sound, viz., the well-known history and social relationships of a disease altogether distinct from the paroxysmal cough and hooping sounds not rarely met with in cases of chronic bronchitis and emphysema; that by "tuberculosis" is intended a condition as well marked by general as by local changes, and probably dependent upon some constitutional vice which determines, and is not determined by, the special form of local change; that by "anæmia" is meant much more than the mere etymology of the word can convey; that by "typhoid fever" is intended the description of a disease having relationships only very inadequately expressed by the words in common usage to denote it; and that by "hysteria" and similar expressions are understood more than the present state of medical science will explain, and much more than the words themselves accurately define.

In this state of medical nomenclature, and of medical science as represented by the names it sanctions, it is difficult to arrive at any other *definition* of "disease," than that it is the sum total of morbid changes in both function and structure; and we must further admit that the *names* by which diseases are recognized are somewhat arbitrary terms used for the purpose of recognition without any constant value as to the meaning of those words in a system of pathology. Sometimes the name expresses what is believed to be the essential or most important fact; sometimes the first link in a long chain of causes and effects; sometimes a characteristic symptom or group of symptoms; sometimes an idea as to the mode of origin of the disease; and sometimes such a negation of all theory as contents itself with words which shall be understood to mean certain things to which they bear no more pathological relation, although they may have more seeming scientific value, than the common

algebraical expressions for "unknown qualities,"—*x*, *y*, *z*.

But there is, underlying all our nosology, an idea of something special or individual in the diseases that we name. This notion is distinctly expressed with regard to some, hinted at in relation to others, and unsuccessfully concealed in respect of the rest. In spite of opinions to the contrary, the physician knows that there is, in his mind, an ideal type of disease, which he cannot define, and cannot find realized in actual practice, but yet to which type he refers the examples that come before him. Disease to him is something more than a group of symptoms, it is that which makes the group; and he is—as indeed he ought to be—like the naturalist in his process of nomenclature, striving to express, by the names he uses, that which occasions the inner relation between essential facts, and not merely that which will denote the outer and often quite superficial assemblage of phenomena.

As, however, the idea above alluded to is very different in different minds, and pathology is, as yet, in such a condition that it cannot furnish a complete scheme of nosology, based upon the attempt to express, by name, the fundamental fact of all the many maladies with which we are acquainted, disease is defined to be the sum total of changes from a condition of health which may be recognized in either function or structure, or both; and the names of diseases are held to be merely convenient expressions for their recognition.

Structural and Functional Disease.—In describing the elements of what we call disease, two terms have been frequently used, "structure" and "function;" and we know that the two phrases "structural disease" and "functional disease" have passed into common usage. Recently it has become somewhat the fashion to object to the latter, and to deny the existence of any such condition. It is necessary therefore to state the grounds upon which the phrase "functional disease" has been retained in this "System of Medicine," and the precise sense in which it is employed.

For this purpose three classes of facts have to be remembered. 1st. That there are some structural alterations, such for example as atheroma in the vessels, which may, if an individual has been killed by an accident, be found extensively distributed throughout the body, the existence of which had been neither known nor suspected by the presentation of any functional change, or symptom, during life. On the other hand, a man may have suffered for many years from discomfort, or marked derangement of the functions of the brain, heart, or lungs, and yet the most practised anatomist, with all means and appliances to help him, may fail to discover, *post-mortem*, any organic change which is sufficient to have accounted for them. 2d. Another class of facts, constantly lost sight of by those who deny the existence of functional disease, is to be found in the relations between structure and function in health. At the end of a day's work, and after a night's repose, we might find the two extreme conditions of the organism as regards function. For twelve hours every muscle, nerve, and organ has been doing its utmost, and, as we know, has been wearing out: during the hours of sleep, many organs have been doing little, and some nothing, whereas others have, as it seems, to work on without repose; but in all repair has been going on. By an examination of the body, killed suddenly at the end of one or the other of these periods, it might be possible to infer which had been the condition immediately preceding death. But this inference would be based upon the relation exhibited between the *products* of functional activity, such as the nature, quantity, and quality of the secretions in their several receptacles; and the *raw materials* upon which the organs have to work, such as the nature, quantity, and quality of the chyle, lymph, and blood. It would not be formed upon regard directed only to the condition of organs which had been either in activity or repose. It could not be so based, because the process of repair in the living, healthy body is one that is simultaneous and commensurate with waste. The muscles are not mended up as we mend a damaged wall, by patching up a hole here, and binding on an iron brace or girder yonder; but the process is interstitial; new material is brought in, and brought everywhere; the existing organ is worn down, and the waste matter is carried away; but, with all this change, there is a persistent textural result. Looking at this question still more closely, we see that function is related to structure, not only in the sense that it is what the organ does, but in the much more important meaning that it is at once the expression of the wear and also of the repair of tissues; or in other words the outcome of their life. In the present state of physiology, it is impossible to conceive of a living organ without believing in the nutritive, molecular changes it is undergoing; and these are the essential conditions of its functional activity: it is equally impossible to imagine the function of any living tissue being called into exercise without recognizing the dependence of this functional operation upon interstitial movements of repair and waste. But we should be wrong on the other side were we to confound function with the nutritive changes which constitute, not the function itself,

but the conditions of its exercise. It is, for example, the peculiar function of a muscle to shorten itself, of a nerve to convey an impulse either of motion or sensation, and of a nerve-centre to convert one of these impulses into the other; the organs referred to, in exercising these functions, undergo certain nutrition-changes; but these molecular changes are not the functions of the organs, but the conditions essential for their performance. This principle, which it seems almost unnecessary to state, in regard of the particular organs or tissues now referred to, is, however, not unfrequently lost sight of in respect of secreting organs. It is the function of the salivary gland to secrete a fluid having special characters; of the liver to do this, and to effect changes in the blood which comes to it; and so of other secreting organs: they receive blood into them, and from all of them it passes away, changed; and the organ, as part-product of this change, gives up its secreted matter. These functions, be its observed, depend for their performance upon nutrition-changes in the cells and tissues of the organs; but those fine processes of change are the conditions of functional activity, and are not to be confounded with the thing itself. The secreting cell has to live, to waste, and be repaired; and it lives at a degree of pressure, and is wasted and repaired at a rate, directly proportioned to the amount of work that it accomplishes; and thus it is conditioned precisely as are the ultimate elements of the muscle or the nerve. But minute as is our knowledge of much that goes on in the secreting organs, and of the chemical nature of the results or products of their work, we know no more of the physical conditions which determine that one set of cells shall separate urea, another set saliva, and a third bile acids from the blood, than we do of those which enable one nerve-fibre to convey impressions of light, another of sound, and another those of motion. These are, at present, ultimate facts of physiological science; the function is the expression of the life of the structure; it is what the latter was constructed for the purpose of doing; in doing it, the structure undergoes change; it is wasted and repaired, but these processes are carried on without any breach in the integrity of tissue. Function is to nutrition, as electricity is to the chemic changes in the galvanic battery, a "correlated force." We do not say that the one is the other, but that it is converted into it; and, as in the inorganic world, the arrangement and nature of the particular materials with which different forces come into contact, determine whether chemic action shall appear as heat or magnetism, whether heat shall be shown in motion, light, or electricity, so do the different materials of the living organs, and their arrangement, determine the nature of the functions they perform: how they do this, we do not know, but the facts of physiological science are well known, viz., that the nerve cell exhibits one class of powers, the muscular cell another class, and the secreting organs a third. 3d. A third class of facts to be remembered is, that in many diseases, the only symptoms to be recognized are changes in the degree of activity with which certain organs perform their functions. No new element is introduced by some diseases into the category of vital actions; such affections as chorea, hysteria, epilepsy, might be shown to consist of mere modifications in the degree, time of occurrence, and combinations of functions, each of which, taken *per se*, is consistent with health. The sudden loss of consciousness in epilepsy, for example, is not more mysterious than is the sudden but every-day recurring passage from wakefulness to sleep; the arrested respiration is similar in kind to that seen when the chest is fixed in the performance of any great muscular exertion involving the upper limbs; and still more similar to that which can scarcely be called morbid, the prolonged apnœa of a screaming child, whether the scream be the expression of terror, temper, or pain: the convulsive movements are neither more nor less than nerve and muscular functions, any of which might separately, and many of which might in combination, be the expression of healthy vital activity.

From these three classes of facts, therefore, we are compelled to admit that, in the present state of science, the *onus probandi* lies with those who assert the constant presence of structural in association with functional change; and we affirm that those who make the assertion have never proved their point. Further, that as a matter of inference from what we know of the relation subsisting between structure and function in health, we should not even expect to find solutions of continuity or coarse changes of texture in those diseases, the essential elements of which are functions altered, not in kind, but only in degree and mode of association; and that, on the other hand, when we do find material changes in association with functional disturbances, we should refer many of the latter only indirectly to what we see of the former, the more numerous and more important of them being dependent upon what we do not see, viz. the finer changes in the interstitial processes of nutrition. A scirrhous tumor of the stomach, for example, may produce certain symptoms easy explicable by its mechanical effects; it may be so situated as to prevent the ingress or egress of food; but vomiting may occur when the orifices are free, or when the tumor is situated in some organ in the

pelvis: the supposed tumor may cut off the supply of food, and so explain some of the changes we see in color and general nutrition; but, on the other hand, the extreme of wasting and of cancerous tinting may be seen when there is no such enforced abstinence from food, but when indeed a large quantity is not only taken, but is digested and enjoyed. So again a clot of blood in the corpus striatum may sever the nerve-fibres, and so explain the severance between the will and certain muscles of the extremities; but it will not so explain the presence of convulsions, or of spasms in those palsied limbs.

For these reasons we retain the words "functional disease;" understanding by them such changes as have no recognized morbid anatomy, but such as depend upon corresponding changes in the finer processes of nutrition. We do not believe that there is any altered function without a correlated change in the nutrition of the organ; but what we assert is that such a change, as a matter of fact, is of such kind as to be undiscoverable by our senses, and as a matter of inference, from what we know of the relation between nutrition and function, is of such nature that it may always be beyond the reach of observation. No healthy function is performed without nutrition-change; no morbid function can exist without altered nutrition-change; but the relation between the two elements, "structure" and "function," is the same in the two conditions.

Nothing is more erroneous than the common notion that "functional" means trivial, and that "structural" means severe. Many diseases, designated by the former word, are long-continued, obstinate, or destructive; many known by the latter are of short duration, are amenable to treatment, or are harmless. Diseases which spoil or shorten life are not trivial because they depend upon such fine changes as may escape our observation; but they are the more serious when they thus elude our notice, just because they have their place in the very centre—the most ultimate process and fact—of life, the conduct of nutrition.

NATURAL HISTORY OF DISEASE.—Under this phrase are recorded the symptoms or phenomena of disease, their causes, the manner of their development, their duration, and the different mode of their termination, whether the termination be in death, or in a return to health. In the natural history of disease no theory is involved; we have to deal only with facts.

Causes.—Commencing with that which precedes the appearance of symptoms, viz. the "causes" of disease, there are some principles which it is important to lay down; inasmuch as our ideas of the causation of disease are determined, in great measure, by our conceptions of what disease itself is. Nothing is more easy sometimes than the discovery of a cause, and its distinct separation from a particular and well-known malady. For example, a healthy child is brought into a room where some one is suffering from scarlet-fever, and after a certain period it exhibits symptoms of the same malady, and passes through all its stages. Here we say there was direct communication of the malady; but we must not forget two facts, first that some children so exposed do not take the fever, although they have not previously had the disease; and secondly, that those who have suffered from it once rarely take it again, although they may be exposed to infection. We suppose a constitutional disposition in one case, an indisposition in a second, and assert the existence of the latter in a third. Still, when the cause has operated we feel that we tread on safe ground when asserting broadly that the cause is "infection."

Nothing, however, is more difficult in some cases than to say what the cause of a disease has been. For example, six people take an indigestible meal, and one of them suffers nothing, a second is troubled with dyspepsia, a third with asthma, a fourth has an epileptic fit, a fifth an attack of gout, and a sixth is disturbed with diarrhœa. One element in the causation of all these maladies is the same, viz. an indigestible meal, but the results vary widely; and we say that this is owing to constitutional conditions which "predispose" to these particular affections. These have been called "predisposing causes;" but we must inquire what they are, and how they are related to these diseases, if we would understand the latter. Among the predisposing causes are reckoned hereditary taint, sex, age, and constitutional peculiarities, either congenital or acquired; and with regard to two of these, hereditary taint and constitutional peculiarity, we must admit that they are, in reality, disease. When we allow the existence of either, we but throw back a few steps further the line and widen the circle which includes all that we mean by the disease itself: we include in the malady more than its name expresses; we partially account for its occurrence, but do not explain its "cause." It is quite true that we may sometimes draw a line between certain so-called causes and effects; we may say, for example, this man, of tuberculous family and with latent tubercle, was yesterday apparently well, but he was exposed to cold, and to-day he has tubercular pneumonia; his constitutional state "predisposed" him to the evil, which the exposure "excited" into activity; but regarding him from a pathological point of view in his present position, that of a sufferer from tubercular pneumonia, we can-

not separate the elements of his disease so easily, for it was not a cause of his malady which was there before, but an integral part of the affection under which he is now laboring. A similar difficulty is to be encountered on almost every hand; the worry of the Stock Exchange is borne by some men bravely, others succumb, but variously, one goes mad, another is "broken down," a third becomes epileptic, and so on; and we fly to the resource of "predisposition," some weakness somewhere, which this wear and tear has pointed out, and urged into morbid activity; but in that very weakness, if there was not the whole of the disease called A, B, or C, there was some important element of it, and not its remote or predisposing "cause." If disease be, and we believe it must be, defined to be the sum total of changes in either structure or function, or both, then almost all of these so-called "predisposing causes" are part of the disease itself.

But those other conditions, sex and age, it may be asked, are they integral elements of disease, or are they its cause? *Sex* cannot be said, accurately, to be a cause of disease any more than the same can be affirmed of life, but yet, in all modern treatises on medicine, it figures in the chapter on etiology. Be it observed, however, that the word "sex" when thus used, has reference to many conditions of difference beyond those which exist in the reproductive organs. The male sex, as a rule, exhibits the masculine frame of body and of mind, while the female sex displays the opposite, and their respective "predispositions" to disease have relation to these characters as well as to the physical structure of the apparatus of reproduction. The existence of an organ is the necessary condition of its becoming diseased—a man cannot suffer from ovarian tumor, nor a woman from orchitis—but there are some organs common to the two sexes, and equally developed in each until the sexual distinctions of puberty are seen; then in the one they become rudimentary and inactive, whereas in the other they take on new and important functions. In these instances sex may be said to be a predisposing condition of disease, but it is so only in this sense, that it exposes certain organs to some of the causes of disease by the simple fact of their functional activity. It must not be supposed that the healthy action of any organ predisposes it to morbid change; on the contrary, it exerts rather a strengthening and protective influence; but such activity simply lays it open to the operation of influences which cannot be brought to bear upon its counterpart which remains in a rudimentary condition in the other sex. But besides these essential differences there are others which are accidental, and which depend upon education, practice, and habits of life, as these are determined by the customs, fashions, or peculiarities of the people and their times. And, further, there are many facts with regard to the relative proclivity of the sexes to special diseases which have not yet been explained by any known relation of these maladies to the reproductive organs. Simple ulcer of the stomach and carcinoma of the same organ afford examples of this difference of proclivity, and we at present have to regard them as ultimate facts of pathology.

Among the conditions which determine, therefore, the differences of sexual predisposition to disease we reckon (1) the actual presence or absence of the organs; (2) the action or almost absolute inaction of the organs; (3) the relation between the amount of activity of some functions of certain systems of organs, and other functions of the same systems, where differences exist *ab initio;* and (4) the effects of habit, education, and fashion, in either producing or lessening functional activity, with all its correlated structural conditions.

In a similar manner must we regard the influence of *age* in the production of disease. It is not *per se* a predisposing cause, but it carries with it certain things which may be. If we can conceive of a perfectly healthy organism, placed in absolutely healthy conditions, then we may believe that it would pass through the stages of growth, dentition, puberty, adult life, and decline; and that it would perform all the functions of self-preservation and reproduction without either hurt or hindrance; sometimes, nay, very often, we do actually see some of these stages passed through with as entire a freedom from discomfort as any ideal being placed in the most Utopian circumstances could wish for; but at some point or another in the long course of life, the chain of good succession is broken by a faulty link or an unexpectedly heavy blow, and then follow one or another of the many ills that make up the miseries of common life, and average health. On the other hand, we see cases in which nothing seems to be capable of going well; every epoch, every change, every organ, seems, as it were, pounced upon at every turn by all the evil agencies that surround it; action or inaction, growth or decay—it matters not which—seems exaggerated into a condition of disease, and life is a lifelong misery. It is clear that no one period of life is in itself a cause of disease, or of exemption from its occurrence, since all periods may be passed through without any disturbance of the health, and no period possesses absolute immunity from its attack. But it is also evident that the changes which take place at certain periods, render some individuals liable to the operation

of other causes, and that this operation may be very effectual in the production of disease. Such periods are those of rapid structural development, and the commencement of new functional activities, or the decline of structure and the arrest of action. That which would seem to be the condition tending to morbid development is the disturbance of the balance of activity and growth between different organs, or systems of organs, so that for a time at least an undue preponderance is given to a certain set. Thus during the first dentition, although there is general growth, there is particular activity of certain organs; and, in like manner, at the commencement of puberty, there is, as it were, undue prominence given to the organs effecting reproduction; and although the particular structures involved in these developmental changes may not be selected as the localities for morbid action, the very fact of their disproportionate activity—by a disturbing balance of general nutritive progression—may become a cause of derangement in other systems of organs, such as the vascular or nervous. In like manner, at the climacteric period, the repression of certain functions, to the operation of which the whole body has become habituated through a long series of years, may prove itself the starting-point of morbid changes in the functions of other organs which are, by this repression, placed in new and trying circumstances. By these considerations we may, for the most part, explain the influence, where it is marked to the highest degree, of age as a so-called "predisposing cause" of disease.

But there are some maladies which appear to have a definite relation to age, and which are not explicable upon these principles; and with regard to these we must admit that certain periods of life are associated with proclivity to disturbances of particular kinds, the nature of the relation between the two classes of conditions being at present unrecognized by medical science. At the same time the history of all science is such that it teaches us to believe that these relations will be hereafter discovered and found to be analogous to those which we already understand and appreciate.

The term "exciting cause" of disease has been applied to another class of condition altogether, and one with regard to which there is much less difficulty. That which is involved in the term is the operation of some influence from without, be that such as to act upon mind, emotion, sensation, nutrition, vascular conditions, temperature, or any other function or property of the living body. A great surprise, or an overwork, may affect the mind; a domestic calamity, or the worry of business, may disturb the emotional centres; a physical injury may set up changes in sensation; a forced rest may weaken muscular nutrition; a tight bandage, or an altered position, may influence the vascular supply; cold or heat may diminish or increase the temperature of parts; and in like manner other agencies may affect the organism, and become the exciting causes of disease. The form the latter take is determined partly by the nature of the exciting cause, and its relation to this or that system of organs; and partly by the condition of the organism, in regard of hereditarily received or congenitally acquired morbid state, the peculiarities of sex, and the period of life.

Two things therefore concur to make up what we term the causes of disease, but they are essentially distinct, and it would be better to denote them by different names, than by one name differently qualified. The one, the so-called "predisposing cause," is a "diseased condition," and is therefore a part of the disease itself; the other has no necessary relation to the individual or his constitution, and is in reality a "cause" of the disease from which he is suffering.

Symptoms of Disease.—The meaning which now we must assign to the word "symptom" or "sign" of disease, is very different from that which some time ago would be conveyed by those terms. So long as disease was regarded as something put into, added to, or engrafted upon the body; a material, or other entity,—having even a more or less substantive existence,—these words described the means by which we might recognize the presence of such an entity: but so soon as disease is recognized to be, as we have defined it, the sum of changes in function and structure presented by the living being, the words "symptom" and "sign" have another meaning, and describe only those parts of the disease which are appreciable by others. Disease is a complex state of a complicated organism, and although the *name* which we may give it may be intended to express its primary or most important fact, we cannot separate this one fact from others with which it is associated, but must regard them as integral parts of the malady we have either to study or to treat. They may differ, from an outside point of view, in proximity of relationship; but the heat of skin, the altered pulse-respiration ratio, the nature of the expectoration, the changes in the secretions, in the nervous system, and in the prospects of life, together with the altered resonance, breath and voice-sounds, are as much parts of the disease called "pneumonia" as are the structural condition of the lung. Some of them may be signs by which we recognize its presence; but they are also essential elements of the malady itself. In like manner it might

be shown with regard to those other diseases, the nomenclature of which differs as we have already described, that a precisely similar relation exists between what we have denominated "symptoms" and what we understand by "disease." We cannot know of the existence, during life, of any disease except by its symptoms; we cannot conceive of disease apart from some recognizable changes in either function or structure; and these changes constitute the disease: nor can we, on the other hand, imagine the existence of what we call "symptoms" apart from the correlative idea of what we conceive to be "disease." The two classes of notion have been, of necessity, distinct in their development; but the maintenance of the distinction between them has been a hindrance to true progress in pathology; and it will be well for us to try and remove that hindrance. So long as "disease" is thought of as a something—it matters not what—distinct from the "phenomena," or "symptoms," by which it makes itself known, so long are we in danger of mistaking its real meaning, and of overlooking those true guides towards the removal or alleviation of its evil, an end to which all medical science ultimately points.

"Disease," we have said, is a change of structure, or of function, or of both; "symptoms" are those changes in structure or function, or both, which we can recognize. The latter, the symptoms, are not separate from, but are parts of the disease, and their only characteristic is that they are such parts as are appreciable during life. But they differ in kind and in value among themselves, and have been known by different names, so that we meet with such distinctions as those between "signs" and "symptoms," between "objective" and "subjective" symptoms; and between "general" and "local" symptoms. These terms almost explain themselves, and are retained because they possess a certain amount of utility; but it is more easy to make use of them in practice than to define their exact meaning in the abstract. The idea underlying the word "sign" is that it is some physical change which can be observed directly; and thus we speak of dulness on percussion, tubular breathing, augmented vocal fremitus, and bronchophony, as "physical signs" of condensation of the lung; whereas we speak of dyspnœa, expectoration, and fever as "symptoms" of pneumonia, or tuberculosis. But heat of skin, an eruption on its surface, the wasting of a muscle, or the fact and quality of a secretion, are as much "physical signs" of a disease as are the particular phenomena we have mentioned. Yet it is almost unknown, it is certainly very unusual, to hear the term "physical sign" applied to any of them.

It is easy to trace the origin of this distinction between symptoms and signs to the period when the physical examination of the chest arrived at its due position. It was felt that an amount of precision in diagnosis was arrived at by percussion and auscultation, such as was never dreamed of in the times before such modes of exploration were employed; and therefore the conditions revealed by their aid were expressed in terms differing from those which had been previously employed, to describe such changes as short-breathing, pain, expectoration, and the like. But it is impossible to maintain the distinction; the number of respirations per minute is as much a "sign" as is the dulness on percussion, and both of them may be "symptoms" of disease. Everything that may be observed is both the one and the other, and the reason why the distinction has been maintained is, because with regard to the former—the "sign"—there may be but one means for its recognition, namely, observation from without; whereas with regard to the latter—the "symptom"—there is a possibility of confounding two things essentially distinct, namely, the sensations of the patient, and the phenomena he presents. Deficiency of resonance is discovered only by percussion; but short-breathing is a sensation of which the patient complains, as well as a phenomenon which may be appreciated and measured by the physician; and in like manner wasting of a limb may make itself at once evident to the observer, whereas paralysis is a condition which the patient may assume.

The really valuable element of distinction between these two classes of phenomena is therefore that which exists between "objective" and "subjective" symptoms; meaning, by the former word, all those elements of disease which can be appreciated by the senses of the observer, and by the latter, those which can only be known through the statements of the patient. Whatever of disease comes to us only through the mind or feelings of the patient, as expressed by language, either of gesture or words, is a "subjective" symptom. Its form and its degree of intensity are subjected to the conditions of the machinery—mental, emotional, and sensational—through which it passes, and it is liable therefore to be changed, either in character or degree. Whatever of disease comes to us through our own senses alone escapes this danger of addition, alteration, or subtraction, and is *pro tanto* of higher value. In one class the patient gives his version of his case; the symptoms are such as we can only get at through his mind, and they are termed "subjective;" in the other, we make our own observations—the feelings or ideas of

the sufferer have nothing to do with them—simply, physical facts are the objects we recognize, and such symptoms are called "objective."

As a general rule the objective are much more valuable than the subjective symptoms; but let it be remembered that the importance of the latter is very widely variable, and that sometimes it may far exceed anything that can be derived from direct observation. In the early stages of some serious diseases of the heart or brain, nothing may be presented to the practised ear or eye; and yet the patient tells us of a deep unrest, or sudden horror, which, although it has no objective sign, may be the herald of a sudden or lingering disease; as true and as important—although to others the mind seems clear, and the heart's beat healthy—as any murmur we might hear with the stethoscope, or any palsy we might measure by the hand. We have to deal with man as a whole; and to ignore or undervalue what he tells us of his ideas, emotions, or sensations, because they may be termed "subjective symptoms," and be held to be therefore unreliable, would be to shut out from ourselves that which—egotistic and fearful, prejudiced and ignorant as man may be—yet forms an integral part of his life, and therefore of his disease. We must be careful to give to both groups of symptoms their true value, and our danger in the present day is to underrate the importance of those which, a few years ago, constituted almost the total symptomatology of disease.

The distinction between "general" and "local" symptoms need not detain us, since the terms are obvious in their meaning, and the difference between them is gradually dying out by the recognition of the fact, that no one organ can have its functions or its structure changed without the existence of some relative change in all the rest.

Course.—In describing some few diseases we have little more to do than to detail the phenomena present at any one given time; whereas in furnishing the natural history of others we are compelled to speak of premonitory symptoms, or prodromata, of the modes of commencement of the illnesses, their forms of attack or their *début*, and of the different stages—two, three, or more—through which they pass. Between these two extremes we have every amount of variation: the natural history of one disease may be compressed into a sentence; that of another may expand into a book; but more commonly we have to deal with histories intermediate in duration, and perhaps less emphatic in their interest.

It often happens, however, that the mode in which one event follows another is of great importance in the diagnosis of disease. For example, the decumbency in pleurisy has different meanings at the commencement of the malady, and at its later stages; and in like manner rigid muscles teach one thing at the onset of a paralytic seizure, another when the immediate effects are passing off and the patient is regaining power; and still a third when without such restoration it makes its appearance at a yet later period, and in a well-known order.

Not merely the mode of sequence, but the actual time of sequence, is of diagnostic and therapeutic value; and this we know full well in the study and recognition of the acute specific diseases, of malarial fevers, and the like; and with equal significance, though with less accuracy of measurement, can we use the element of time in the diagnosis of many chronic diseases; and such element may sometimes be sufficient to determine a question left wholly in the dark by other elements which we have had before us. In this manner time enters occasionally into the diagnosis of tubercle, of cancer, of hemorrhage, and of other maladies; and it would be difficult to overrate its value in those special cases which are left in obscurity by the absence of special or pathognomonic symptoms.

Again, the relative intensity of symptoms is a point to which attention must be frequently directed, for by this alone a diagnosis may be possible. If we regarded the relative intensity of pain, dyspnœa, fever, cough, and general nutrition-change, each of which might be present in bronchitis, pneumonia, pleurisy, phthisis, or asthma, it might be possible to say which disease existed, and this without the aid of auscultation or percussion. And in like manner by regarding the relative amount of coma, paralysis, spasm, and rigidity, it would be possible to distinguish, in some cases, between cerebral hemorrhage, acute softening of the brain, congestion of the brain, or urinæmia.

All these particulars have to be described under the head "course" of symptoms; and they form an essential part of the natural history of disease.

Duration.—The importance of a consideration of time in the diagnosis of some classes of disease has already been referred to; it is of no less importance when, diagnosis being determined, a knowledge of the natural history of disease gives us power to foretell, with some approximation to accuracy, its probable results.

We know approximately the duration of herpes zoster, of vaccine, or variolous pustules, of typhus, or scarlet fever; and we know also, approximately, the duration of phthisis pulmonalis, of carcinoma ventriculi, and of other maladies. With regard to the latter—the chronic class—our knowledge is much less definite, or

rather it lies within a larger range, and is of less practical utility. It may, regarded from a pathological point of view, be even more accurate than that which we boast of in respect of the acute diseases; for the relative range of variation is not greater, although in the one case we deal with days or even hours, and in the other with months or years. But life is on one side of the balance, and death is on the other, and the balance is struck between these two, whether the beam be long or short; and so we congratulate ourselves upon a readily used and readily appreciated forecasting of the immediate, present evil, and perhaps underrate our sounder knowledge, with regard to that which may not happen for ten, twenty, or a hundred months.

The duration of symptoms, therefore, though of variable social value, is of vast interest in the natural history of disease

Termination.—A disease may end in various ways: (1) The patient may gradually get rid of it altogether. (2) He may lose all the urgent symptoms—all that at one time seemed to constitute the malady—and yet retain some less urgent symptoms; or some which are not recognized at all; or some which—so far as the patient's own feelings are concerned—are not, even after recovery, recognizable, but which may be discovered by a physical examination of the organs instituted by the physician. (3) He may continue to present all the local disturbances, while the general or constitutional changes pass away, and thus the disease persists, but in an altered and what is called a "chronic" form; or (4) the disease may become worse, pass from one stage of weakness to another, and end by the destruction of the patient, which may be brought about in various ways. Now, all these points in the natural history of disease, are of much interest in regard of prognosis and of treatment. The recognition of early signs of recovery is a great help towards the choice between therapeutic means, and is a good ground for employing one of the strongest of these means of cure, viz., hope. The search for and discovery of the effects of an acute illness—although in that acute form it has passed away—are of great importance to the life, prospects, work and career of the unconscious sufferer. We must know these probable consequences if we would perform our duties as the advisers of those who place themselves under our care. It is not enough to guide a disease to an apparently successful issue; we must know what weak point it may have left behind, and we must guard this with the utmost caution. Again, the tendency to pass into a chronic form often reveals the existence of some constitutional vice we had not before suspected, and thus renders itself available for a more effective direction of our therapeutic agencies. And, lastly, the perception of the early indications of a fatal issue may be of great social or individual value; whereas the recognition of the mode in which death threatens to approach, may be the means of teaching us to select such measures as shall temporarily, or even indefinitely, postpone the evil. Thus the "terminations" of disease, whether they be in health, in impaired health, or in death, are not only of interest to the natural historian and the pathologist, but are full of teaching to him whose aim is to render natural history and pathology subservient to the great work of healing diseases, of relieving them, or of measuring their duration, and lessening the pain with which they do their work.

Diagnosis of Disease.—In the earlier days of medical science, the problem of diagnosis might have been stated thus: "given the symptoms, to find the disease;" but, in these days, such problem must be translated into the following terms: "given some of the elements of disease, to discover the others." We do not now regard eruptions on the skin, peculiar changes in the mucous membrane of the throat, an elevated temperature, and a disturbed innervation as the signs by which some morbid "entity" in the body reveals its presence; but as parts of the morbid condition of the organism, from which we may infer the existence of simultaneous changes in stomach, intestines, liver, spleen, or kidneys. The rose rash of typhoid is as much a part of the disease as is the ulceration of Peyer's glands; the dulness on percussion, the altered condition of the urine, the changed nervous power, are as truly elements of the disease called pneumonia as are the minute alterations which constitute inflammation of the lung. Some of the elements of disease escape our observation during life; some are directly perceived by us; and others are appreciated indirectly by such processes as percussion, chemical examination, laryngoscopy, spirometry, and the like: but the only difference between these is their relation to the observer; they occupy precisely the same position in regard to the disease itself. The process of diagnosis therefore is the passage, not from effects to causes, not from phenomena to noumena, for, strictly speaking, we do not at all know what are either the causes or the noumena of disease; but diagnosis is the process by which, perceiving some particulars, we infer the existence of others, which we know to be commonly associated with them. This is however the pathologic or scientific side of diagnosis; and the question may still be asked, What is it practi-

cally? Practically, diagnosis is the process by which to a certain set of symptoms we affix a more or less familiar name; and become able to say that such a one is suffering from typhoid fever, meningitis, hooping-cough, epilepsy, or gout. And when we have given some names to diseases, we have, more or less intentionally and with differing degrees of accuracy, conveyed some theory into the process of diagnosis. Remembering what was said with regard to "names of disease," we shall find that, in this practical sense, diagnosis is sometimes the discovery of and the calling of a disease by what we conceive to be the most important structural change by which it is accompanied, such as pneumonia; at other times diagnosis stops short at the recognition of a pathognomonic symptom, such as hooping-cough; again, it may mean to express the starting-point or principal fact in a group of symptoms; or some general condition but imperfectly understood, or some condition which is not understood at all. In all of these instances we notice two things: first, that diagnosis—looked at from its scientific side—is the step from particulars to particulars, from those which are observed to those which during life can only be inferred; the step from the one or the one hundred to the whole; second, that diagnosis—regarded practically—is the giving of a name to the disease from which an individual suffers; the ticketing, and as it were placing of that malady in some niche with others that resemble it, so that it may be known and brought out when required. But let it be remembered that the principles upon which names are constructed are so various that no expression can be framed to convey their meaning, in so far forth as that it should carry further what is meant by diagnosis.

Pathology.—When a patient presents himself for examination or for treatment, he tells us of certain things that he feels or sees, which are wrong, and which we call "symptoms;" we ask him questions, and learn additional facts of a similar class; we observe him, and notice other facts which he can neither feel nor know by independent means, and these we call "signs," or "objective symptoms." From what we are informed, and from what we observe, we pass on to the inference of other facts: we believe, when some three, four, or more changes from the healthy state are present, that other alterations exist; and this belief is more or less strong, and its character more or less definite, according to the state of medical science, and our knowledge of it, at the time that the patient presents himself. We *infer* certain things, certain conditions which we cannot see, but which we may, in some measure, classify, and therefore call by distinctive names. Thus heat of skin is a symptom from which we infer a number of ulterior conditions. The reason for this heat of skin may—other symptoms being regarded—be conjectured by us to be an altered blood-state, such as typhoid fever, tuberculosis, or the like: it may, on other symptomatic evidence, be referred to a change in some particular organ, such as inflammation of the lung, or destruction of a portion of the spinal cord: and we may give names to the general conditions, part of which we have observed, part of which we have inferred. This is what we call "diagnosis" in its practical sense; but that which makes diagnosis possible is the existence of a certain amount of information about the relations subsisting between different organs, and about the modes in which particular organs may become altered in function or in structure. We observe a change in the quantity, color, or other qualities of a certain secretion, and we infer from this that there is diminished, increased, or perverted action of a certain organ. We examine further, we discover other changes, and we infer the nature of the disease which that organ has undergone, or through which it is now passing. What in this sense is true of one organ is, more or less, true of all, and of the organism as a whole, so that we are able to give some general expression to its general state; and thus we speak of paralysis, fever, anæmia, suppression of urine, weakness, etc. etc., and so advance from symptoms—by diagnosis—to pathology. This is what we do in particular cases; finding certain symptoms, and knowing certain principles, we place the individual who presents those symptoms in a particular category, and call his disease by a name that is understood: and in this way the process of thought which we call "diagnosis" is a bridge across from "symptoms" to "pathology." Diagnosis is, then, the practical application of what we know about pathology; and its accuracy will be in proportion to the amount of our information, and to the keenness and readiness of our observing power. Pathology, therefore, is the foundation, the essential condition of diagnosis; for it would be simply impossible to advance, from the observation of symptoms, one step towards the recognition of the nature of disease, unless that step were based upon pathology; *i. e.*, upon a knowledge of the conditions under which morbid changes occur in structure and in function, and also of the modes in which such changes may so affect organs as to render themselves appreciable during life. Practically, as cases come before us, we observe symptoms and we diagnosticate pathological conditions; but we could form no diagnosis without pathologic knowledge, and diagnosis is in reality but

a portion of, or the practical application of pathology. Theoretically, however, pathology is the groundwork of diagnosis; and not only so, but of all valuable and correct observation: and thus the one is seen to be but the necessary complement of the other in the practice of physic. The one is a science, the other is an art: the latter could not exist without the former; the former grows daily by the application of the latter.

By Pathology, then, we mean the general doctrine of disease, the knowledge of the conditions under which it occurs, and of the kind of change which it expresses in the functions of the body. We mean also the inter-relations of different organs, or systems of organs, whether these may be compensative, or sympathetic, or antagonistic of one another. By the pathology of a disease we mean the scientific classification and nomenclature of its phenomena, and the interpretation of the conditions under which they have arisen. Pathology is to the body, under diseased conditions, what physiology is to the healthy organism, viz. the law of its being, or rather the best expression that we can give to what we believe that law to be. In its detail it must embrace all changes in either structure or in function; but in its common and general acceptation it is used to express the idea that is entertained of the primary or essential or most important change in both; and the bearing that this has directly upon life as a whole is, in its statement, the unravelling of the problem of what we ordinarily mean by "Pathology."

Pathological Anatomy.—Structural changes in some organs are so constantly and definitely related to functional alterations observed during life that "Pathological Anatomy" in some maladies constitutes the most important, if not the major, part of their pathology. There are other cases in which structure-changes cannot be shown so to account for all the detail of symptoms; and in these, pathological anatomy has its interest, but is of less immediate value. And further, there are diseases with regard to which pathological anatomy has hitherto taught nothing that is worth our knowing; while, on the other hand, it has not seldom exhibited the existence of disease, never suspected during life, and yet dire enough to be the cause of death, and thus to show that our diagnostic powers are not such, that we may boast of them.

Unquestioned as is the value of Pathological Anatomy, it is no less unquestionable that this value may be overrated and misapplied; and it is possible that such mistake may exist at the present day. Let us, therefore, remember what Pathological Anatomy is, and what it teaches. It is the condition of an organ after death, when tissues are cold, ordinary chemic changes have begun, secretion and living motion have ceased, circulation has stopped, and all that we know to be unexplained by ordinary physical conditions, and that we call "vital," has gone, and gone we know not whither, and know not how. Pathology, on the other hand, refers to the conditions and modes of action of the organs when they are warm; when chemic changes seem to be directed by a power that cannot yet be explained by chemic force; when sensation and motion are constantly placing the organ in new positions, in regard to other organs, and in new conditions, in so far as relates to their own integral parts; when the blood is constantly flowing, and affecting. as it does so, both the tissues and itself, not leaving them as it found them, not leaving them as it came; when all that we understand, and all that we dimly guess at about that complex force or principle, combination of forces, condition or what-not, that we call "life," is the director or harmonizer of all we see, or is engaged in what seems a fierce struggle with powers it has long directed, but which, having now risen in defiance of its authority, seem likely to accomplish its dethronement.

Everything that we can see is altered by that change from life to death; and Pathological Anatomy has to do directly with physical conditions, which may be partially causes, partially effects, partially conditions of, and partially unrelated to the disease of which they are but a part, although an important one. If everything that we can see is thus altered in the step that all bodies make before they come under the scalpel or the microscope of the pathologist, still more altered is almost everything that we cannot see, but which we have inferred to exist from our laborious physiological investigations. It would seem therefore that Pathological Anatomy is about as capable of furnishing, *per se*, a notion of disease as dissection would be of teaching physiology; and if we are to exalt into such undue prominence—as it has been the fashion of late years to do—this branch of science, which is yet only a branch and not the science itself, it would be nothing more than consistent to attempt to write biographies from post-mortem examination and to construct the history of a nation by exhuming the bodies from its graveyards.

Pathological Anatomy has reference to one element of disease—structural or tissue change—and that only under conditions very different from those which exist during life, and when only disease can be said to exist. Further, we get the materials for examination when disease has passed through all its stages, and through that final one which cannot be said to

form a part of either life or of disease. It is only when, by accident or intercurrent malady, a patient is struck down in the earlier stages of morbid change, that we have the opportunity for observing the kind of alteration which exists at such most important periods; and even to them is added all that we have already described, and probably much more than we can even guess at, by the great fact of death. Disease, being a complex of change in function and in structure, is represented anatomically only by the latter: disease, having a history, and passing through stages, is shown to us only in one stage, and with infinite predominance of frequency in the last stage: disease, being a change in the conditions of life, is shown to us anatomically only in the condition of death: while General Pathology, therefore, may embrace all that we know or can know of the mechanism of human suffering. Pathological Anatomy but touches the human body when the period of suffering has passed.

But the facts revealed by a study of Pathological Anatomy have a peculiar kind and a high degree of value, which we would most unwillingly underrate. There is an intimate and constant relation between function and structure (see p. 19); and we believe that, although certain structural conditions have yet escaped our observation, they may hereafter be rendered patent to the senses of the observer; and on the other hand, we hold that there are some structural changes which may always transcend our powers for their immediate recognition; but, notwithstanding these facts and principles, we must remember that where anatomical changes have been discovered and verified, they have thrown unexpected light upon previously dark and complicated problems; and that they have possessed—in themselves and by their very nature—a definiteness, or precision, and have been susceptible of description and measurement to a degree that is quite unattainable by our present modes of research in regard of functional change. In Pathological Anatomy, what we have, however little it may be, is definite, describable, demonstrable, and measurable. These characters constitute its great value; and it is against its undue exaltation—and not its proper use—that we raise our protest. Let us be as exact in our inferences from its facts as we may be in our observation and description of them, and then Pathological Anatomy will take its proper place, and we shall learn from it its most useful lessons.

PROGNOSIS.—The practical test of a true science is the power which it confers of "prevision," or of knowing now what will follow hereafter. Some sciences have attained to this point, as we see daily illustrated by physics and chemistry; but as yet medical science has arrived at only very partial security of forecast. And yet the fore-knowledge of the consequences of a present disease is that for which patients and their friends often seek from the physician with the greatest eagerness. When we can prognosticate with certainty, medicine will have become a "science." At present we only, with different degrees of nearness, approach this end. We may describe the "probabilities" of a given disease; we may even measure them; we may accept or reject lives at insurance offices; or we may affix a numerical value to their duration;[1] but we deal with doubts, and not with certainties. Life is too subtle for us to know or measure all its possible contingencies; and our information is too scanty to render us thoroughly satisfactory interpreters of the outcome of any malady. But, with all this doubt, much may be accomplished for the safety of society, and the relief of individual anxiety or care.

In prognosis we have almost always two ends to be considered: the immediate effects of the present illness, and its remote consequences upon life. We have also two main elements by which we are guided in judging of these ends, viz.: the local changes which we may directly appreciate, and the general conditions which we may infer to underlie them, or to be, in some way, their consequence.

Prognosis with regard to the immediate effects of a present malady is guided mainly by the degree to which it interferes with any or all of the great vital functions, the circulation, respiration, innervation, or nutrition of the body. Prognosis in regard of the ultimate issue of a malady is based upon all these, but still more frequently upon the recognition of changes, often minute, in either function or structure, which our knowledge of pathology leads us to interpret for either good or evil. For example, a man is suffering from pneumonia, and we may—finding his general functions performed with an amount of ease consistent with life—infer that he will recover from the attack which to other eyes may appear most threatening; but we may discover physical signs of tubercular disease—slight, even unnoticed previously—and these teach us that the ultimate prognosis is unfavorable. In like manner, a child may be taken with convulsions which appear as frightful as only convulsions can do, and yet we may give a favorable opinion as to the present illness; while, on the other hand, some much slighter convulsive movement accompanied by heat of skin, a variable pulse, an obstinate

[1] See pp. 25, 26, on the duration and termination of diseases.

vomiting and constipation, and a history of failing health, or of hereditary tubercular taint, may lead us, in the midst of what seems a trifling malady, to augur the worst results from what we believe to be the onset of tubercular meningitis.

In prognosis, therefore, we must bear in mind the two objects we have set before us, and the two classes of means by which we may advance towards them. The immediate prognosis turns upon the degree to which great vital functions are interfered with: the remote depends upon the nature of slighter changes, of which pathology teaches us the meaning.

Therapeutics and Hygienics.—In the prevention or treatment of a disease our science culminates and becomes an art. Unless it can accomplish one or the other of these ends, the world would do as well without as with our aid. It is of some value to know the probabilities of our state, but it is of comparatively small value to have this knowledge if we can do nothing either to ward off, alleviate, or cure disease. We may prepare some people for the worst, we may dispel some groundless fears; but our mission is to do more than this: we have to try to "cure the curable, and comfort the incurable."

In the prevention of disease regard is had to the condition in which the individual is placed, to his hereditary or acquired constitutional peculiarities, and to the minute physical or functional departures from health which we may discover. In the treatment of disease no one of these can be lost sight of with impunity, but we are guided principally by the actual symptoms present at the time. These symptoms, however, it must be remembered, are of two orders: from the one set we learn the actual physical condition of an organ, or group of organs, such as inflammation of a lung, congestion of the liver, or paralysis of one side of the body; while from the other series we become acquainted with the state of the system generally, whether this be antecedent to the local change, its cause, its effect, or a mere coincidence of its existence; and from such a group of symptoms we infer the existence of dyscrasiæ; such as tuberculosis, carcinoma, or the like, and appreciate the presence of vigor, or of asthenia, of sound constitution, or of impaired health and wasted strength.

To the most superficial observer it must be obvious that therapeutics has undergone great and important changes, that the mode of treatment now adopted for many diseases is just the opposite of that which was in vogue a generation ago, and which lingers even in recent editions of standard books, although their authors have long since ceased to follow the directions which they still give to others. A few years ago the treatment of inflammation of an important organ was laid down definitely; such and such things were to be done, and no questions were to be asked as to whether the case was of this, that, or the other type. Inflammation was there, and blood was to be taken; low diet was to be enjoined, and lowering medicines were to be exhibited; and supposing the inflammation did not yield, the forces of attack were to be again placed in action: but here evidently there crept in some distrust of the theory at the bottom of the practice; for, instead of general bleeding, leeches or cupping were to be employed, and then only to a mild degree. Somehow or another the inflammation was to be put down, and it not rarely happened that the process urged against the bugbear "inflammation" proved fatal or highly injurious to the patient. If we can, by bleeding, and by it alone, save the eyesight which may be threatened by iritis, or if we can by depletion save a life which is endangered by laryngitis, we are quite justified in adopting that measure, although it may entail some injurious consequences. It would, however, be as unkind as it would be unphilosophical to relieve the pain of a simple pleurisy by abstracting blood, in such amount as should damage the individual in after years, when equal relief might be obtained by poultices and patience. We still find it written, if these conditions are found — a hard, full, strong, frequent pulse, with great heat of skin, no prostration, impending evil from this condition being patent as the phenomena themselves — then bleeding, antiphlogistics, and the like must be employed. But, as a matter of fact, we do not find these cases, and the more common *on dit* of medical practice is to the effect that as the inflammation seemed extending, the quantity of wine has been doubled, the supplies of beef-tea increased, and bark and ammonia given more frequently. Partly to account for, and partly to justify, so material a change in our modes of dealing with disease, it has been assumed that the *vis vitæ* of the British constitution has been lessened, or that the so-called "type" of its maladies has altered; an assumption which has little to be said in its defence, and still less that can be regarded as its establishment. A more simple, and we believe accurate, explanation of the change is to be found in this, that previously theory was the groundwork of therapeutics, and that now fact is the basis of treatment: that, years ago, diseases were treated by their names, and that now they are treated by their known conditions: that local changes were the main guides in times gone by, but that the general state of the patient is that which in these days the physician esteems

as his therapeutical informant. When pathology scarcely existed, medical practice was an empirical art; and had, with the few advantages of that position, all its evils: whereas, with the growth of pathology, therapeutics, still an art, has become, or is becoming, a science; and, knowing more accurately the limits of its powers, is content to attempt less heroic measures, being convinced that it does less harm. Much is done by medical treatment now, more real good than ever was done before, but it is done in a different way, and with another aim. Disease is detected in its earlier stages, and often arrested there; and when developed the patient is guided through it, if he can be, and is not sacrificed to some wild attempt at its destruction.

What we now believe and act upon is no set theory regarding the nature of particular diseases, or disease in general—modern times have not been devoid of theories upon which the fathers of medicine would justly have turned their backs in derision; but such notions, although they may have misguided a few individuals, have soon found their proper place, or no place at all, in the science of the day—what we do believe and act upon is a better knowledge of the laws and relations of morbid change: when we see that the man is greater than his maladies; that his general condition is of more importance than his local ailments; that disease is a change in him rather than in some part of him; and that no treatment can be of any real service which sacrifices the greater to the less. In all treatment, therefore, what is general is to be dealt with upon the basis of a true appreciation of the general pathological condition, and this in spite of all theories in regard of local changes, however they may be termed, whether they come to us with names hoary with age, or scarcely intelligible, and even sometimes ludicrous from their novelty. If the general condition be one of weakness, it matters not that the brain, the heart, or the lungs may be in a state of so-called "inflammation;" the weakness is the one thing that demands immediate treatment, and to neglect its treatment is to run the risk of sacrificing the patient to a theory of a compound state even now but imperfectly understood. This is the starting-point, the essential element in therapeutics; but the mode in which the treatment should be applied will often be determined by the nature and position and origin of the special lesion; and these conditions of the latter will direct the management of those means and appliances which, employed locally, will prove of service to the injured organ.

[While the view above given of the therapeutics of inflammation may be regarded as corresponding with the actual practice of a large number of physicians at the present time, in Great Britain and Ireland, on the European continent, and in America, there is reason to believe that more practitioners will partially dissent from it now (1879) than would have been the case ten or twelve years ago. Certainly it is a sound principle, not only to disregard, but to annul, in practical books and teaching, all merely traditional or "theoretical" precepts, which either improved pathology or extended experience has shown to have been erroneous. But, was it only, or chiefly, *local* changes, that were the guides of Sydenham, Cullen, Rush, and others in the old days of the lancet and other "antiphlogistic" measures? The pulse, the skin, the period of the attack, and the absence of evidence of exhaustion of the recuperative energy of the system; these, rather than merely organic conditions, determined, with them, whether or not depletion should be resorted to. It would seem to be a change in the *interpretation* of general indications, and a *different theory* of therapeutics, that have introduced, instead of the somewhat overdone antiphlogistic measures of our predecessors, the expectancy with some, and the stimulism with others, that have chiefly characterized the middle portion of the present century, in general practice.

That some important qualification of the view set forth in the above paragraphs would now be approved by a considerable number of the most eminent practitioners, might be shown by many citations. It is illustrated by some examples in the work to which this essay furnishes so able and fitting an Introduction. Thus, W. Squire, L.R.C.P., advises, in croup, under certain circumstances, "a free abstraction of blood." J. Hughlings-Jackson, M.D., in the instance of repeated convulsions, thinks that "we neglect to bleed as often as we ought to do, on the principle Markham has laid down." J. Spence Ramskill, M.D., in regard to the treatment of simple meningitis, refers to three great remedial measures, of which the first is bloodletting. Dr. J. S. Bristowe speaks well of the same remedy in certain cases of enteritis; and Dr. J. S. Wardell designates it as our best ally in sthenic acute peritonitis.

The intention of these remarks is not to antagonize, but to qualify, the summary conclusion which the language of Dr. Reynolds appears to convey, that venesection and kindred measures of treatment may be with advantage dismissed as obsolete procedures. Of names not yet antiquated, in favor of the *occasional* and *moderate* use of the lancet, in the *early stage* of acute inflammatory disorders, it may suffice to add here as examples those of Aitken and B. W. Richardson in Eng-

land; Niemeyer and Wunderlich in Germany; Jaccoud, Hérard, and Cornil in France; S. D. Gross and Fordyce Barker in America.

The most important qualification of the expressions upon which these comments are made is, in regard to "weakness" being the "one thing that demands immediate treatment;" this being "the starting-point, the essential element in therapeutics." Few, if any, pathologists will hesitate to admit that a difference, often momentous, exists, between the *exhaustion* of a system weakened by the continuance for some time of severe disease, and that *oppressive* debility with which the most robust person may be temporarily affected, under the influence of an acute malady, such as pneumonia, meningitis, or croup.

Those physicians who (like the present writer) were trained under the "antiphlogistic" régime, and had the opportunity of seeing something of its clinical results, which they may compare with those of the last two decades under a different prevailing practice, will hardly be able to insist, from their own observation, that the general effect of the abandonment of the lancet and of local depletion in *private* practice has proved advantageous. The mortality of pneumonia in Philadelphia has, for some reason, certainly increased; not in the hospitals, where the average character of the patients has always made them unfavorable subjects for depletion; but in private practice. It is not too bold an assertion, indeed, for one who, without partisanship, maintains his conviction of the occasional importance of venesection and local depletion as remedies,—that, while acute, uncomplicated pneumonia under moderate depletory treatment was, thirty years ago, rarely fatal outside of hospitals, and not at all frequently so within them, the indiscriminately stimulating method, now often applied to all classes of cases, has increased very considerably the fatality of this and other inflammatory diseases in private practice.—H.]

Classification of Diseases.—A correct classification is a condition of the existence of a science, and an essential for its teaching; but we do not think that "medicine" has yet arrived at this high position. That it will advance to it, that it is making progress towards it, we have no doubt; but, at the present time, we must admit that imperfections abound in every system that has been propounded. The problem is too vast and too complicated for solution now, and we have therefore to adopt that which appears to possess the greatest amount of practical advantage.

It would be useless here to spend time upon criticism of the various schemes which have been proposed; we prefer rather to state briefly the very simple plan upon which this book will be constructed.

It is proposed to make the first division of diseases into their two great groups; 1st. Those in which the whole organism appears primarily and prominently deranged, and 2d, those in which special organs or systems of organs are, in like manner, affected. Subdividing the first group we have two classes; A, those in which the disease appears to be developed by causes operating from outside the body; and B, those in which the malady seems to depend upon some internal change. Thus in the first subdivision we find the acute specific diseases, and their analogous affections; in the second, gout, rheumatism, scorbutus, and the like. Subdividing the second group we have many classes, consisting of diseases of systems of organs, such as: A, diseases of the nervous system; B, diseases of the digestive system and its appendages; C, diseases of the circulatory system; D, diseases of the respiratory system; E, diseases of the urinary system; F, diseases of the reproductive system; G, diseases of the locomotive system; and H, diseases of the cutaneous system.

Each of these is, in its turn, again subdivided, upon the primary principle of general or partial change, so that, in regard of the nervous system, for example, we have 1st, those of general or undetermined seat, and 2d, those depending upon distinct local change in its parts, anatomically considered: and this leads to further reduction into affection of parts, such as, *a*, brain; *b*, spine; *c*, nerves: whereas the final division is based upon the nature of the changes which these portions of systems undergo.

If this mode of arranging diseases has no other merit, it has that of simplicity; and it will, we believe, bring, as a general rule, into closer proximity than some more ambitious systems would allow, those diseases which have the most intimate clinical association. It involves little theory in any case, none in many, and may therefore commend itself to those who realize, amid the great progressive science of medicine, the difficulties and dangers which attend upon all nosologies which, based on theories, partly right and partly wrong, carry with them, and only with great effort disentangle themselves from, what is erroneous in their groundwork, and *à fortiori* luxuriant in their after-growth.

J. RUSSELL REYNOLDS.

PART I.

In the First Part of this System of Medicine are included those diseases in which the whole organism is primarily and prominently deranged. We have therefore to deal with:—

General Diseases, or Affections of the Whole System; *and dividing these into two sections, we have to consider first:—*

§ I.—*Those determined by agents operating from without, such as malarial diseases, the exanthemata, and their allies.*

Influenza.	Varicella.	Epidemic Cerebro-Spinal Meningitis.
Hooping-Cough.	Variola.	Plague.
Diphtheria.	Vaccination.	Erysipelas.
Scarlet Fever.	Glanders.	Pyæmia.
Dengue.	Hydrophobia.	Malarial Fevers
Roseola.	Enteric Fever.	Dysentery.
Measles.	Typhus Fever.	Cholera.
Mumps.	Relapsing Fever.	Syphilis.
Sudamina and Miliaria.	Yellow Fever.	

INFLUENZA.

By Edmund A. Parkes, M.D., F.R.S.

Definition. — An epidemic specific fever, with special and early implication of the naso-laryngo-bronchial mucous membrane; duration definite of from four to eight days; one attack not preservative in future epidemics.

Synonyms.— *Scientific Names.* — Peripneumonia Notha (*Sydenham, Boerhaave*). Peripneumonia Catarrhalis (*Huxham*). Pleuritis Humida (*Stoll*). Febris Catarrhalis (*F. Hoffman, Sauvages*). Catarrhe Pulmonaire (*Pinel*). Catarrhus à Contagio (*Cullen*). Defluxio Catarrhalis. Cephalalgia Contagiosa. Rheuma Epidemicum.

Popular Names.—Pose (in old English writings, from the Anglo-Saxon *gepose*, heaviness). Tac or Horion (in France in 1411). Coqueluche (in France in 1414, and in subsequent epidemics, because the sick wore a cap over their heads.) La Dando, or Ladendo (in France, 1427). Quinte (in France in 1578, because the paroxysms of cough returned every five hours). Follette (in France in several epidemics). Ziep (in Germany in 1580, probably from *zieppen*, to pipe or chirp). Schaffhusten and Schaffkrankheit (in Germany in 1580, because the cough was like the cough of a sheep, or because the vertigo was like the sudden giddiness of sheep). Hühner-weh (in Germany in 1580, because the cough was like the crowing of a cock). Blitz-katarrh (from the suddenness of the attack). Mal del Castrone (in Italy in 1580, because the giddiness was like the common disease "turnsick," of the sheep).

In the seventeenth century it was first called Influenza, in Italy, because it was attributed to the "influence" of the stars, and this term has passed into medical use.

In 1743, it was called La Grippe in

France, from the Polish Grypka (Raucedo), a term which, like Influenza, has passed into medical writings.

It has been called in Russia "Chinese Catarrh;" in Germany and Italy, "the Russian disease;" in France, "Italian Fever," &c.

A great number of other popular names have been given to it: Petite poste; Petit courier; Follette; Coquette; Cocote; Allure; Baraquette; Générale, &c.

History.—Supposed to be referred to by Hippocrates, who yet gives no perfect description. The epidemic among the Athenian army in Sicily (415 before Christ), recorded by Diodorus Siculus (lib. xiii.), has been supposed to have been Influenza. In A. D. 827 an attack of cough, which spread like the plague, was recorded. Again, in 876, Italy, and then the whole of Europe,[1] was attacked, and the army of Charlemagne, returning from Italy, suffered greatly;[2] dogs and birds were both attacked by this disease. In 976 the whole of France and Germany was attacked by a fever, whose principal symptom was a cough. In 1173 another catarrhal epidemic was widely spread; and in 1239 and 1299 other slighter epidemics are noticed.

It is not, however, till the 14th century that the records became numerous and precise:—

In the 14th	century	6	epidemics are recorded.
15th	"	7	"
16th	"	11	"
17th	"	16	"
18th	"	18	"
19th (first half)		10	"

In some cases, however, the same epidemics may have been recorded twice, though I have excluded several that appear to have been so. Probably, also, among the lesser epidemics are some of hooping-cough wrongly diagnosed as Influenza.

There is little doubt that the apparent increase of prevalence in the last centuries is merely due to more accurate recording of minor epidemics.

Of these epidemics, some have been very widely spread over a great part of the known world, as in 1311, 1557, 1580, 1590, 1729, 1762, 1775, 1780–2, 1830–2, 1847. In other epidemics the disease has either been partial, or not recorded in many places; in some instances it has spread only over comparatively small tracts of country.

The first epidemic which was carefully described was that in 1557, by Riverius. The great epidemic of 1580 was described by Sennert.

In England the following epidemics have been recorded,[1] many of them with great care: 1510 and 1557 by Thomas Short; 1658 by Willis; 1675 by Sydenham; 1729–1743 by Huxham; 1732–3 by Arbuthnot; 1758 by Whytt; 1762 by Baker and Rutty; 1767 by Heberden; 1775 by Fothergill, who collected observations from many physicians; 1782 by Gray, Haygath, and Carmichael Smith; 1803 by Pearson and Falconer, and a great number of others; 1833 by Hingeston and others; 1837 by Streeten, Graves, and Bryson, &c.; 1847 by Peacock,[2] Laycock, and many others.[3]

Spread of the Disease.—*Etiology.*—Before entering on the consideration of the external nature of the causes, it is necessary to state the facts which have been ascertained in respect of the spread of Influenza.

It has prevailed in most places of the habitable globe: in the whole of Europe; in China, Tartary, Egypt, India, and other parts of Asia; in Australia, Polynesia; in North and South America, and in the West Indies; that is to say, in both hemispheres and in all latitudes. It has occasionally occurred in both hemispheres at the same time, but more usually has appeared successively in different places, and has been seen at some point or other of the earth's surface for two to four years, after which it has disappeared. In some years, as in 1580, 1730, 1762, 1775, its prevalence has been so great, that almost all parts of the known world have been attacked; at other times it has been more partial, affecting only a part of a continent, or even a single country.[4] It has been supposed, indeed, occasionally to prevail in quite a limited area, in a single city for example,[5] but it is possible that local catarrhal fevers of this kind are not identical with the true influenza.

When it has been pandemic, *i. e.*, when it has invaded a large portion of the earth's surface, its progress has usually been ra-

[1] Schnurrer. Chronik der Seuchen, Band i. p. 175.

[2] Schnurrer states that measles followed, and appeared indeed to be developed out of this epidemic, but the records are necessarily very imperfect.

[1] Annals of Influenza, by Theophilus Thomson (Sydenham Society, 1852). I have only quoted the principal works.

[2] The Influenza, or Epidemic Catarrhal Fever of 1847–8. By G. T. B. Peacock, M.D. London, 1848.

[3] Since 1847 there has been no pandemic Influenza, but several minor outbreaks, the widest diffusion of which was in 1857–8. In Paris in 1867 (February and March) there was a severe outbreak, characterized as usual by great prostration. There was also an outbreak almost at the same time at Strasburg.

[4] See Hirsch, Hist. Geog. Pathol. vol. i. p. 286, for twenty-four examples of this fact.

[5] Hirsch has collected seventeen examples.

pid, yet not to such an extent as is commonly supposed, and sometimes it has travelled slowly. It is said to have overspread Europe in six weeks, and at another time to have taken six months or more to do so. In any particular country its progress may also be comparatively slow; thus, between the invasion of London, and of provincial towns, or of Scotland, weeks, and even sometimes months, have elapsed. Thus in 1762 it appeared in London in the beginning of April; at Edinburgh in the beginning of May; in some parts of Cumberland in June. In 1782 it attacked London in the middle of May; Exeter at the end of May; and Newcastle-upon-Tyne and Edinburgh in the beginning of June.

In 1830-31-32, it prevailed in Moscow and St. Petersburg, and from thence took no less than eight months to spread over the whole of Germany. In Europe it has sometimes prevailed simultaneously at several points, as in 1847, when it was raging at the same time in Copenhagen, London, and Marseilles. In spreading over a large tract of country, it has often been supposed to follow a regular course; which has been believed to be from the high north or northeast to the south and west. Thus it has been supposed to pass from Chinese Tartary to Russia, Germany, Holland, England, Scotland, France, and then to Italy and the Mediterranean, or to America in succession, and certainly in some epidemics there has been a course of this kind. But this is by no means invariable, and may indeed have been accidental, or our knowledge of the successive steps of the spread may have been inaccurate. Thus the epidemic of 1762 was said to follow this course,[1] and to reach America in October, 1762, having affected Germany in February and March, London in April, and France in July. But Influenza prevailed in America the year before (1761) and thence passed into Europe,[2] taking thus the exact contrary of the tract assigned to it, unless indeed it passed round by the icy regions of North British and Russian America, of which there is no evidence. So again it has sometimes (1775) passed from the south to the north of Europe, or from the south or west of a particular country to the north or east. Gluge,[3] indeed, from an examination of the epidemics of the last 300 years, believed he had discovered that its course is from west to east. It is obvious that, in former times, the want of reliable information, and of intercourse between nations, must have rendered all evidence of dates very uncertain. The next epidemic will give more reliable information than any of the former.

When it has entered any large town, it remains there for from six weeks to two months as a rule, but occasionally longer, as at Paris in 1831, when it was more or less prevalent for nine or ten months. It has never, however, failed entirely to disappear eventually, and sporadic cases are not seen in the intervals of the epidemics. In its course it appears to pass over seas, and it is said to have attacked the crews of ships far from land, who had not sailed from an infected port.[1]

The exact spot on the earth's surface where an epidemic commences has not yet been made out, and two opinions prevail. One is, that every epidemic owns one unknown source, whence it spreads; each nation, in turn, attributing to its neighbor from whom it derived the disease, the un-

[1] Robert Williams. On Morbid Poisons (1841), vol. ii. p. 663.

[2] Noah Webster. A Brief History of Epidemic and Pestilential Diseases (London, 1800), vol. ii. p. 44.

[3] Quoted by Hirsch, op. cit. p. 287, footnote.

[1] The statement that Influenza will thus break out in mid-sea, without there being any possibility of the disease having been introduced on board, is a most important piece of evidence, as it would prove that the atmosphere can not only carry the poison, but that no degree of dilution can destroy it. Without denying the occurrence of such outbreaks, I cannot but consider we require better evidence of ships being attacked in mid-ocean. In some of the quoted instances, the ship had been at a port either known to be infected, or in which Influenza was really present, though it had not become epidemic. As we are ignorant of the exact period of incubation, some men may have been infected before sailing. In other cases the examples are of old date, and it is impossible to feel quite sure that the evidence is correct. Such for example as the celebrated case of the *Atlas* East Indiaman, which was attacked with Influenza, on a voyage from Malacca to Canton; Malacca being healthy at the time, but Canton being affected at the same time as the ship. (Robert Williams: On Morbid Poisons, vol. ii. p. 667.) In 1782, Admiral Kempenfeldt's and Lord Howe's squadron, cruising at different parts of the Channel, were each attacked, although, it is said, they had been at least twenty-two to twenty-seven days at sea.

There is better evidence that ships near the land have suffered. In 1833 the *Stag* frigate was coming up Channel, and when off Beechy Head, in Devonshire, the wind was easterly and off the shore at two o'clock, the crew being then quite healthy (and it is presumed no communication having taken place, but this is not stated)—40 men were, at half-past two, suddenly attacked with Influenza; at six o'clock 60 men were down, and by the next day 160. (Watson: Principles and Practice of Medicine, 4th Edition, vol. ii. p. 44.) If it were certain that there had been no communication with the shore, the cause must have drifted over the sea.

enviable honor of originating it. Thus the Italians have termed it the German disease; the Germans, the Russian pest; the Russians, the Chinese Catarrh; and these names are indeed some evidence of its usual track. Noah Webster attributed its origin to America in 1698, 1757, 1761, and 1781, while in 1788 he believed it arose in Europe, and several writers have fixed it in Chinese Tartary, or in India. The other opinion is, that it has no special place of origin, but may arise anywhere; and some,[1] indeed, have questioned whether such "autochthonic" developments are not the rule, and whether we are right in believing in a "genetic connection" of the various local outbreaks. But surely no one can doubt the connection of the various attacks in the great epidemics of Influenza with some general and pandemic influence.

If it may arise thus spontaneously in various places, no one has yet precisely indicated its first origin.

It has been also supposed to have a cyclical course, and to return pretty regularly in periods of years. The older writers thought it had a cycle of about 100 years, but it has returned much more frequently than this; about every twenty-five to thirty-five years it has been pandemic, and lesser outbreaks have occurred more frequently. But no regular period can be at present perceived.

In passing through a country it does not attack all parts of it; it more usually spares the country places, but sometimes even large towns escape.

When the disease enters a town, it has occasionally attacked numbers of the inhabitants almost simultaneously. But more frequently its course is somewhat slower; it attacks a few families first, and then in a few days rapidly spreads; the accounts of thousands of persons being at once attacked at the onset of the disease are chiefly taken from the older records, in which the suddenness of the outbreak is exaggerated. Frequently, perhaps always, in a great city the outbreak is made up by a number of localized attacks, certain streets or districts being more affected than others, or being for a time solely affected, and in this way it successively passes to different parts of the city. It has generally occurred in a great city before appearing in the smaller towns and villages round it, and sometimes these towns, though in the neighborhood, have not been invaded for some weeks.

In some cases, and perhaps a large number, it breaks out after persons ill with Influenza have arrived from infected places.

The decline in any great town is less rapid than its rise, and usually occupies from four to six weeks, or sometimes longer.

In every epidemic the symptoms so closely resemble each other, that there is no difficulty in recognizing it from the descriptions even of ancient and unlearned writers; yet there are said to be certain slight differences in symptoms between different epidemics, to which reference will be hereafter made.

Different epidemics have varied somewhat in the number of persons they affect, but on the whole a large number suffer.

In London in the last epidemic (1847) it has been calculated that at least 250,000 persons suffered; in Paris, between one-fourth and one-half of the population suffered, and in Geneva not less than one-third.[1]

When the different telluric and atmospheric conditions which are coincident with the attacks are considered, the following are the conclusions:—

Soil.—It prevails on every soil and geological formation, and apparently equally on all. It has been supposed to be worst on marshy soils, and some have even believed it to arise in very malarious regions, as Lower Bengal, or the plains of China, yet very malarious countries, as Holland, do not suffer more than others; Holland, indeed, has escaped some epidemics which have traversed Europe. Lowlands have been sometimes affected more than the adjacent hills, as in the lowlands in Jamaica, in 1802, and in several epidemics in the Lombard plains, as compared with the Blue Mountains and the Alps.

Volcanic Eruptions.—Telluric Emanations.—Noah Webster[2] and Schnurrer[3] have collected the available evidence on this point, but it is entirely negative. There have been constant volcanic eruptions without Influenza, and epidemics of Influenza without great volcanic eruptions. It has been thought that emanations of seleniuretted hydrogen from volcanoes might excite Influenza, but no proof has ever been given of the existence of this substance in the atmosphere.

Electrical Conditions.—No evidence has been collected which shows any connection with conditions of telluric magnetism or atmospheric electricity; and indeed the peculiar spread and frequent localization of Influenza seem inconsistent with general magnetic conditions.

Seasons.—The disease appears at all times of the year;[4] nor is there any reason

[1] Biermer, in Virchow's Handbuch der Path. und Ther., vol v. p. 607. (1865.)

[1] Peacock on Influenza, p. 13, Introduction.

[2] A Brief History of Epidemic and Pestilential Diseases. 1800. Vols. i. and ii.

[3] Chronik der Seuchen. 1825.

[4] See Hirsch, op. cit. p. 287, for evidence on this point, but almost all writers have noticed it.

for considering it an affection of the late summer, autumn, and winter, as has been stated.

Temperature of the Air.—Owing to the confusion in the popular mind between Influenza and common catarrhs or catarrhal fevers, it has been always a common opinion that Influenza depends either on a low temperature, or a sudden variation of temperature. This error has taken a long time to kill; but almost every writer, since the epidemic of 1580, has examined this point,[1] and has decided that there is no connection between either low temperature, or variations in temperature, and Influenza. As respects high temperature, it has prevailed in the West Indies at a temperature of 72°–82°;[2] on the hot sea-coast of Java; in South India; in Egypt; at the Cape of Good Hope, in the most genial season; in the south of Europe in summer. So also there is abundant evidence to show that the changes of weather, which may appear to have accompanied or preceded its outbreak, were mere coincidences.[3]

Moisture of the Air.—It has prevailed in the dry air of Upper Egypt; in the moist air of sea-coasts, and on the sea itself, without being apparently in any way influenced.

Barometrical Condition.—No coincident alteration can be traced.

Ozone.—Although ozone was known before 1847, the observations during that year led to no result, and since that time there has been no epidemic of Influenza. But the observations hitherto made on the effect of ozone on other diseases[4] seem to render it improbable that any connection will be traced between the development of ozone and Influenza. The statements of Schönbein are based merely on the effect of large quantities of ozone artificially produced, on the mucous membrane of the nose. Applied in large quantities, ozone is irritating, and may produce simple catarrh, but nothing like the specific symptoms of Influenza.

Fogs and Mists.—In some cases, as in Paris in 1675, France in 1733 and 1775, England in 1782, a thick and acrid fog has shortly preceded, or has immediately ushered in, the Influenza; but so many outbreaks have occurred without such a coincidence, that it is impossible to attach any weight to it.

Wind.—Its main spread is not influenced by the wind; it does not move with the same velocity; it often moves against it. Yet it appears to be sometimes carried by the wind for a short distance. (See case of the *Stag* frigate, previously quoted.)

In fine, if there is any special atmospheric condition which invariably attends epidemics of Influenza, it has yet to be discovered, and the words of Pearson are still true:—

"Between the epidemic and the condition of the atmosphere, there appears to be a connection different from that which depends on a mere alteration of temperature, or of dryness or moisture, but what that peculiar connection is we shall not attempt to explain."[1]

Fungi in Atmosphere. — Abundance of flies, caterpillars, etc.—Attempts have been made to show that during epidemics there are indications of an unusual development of animal or vegetable life, and that "bloody or red snow," "blood rain," "flights of locusts, or insects," &c., are more common in Influenza years. These speculations have, at present, even more than usual interest, and certainly should be brought to the test of close inquiry. At present, all that can be said is that no facts of any moment exist which connect an unusual fungoid development with the spread of Influenza.

We must now pass on to a different order of facts.

Human Intercourse.[2]—The rapidity of the spread would seem at once to negative any connection between human intercourse and the propagation of the disease; yet there is some affirmative evidence. It does not appear to follow the great lines of commerce; but when it has entered towns and villages in which the investigation can be carried on, it is curious how

[1] Salius Diversus (1580); Molineux (1693); Whytt (1757); Baker (1762); Haygarth (1775–1782); Fothergill (1775); Metzger (1800); Lombard (1831), &c.

[2] Observations relative to the West India Islands, by John Williamson, M.D. 1817. Vol. ii. p. 110.

[3] In his late work (Catarrh and Influenza, 1865), Seitz attributes more influence to the effect of vicissitudes of weather in causing epidemic Influenza than appears to me to be warranted by the facts.

[4] Especially those of Shiefferdecker: Sitzungsbericht der Math. Naturw. Classe der Wien. Akad. July 1855, Band xvii. Seite 191. The ozonic results had no connection with any malady, and were in all cases proportionate to a numerical range, derived from a consideration of the strength of the wind and of the moisture of the air. See also Seitz (Catarrh and Influenza, 1865, p. 360).

[1] Observations on the present Catarrhal Fever, or Influenza, by Richard Pearson, M.D. London, 1803. P. 3, footnote.

[2] The presumed importation of Influenza into Iceland and the Faroe Islands, as described, especially recently, by Schleissner and Panum, as well as by older writers, is doubtful. It would seem probable that the endemic catarrh of these islands, said to follow each year the arrival of the first ship, is different from the true Influenza, which comes more rarely, and only when it is prevailing elsewhere in Europe.

frequently the first cases have been introduced, and how often the townspeople nearest the invalids have been first affected In this country especially, Haygarth in 1775 and 1782, and Falconer in 1802, collected so many instances of this that they became convinced that its propagation was due entirely to human intercourse.[1] So also, when it passes through a house, it occasionally attacks one person after another. But if it is introduced in this way, it afterwards develops with marvellous rapidity, for we cannot discredit the accounts of many thousand persons being attacked within a day or two, which is quite different from the comparatively slow spread of the contagious diseases. This *sudden* invasion of a community makes it, to many persons, appear highly improbable that any effluvia passing off from the sick should thus so rapidly contaminate the atmosphere of a whole town.

Still, we must remember how singularly, of late years, the knowledge of the introduction of cholera by persons coming from infected districts has increased, and how very striking are the instances of this kind already recorded in several works on Influenza.[2]

In some cases, again, isolation or seclusion of a community, as in prisons, have given immunity; or at least that community has not been attacked.

Inoculability.—The disease is not inoculable; at least, when horses are attacked, it cannot be transferred from one horse to another. (Hertwig.)

Incubative Period.—All the contagions have one remarkable property; there is a time when they are said to lie dormant, and to be undergoing or inducing in parts of the body those changes which lead at last to the symptoms of the declared disease. During this period there are either no symptoms, or, what is more probable, they have not been determined.

Such a period has been supposed not to exist in Influenza,[1] which has been said to strike down persons in perfect health, as with a stroke of lightning. But the suddenness does not exclude an incubative period without subjective symptoms. It is also certain that the incubative period sometimes exists. It is sometimes very short;[2] sometimes of many days' duration.

Preservation from Second Attack.—There is some discrepancy of evidence; but, on the whole, it seems clear that, while persons seldom have a second attack in the same epidemic (though even this may occur), an attack in one does not protect against a subsequent epidemic. Indeed, it has been supposed rather to render the body more liable.

Relation of other Epidemic Diseases of Man.—It has been attempted to trace out a connection between Influenza and measles, the plague, yellow fever,[3] and cynanche maligna (diphtheria). It has been

[1] Sir Thomas Watson, M.D., whose care and accuracy inspire such faith, says also on this point: "The instances are very numerous, too numerous to be attributed to mere chance, in which the complaint has first broken out in those particular houses of a town at which travellers have arrived from infected places." (Principles and Practice of Medicine, vol. ii. p. 43. 4th Edition.)

Sir George Baker was one of the first who noticed this fact. (Opuscula Medica; Edition of 1814, p. 27.)

Cullen's term, "Catarrhus à Contagio," seems to me to imply, however, merely the idea of origin from a special virus.

[2 May not this analogy, however, be, with equal facility, used in the reverse manner as an argument? As the general history of Influenza seems so cogently to negative any connection between human intercourse and its propagation, therefore the occurrence of some apparent instances of the introduction of cholera by persons should be regarded as most probably explicable otherwise than by contagion.—H.]

[1] Biermer, op. cit.: "The disease seems to come on without an incubative stage; the causes of Influenza do not work after many days, as a contagion; but rapidly, like a poison."—P. 604.

[2] In the Transactions of the College of Physicians (vol. iii.), it is stated that in the epidemic of 1782, seventeen persons came to London to an hotel, and on the following day three were attacked with Influenza. Haygarth (On the Manner in which the Influenza of 1775 and 1782 spead by Contagion in Chester and its Neighborhood, by John Haygarth, M.D., F.R.S.) says that a gentleman came to Chester from London, on the 24th of May, 1782, ill of Influenza; a lady, into whose family he came, was seized on the 26th, and was the first case in the town. Haygarth states, evidently with the wish to point out the possibility of a direct contagion, that the gentleman was engaged to be, and was afterwards, married to this lady. In this case the longest possible incubative period was two days. In 1782 a family landed at Harwich, from Portugal, and came to London directly; the day after arrival, the lady, two servants, and two children were all seized. Two men-of-war arrived at Gravesend from the West Indies; three Custom-house officers went on board; a few hours afterwards the crews of both vessels were attacked. (Robert Williams, on Morbid Poisons, vol. ii.) Some other cases are on record where the incubative period, if it existed, could not have been more than a single day. On the other hand, some cases are on record in which the incubative period must have been two or three weeks. (Ibid. vol. ii. p. 674.)

[3] Noah Webster, op. cit. vol. ii. p. 48. To some extent Schnurrer held that there is some connection between measles and Influenza.

supposed also to precede and herald cholera. On the other hand, it has been stated that epidemic scarlet fever disappeared when Influenza prevailed, and reappeared when this ceased. The same fact has been affirmed of smallpox.[1] During its prevalence other severe inflammatory diseases have been supposed to lessen. With regard to all these supposed relations, the evidence is most unsatisfactory. Coincidences between the prevalence of different epidemic diseases must be expected, but it would require repeated instances to prove any connection. Measles constantly prevail without Influenza; and if an epidemic has occasionally followed an Influenza epidemic, this really proves nothing. The utter want of connection between cholera and Influenza is evident at a glance.

So also the very imperfect knowledge we have of the relative prevalence of the acute inflammatory affections, makes it quite uncertain whether cases of simple bronchitis, rheumatism, and pneumonia really lessen in number during influenza. According to Graves (Clinical Med., vol. i. p. 425), during acute diseases persons are less liable, but they may be attacked at convalescence.

It has not been shown to prevail especially in years when intermittents have been more common, yet there may be some connection between the diseases (see *Symptoms*). Instances have been given in which intermittents seemed to disappear, and others in which the Influenza seemed to cause intermittents.

Relation to the Diseases of Brutes.—In some epidemics of Influenza, dogs,[2] horses, cats, and, it is said, birds, have been affected simultaneously with an epidemic catarrh. Horses are subject to an epidemic catarrhal disease (1827[3]) even when no Influenza prevails among men, and this disease, to which veterinary surgeons now give the name of Influenza, appears closely to resemble human Influenza. Its contagiousness has been warmly debated, and it certainly appears incapable of inoculation, but yet some believe it to pass from horse to horse. It appears to be now generally thought an epizootic affection, and dependent on a specific cause.[4]

After this statement of the facts connected with the spread of Influenza, we proceed to notice the speculative subject of the

Nature of the Exciting or External Causes.—So enigmatical are the phenomena connected with Influenza, that caution is necessary in attempting to form some idea of what the nature of the exciting cause may be.

It must be a specific agent of some kind. From the earliest times authors have come to this conclusion; the similarity of the symptoms in different epidemics show that this agent is the same in its successive invasions. If it be connected with an unusual meteorological or atmospheric condition, this has not been detected, and cannot be at present even guessed at.

At the same time this agent must be in the air; the diffusion is too rapid to suppose it to be conveyed by water; besides, water-poisoning is usually localized. It cannot be attributable to food. There remains only the air as a medium of communication; and that this is so, seems also shown by the way in which it can attack vessels at some distance at sea.

There is, then, some special agent in the air. But this cannot be a gas; no gas could spread in this way without utter dispersion and destruction. Besides, the manner in which it is located in a part of a town, a street, even one side of a street, for a time, or affects a town without touching a village a mile or two off, is quite conclusive against the hypothesis of seleniuretted hydrogen, allotropic oxygen, or any other gas being the cause. Nor can it be any molecular matter driven through the air, arising from some unknown telluric source, for this would be equally diluted and dispersed. The agent evidently cannot own one single and primary origin; it may, indeed, issue from one spot, but all the phenomena of its spread show that it must, in its transit, reproduce itself. Otherwise, if a gas, it must be rendered innocuous by dilution; if an organic matter, by oxidation; if a suspended mineral matter, by subsidence. It must increase, and the more the subject is gone into, the more firmly will the idea gain upon the mind, that there must be a continual reproduction of the agent, to a greater or less extent, in different places.

Now this reproduction must either be in the air or in the bodies of the sick, in which latter case the agent would be a true contagion. If it grows in the air, the only conceptions we can form are, either that some force changes successively the atmospheric elements in some way, or that the increase is a vital one, and consists of microscopic plants or animals. The first idea is supported by no evidence; and as to the second, we find ourselves in the presence of the so-called fungoid

[1] See Biermer, op. cit. p. 619, for references on these points.

[2] In the great epidemic in Australia, in 1851–52, dogs were affected in great numbers.

[3] In this Influenza of horses which spread over almost all Europe, no cause could be found in the weather, food, or work of the horses; transfusion of blood of a diseased horse did not communicate it to another; many veterinary surgeons considered it to be contagious; others did not hold this opinion. Influenza prevailed among men in North America, Mexico, and Siberia, but not in Europe.

[4] Turpentine has been used beneficially in the so-called Influenza of horses.

theory of Influenza. There are many phenomena consistent with the hypothesis of a vital and growing cause: the occasional introduction of the disease by persons about whose bodies or clothes the fungi may cling; its passage at times with the wind, contrasted with the occasional passage against it when other modes of conveyance may be presumed to come into play; the gradual development of the disease to a climax, and then its decline, contrasted with its occasional persistence when the conditions of growth may be supposed to be more persistently in the same place; the entire disappearance of the disease, and its extremely rapid resuscitation when it again appears; its birth, apparent in various parts of the world, and yet its evident incapability of originating in some countries, as France and England (whither it has always passed from other lands), are all easily explicable if we assume a fungoid origin, and remember the different conditions which can effect the development of fungi. The remarkable powers which have lately been ascribed (with what justice time must show) to those lower forms of life increase the interest with which this question must be regarded.

But, on the other hand, there is a complete want of direct evidence, without which the argument in favor of a special living agency is worth little. It is impossible to make a certain and assured step without some tangible evidence.

Moreover, for the rapid increase of fungi we should suppose certain meteorological conditions to be necessary—a certain temperature, moisture, organic effluvia; but the spread of Influenza has little, if any, connection with these conditions.

If the cause be a fungus, or some allied organism, it may increase in the body as well as out of it, and if so would be found in the secretions, especially in the nasal, buccal, and bronchial mucus. In this way human intercourse would spread it. A thorough microscopic examination of these discharges is yet wanting, but possibly the next epidemic may supply this link.

If the agent is not a fungus, the only other ready explanation which presents itself is that of a true contagion; namely, that particles of the sick body being thrown off are in some special condition, or are undergoing certain putrefactive or other chemical changes, which can excite a similar action on particular parts of other human bodies. And in this case, to account for the spread of Influenza, we must believe that these particles pass off in myriads from each sick person, are excessively small and light, perhaps become dried up, and floating through the air, to greater or less distances, are breathed or swallowed by other persons, and then set up in their bodies the same series of changes which the particles themselves are undergoing. This view seems to me to involve greater difficulties than the fungoid theory, *i. e.*, it accounts less satisfactorily for the spread of Influenza.

If neither of these views be correct, then the cause of Influenza is something of which we have no conception whatever. It seems to me to be impossible at present to come to any conclusion as to the nature of the cause. [Two alternatives seem to remain for further investigation, in connection with the etiology of Influenza, as well as of several other epidemic diseases; especially Yellow Fever and Cholera. One of these is an expansion of the idea proposed and elaborated especially in regard to Cholera, by the late Dr. Snow, of London; designated by him as the theory of "continuous molecular change;" supposed by him rather to extend than to substitute the common notion of contagion. The other, very probable, view is, that the "disease germs" of many epidemics, although really organic in nature, are *ultramicroscopic* in minuteness, and therefore not demonstrable except by their effects.—H.]

Predisposing or Internal Causes.—Race has no influence, sex probably none, or, if at all, women are slightly more affected; age has only a slight effect; young children are, it is said, rather less affected than old persons. If any special bodily predisposition is necessary, it is common to the whole human race, and apparently to horses, dogs, cats, &c., herein differing greatly from several of the true contagions.

Persons in overcrowded habitations have, particularly in some epidemics, especially suffered, and several instances are on record of a large school or a barrack for soldiers being first attacked, and of the disease prevailing there for some days before it began to prevail in the town around. Sometimes, on the other hand, schools and prisons have escaped.

A low, damp, ill-ventilated and unhealthy situation appears to predispose to it,[1] and in some instances, in hospital patients, it has assumed a malignant character (Sir George Baker, Gray). In other cases again, hospital patients have escaped; for example, the old people in the Salpétrière in 1837, when the younger attendants were attacked.

It has been supposed that persons with chronic lung diseases, especially emphysema, and chronic heart affections, are particularly liable, but this seems uncertain; it is probable that the Influenza being more serious in such persons, creates the impression that they are as a

[1] Pearson noticed this in both 1762 and 1782.

class more liable. The Registrar-General has shown that in 1847 the increase of deaths by Influenza was much greater in the districts in which ordinarily there is a high mortality than in healthier places; this must indicate either greater prevalence or greater severity of the disease.

Symptoms.—*General Course of the Disease.*—The symptoms of Influenza are compounded of two conditions—a general fever of determinate duration, and a marked and evidently specific affection of the mucous membrane of the nose, mouth, throat, and respiratory tract, which has also a determinate course.

Individual cases differ in the proportion of these two conditions, and in addition there may be superventions of true inflammation of the lungs or pleura, or implication of other mucous membranes, those of the stomach and intestines in particular, and less frequently of the bladder and kidneys.

It would appear that the fever has the priority, and that shivering or coldness down the spine, with heat and flushing and dry skin, quick pulse, thirst, and severe headache, very frequently usher in the attack. These symptoms precede any local signs. But it would be very desirable to re-investigate this point. The febrile symptoms sometimes come on quite suddenly, sometimes develop slowly, in from twelve to thirty-six hours, or even to four days. When they commence suddenly, the first symptom is often an extreme frontal headache, with pain and aching in the eyes.

They last for four or five days usually, or sometimes a few days longer, and then disappear gradually, or occasionally rather rapidly, with profuse perspirations, or spontaneous diarrhœa. Sometimes they continue ten or twelve days, but this is generally when pneumonic complication supervenes.

The specific catarrhal affection usually follows the early symptoms of fever; sometimes occurs at the same time, perhaps sometimes precedes them. It appears to commence in extreme hyperæmic swelling and dryness of the mucous membrane of the frontal sinuses, the nose, and, in a less degree, of the conjunctivæ, causing intense pain across the brows, great sneezing, sometimes epistaxis and thin acrid discharges from the nose and eyes; the same condition then occurs in the pharyngeal, the laryngeal, tracheal, and pulmonary mucous membrane to the minutest ramifications. Usually, perhaps, the affection commences above and passes rapidly down, but sometimes the whole tract is attacked at once. The inside of the mouth and the tongue are also, but less, affected, and the pharynx is also not so marked by hyperæmia as the other parts. A punctiform redness of the mucous membrane of the palate, something like the eruption of measles, has been lately described by Tigri, and considered to be pathognomonic. The discharge from these membranes, when it occurs, is first thin and acrid, and sometimes bloody; it becomes afterwards thicker, more tenacious, and at length purulent; great sneezing, sore throat, difficulty in smelling, violent paroxysmal cough, pains in the chest; occasionally very sharp stitches in the side, which are apparently often nervous, and not pleuritic, accompany the specific condition of the respiratory tracts. Great dyspnœa and the stethoscopic examination show that there is immense congestion of the lungs, and often the face and lips show very considerable impairment in the aëration of the blood. In pure cases the catarrh is at its height on the second and third, or fourth day, and declines about the fifth to the seventh; but cough expectorations often remain for some time. In severe cases the disease lasts with great severity even to the tenth or twelfth day.

Attendant upon these symptoms, and in proportion, it is usually supposed, to the fever, though some have thought it to be in more direct ratio to the extent and violence of the membranous catarrh, is a peculiar state of the nervous system. Very early in the disease there is a remarkable nervous depression, loss of strength, and lowness of spirits, combined often with great aching in the muscles, and severe nerve pains in different parts, which certainly give one the impression that both muscles and nerves are undergoing some profound nutritional alteration. The mind, too, becomes weak, and sometimes there is even stupor or delirium. In some epidemics, indeed, the early sopor or cerebral heaviness is very remarkable.

These nervous symptoms often last longer than either the fever or catarrh; hence convalescence is tedious and mental activity slowly regained.

In pure cases, when the disease is over, the nasal and respiratory mucous membranes do not for some short time entirely recover their structure, at least if it be true that there is increased liability to common catarrh. Also if it be true that there is a greater liability in future epidemics of Influenza, it is possible that some structural change may permanently remain. The severity of the cases differs greatly, and sometimes the affection is very slight, sometimes very severe.

Consideration of the Special Symptoms.—1. *Temperature of the Body.*—No observations have yet been made with the thermometer. In some epidemics (1580, Salius Diversus) there has been intense heat of skin; in others (1775,

Fothergill) the skin has not been particularly hot. But perhaps this might depend upon individual cases; for in the same epidemic some have great, others have slight, fever.

2. *Condition of the Skin.*—Sweating at first is usually absent or partial. If it is profuse in the early stages, the disease is sometimes arrested. The perspiration is often sour smelling, and is said to be very acid. In the epidemic of 1782 in London, the sweating was so profuse as to cause the name of sweating sickness to be given to the Influenza. Sudamina are sometimes seen in great numbers, so that the case looks like miliaria. A pustular or herpetic (Peacock) eruption about the mouth sometimes occurs. There is no decided eruption peculiar to Influenza, but occasionally it is said rose-colored little blotches, and sometimes urticaria are seen. There is sometimes most decided hyperæsthesia of the skin of the neck and head; this is usually coincident with severe headache.

3. *Nervous and Muscular Symptoms.*—The headache is often excruciating; frontal most usually; limited to the region of the frontal sinuses, or extending more or less over the head, or over the face, (Antrim of Highmore); there is often great heaviness, sometimes torpor, and occasionally delirium. In some epidemics high delirium has been considered a mortal symptom (Huxham in 1737). Severe vertigo is a common symptom. There is a general lowering in the acuteness of all the special senses. The spirits are low, mind weak; the nights restless, and this loss of sleep is not in relation to the fever; it is seen often in patients without fever.[1]

Meningitis occasionally occurs, and sometimes otitis, and there is often severe pain in the region of the Eustachian tube. There are also neuralgic or rheumatic-like pains of many parts of the body, especially of the muscles of the neck, loins, legs, and the intercostals.

The extreme prostration of muscular strength has been already noticed; it is often a very early symptom, and in some epidemics has given almost a special character to the disease; the complete return of strength does not occur till after convalescence is far advanced.

4. *Respiratory System.*—The paroxysmal cough is one of the most distressing symptoms, and sometimes causes hernia, or abortion in pregnant women. At first dry, the cough is soon attended with stringy, often bloody sputa; as soon as the sputa get more consistent, thicker, more opaque, and purulent, the cough lessens. In different epidemics the amount of cough has varied, but this, may be, in part depends on erroneous observations, as formerly no doubt epidemic hooping-cough was confounded with Influenza. Dyspnœa is often considerable, and is dependent either on the great congestion of the respiratory tract, or on pneumonic complications, or possibly, as suggested by Graves, on some special implication (paralysis?) of the vagus. There are often remissions in the dyspnœa not accounted for by stethoscopic signs. Occasionally there are orthopnœa and suffocative attacks. Sometimes there is intense and oppressive feeling across the chest. The number of respirations is often great, and the pulse-respiration ratio becomes one to two-and-a-half or one to three. In bad lung cases the voice is often very weak as well as hoarse.

At first the stethoscopic signs are almost wanting; the vesicular murmur is feeble, even though the percussion note be clear; if there be dulness, it is equal and indectible. Afterwards when œdema of the lung occurs they are fine moist râles, and sonorous and sibilant rhonchi are present in some cases.

Capillary bronchitis, pneumonia (which is usually combined with pleurisy), and pleurisy are present in some cases, though it is impossible to state in how many. It has been supposed that pneumonia occurs in from five to ten per cent.[1] In some epidemics pneumonic complication is supposed to be more common, as in 1837; the pneumonia is said to be of the catarrhal variety when it occurs during the attack (about the fourth to the sixth day), and of the croupous kind when it occurs, as it sometimes does, in convalescence (Lombard). The supervention of pneumonia is not easily detected by stethoscopic signs before consolidation, in consequence of the œdema. The same reason makes it sometimes difficult to detect true capillary bronchitis unless one lung is more affected than the other. Pleurisy is easily detected.

Sometimes it is supposed that a sort of paralysis of the lungs occurs with great œdema (Graves), possibly from affection of the vagus. Collapse of some portion of the lungs often occurs. That during the height of the disease aëration is most imperfect, is evident from the dark lips, congested cheeks, and great distress, which are often seen.

As sequelæ to the chest affection, chronic laryngitis, chronic bronchitis, emphysema, and tuberculosis are sometimes seen. Yet it is well known that some phthisical patients pass well through Influenza without increase of their disease.

5. *Circulatory System.*—At first strong and quick, the pulse soon becomes soft,

[1] The epidemic of 1712 was attended in Tubingen by great drowsiness, and in that outbreak the brain symptoms appear to have been unusually heavy.

[1] Biermer, op cit. p. 624.

and in the latter stages feeble, and even slow. It is often singularly changeable within a few hours. Heart affections are not common, yet pericarditis will occur, and is then usually complicated with pleurisy.

The blood is buffed and cupped in pneumonic complications, perhaps in all cases. (Vigla.)

6. *Digestive System.*—Nausea and vomiting are sometimes seen in the commencement; diarrhœa is much less frequent till towards the end, when there are often rather profuse discharges; thirst and complete anorexia are very usual. There is sometimes pain in the right hypochondrium, and a yellowish tint of eye and skin. (Peacock.) In some cases there is a decided icteric state of the skin. The great depression and languor is very similar to that which accompanies some cases of jaundice when the bile is accumulating rapidly in the blood. Sometimes the bilious vomiting, fever, and oppression of the brain cause the case to resemble the bilious fever described by authors. There is no evidence of any splenic affection. In some epidemics these gastro-enteric symptoms have been, it is said, more pronounced than in others, but there is no doubt that cases of typhoid fever complicated with or following Influenza have often been described As a rule, in pure cases, the symptoms of stomach and bowel implications are not marked, or are caused by medicine or food.

Urinary System.—The urine is at first scanty and high colored; at a later period it becomes sedimentous from lithates, which are often pink; it is believed there is no albumen nor bile, but good observations fail on these points, as well as on the composition of the urine in twenty-four hours. Occasionally there is almost complete or entire ischuria.

Genital System.—The catamenia are sometimes induced, and amenorrhœa has been thus cured. Abortions are frequent, especially in some epidemics, probably from the violence of the cough.

Lymphatic System.—Swellings of the parotid, the submaxillary, and sometimes the cervical glands are observed, and occasionally, but rarely, severe parotitis follows.

It is somewhat curious that either an intermittent fever has been united to Influenza in some epidemics, or that the Influenza has had an intermittent character. Thus in 1580 Sennert mentions that the quartan fever was joined to the epidemic; in 1658 Willis states that the epidemical catarrhal fever often had an intermitting character, usually tertian, rarely quotidian. In 1762 Baker says that Influenza appeared under the form of an intermittent with tertian periods. In 1767 Donald Monro also saw an intermittent character, but not so marked as in the epidemic at Bremen in 1762. In 1775 he says that few persons had such distinct paroxysms as to resemble those of an ague; but Fothergill (1775) states that "in many instances the disease assumed the type of an intermittent towards its decline." In 1803 Pearson noticed that the lassitude and depression which continued after the fever had gone had an intermittent character, and were worse every other day. The histories of the recent epidemics show no character of this kind, and it is possible that in former centuries the far greater prevalence of malaria impressed on other diseases a periodical character which was not in their own nature. But the observations are curious in connection with the opinions of those who have connected Influenza with malaria.

Varieties of Influenza.—The varieties in different epidemics have been already referred to. In the same epidemic Influenza differs in intensity in different people. In some persons it is an extremely slight disease; in others, a very severe one; this is especially the case if there are pulmonic or gastric complications. So also in some cases an unusual nervous depression prolongs a case which might be otherwise a mild one, or paroxsymal cough and expectoration, or flying neuralgia, or rheumatic-like pains continue for some time during convalescence.

Mortality.—This seems to vary greatly in different epidemics (1837 and 1847 were more fatal than 1833–4; Graves), and is also partly, perhaps, dependent on treatment. Wierus says that the great mortality in Italy in 1580 was owing to the promptitude with which the Italians bled; the mortality in London has sometimes been severe, while it has been slight in Germany. In 1837 the rate of mortality was calculated at two per cent., but it was considered that this was a very severe epidemic.

There appears no doubt that mortality increases greatly with age. It is also higher in persons with chronic bronchitis, emphysema, and chronic heart-diseases, especially dilated and fatty hearts. Mere valvular disease, without loss of power, has little influence. During the prevalence of Influenza, other causes of death show an increase, especially pulmonary complaints, and typhoid, and typhus. This depends probably on the superventionof Influenza upon those affections.

Diagnosis.—If the term Influenza is restricted to the truly epidemic disease which spreads over large tracts of country, there is no difficulty in the diagnosis. Although there is no special eruption as in the exanthemata, or peculiar cough as

in pertussis, or membranous pellicle as in diphtheria, the collection of symptoms is peculiar.

Nor can there be any confusion between cases of epidemic cough and isolated cases of catarrh, arising usually from marked meteorological conditions. However common such attacks, however severe and *influenzoid* they may be in certain cases, they do not constitute an epidemic; there is no disease spreading over the country. Moreover, the symptoms are really dissimilar in their mode of connection and succession. Far more difficult is the diagnosis between true Influenza and catarrhal fevers invading a town or district. That there are such local or endemic attacks of catarrhal fever seems certain, and it is doubtful whether or not they should be classed with Influenza. They want the power of travelling; they attack more slowly, and are far less common among the population. There is for the most part less of the overwhelming prostration, and fewer mucous membranes are attacked. They appear usually to be merely the common catarrh developed into unusual proportions by changeable or severe weather, and possibly this may be the simple explanation of their occurrence. If by the use of the thermometer a typical course of temperature is discovered in Influenza, or if the examination of the excretions detects any special characters, the diagnosis will be easy. Till these points are determined some doubt must exist, nor does it seem to me possible to lay down any precise rules of diagnosis. There are few points more deserving careful study than the precise characters and causes of catarrhal fevers, localized in a town or district, and not forming part of a general epidemic.

There is no other disease with which Influenza can be confounded, but during its prevalence many other diseases—bronchitis, typhoid fever, etc.—are often called Influenza, and this probably has given rise to the opinion that during epidemics of Influenza such diseases lessen, to reappear at its close.

Pathology.—We are not yet in a position to discuss the pathology of this disease. Does the agent enter the blood, act on the nervous system, and then by election seize upon and irritate the mucous membrane of the respiratory tract? Or is it really a membranous local disease, acting very promptly (just as simple angina will act on the system at large) in the secondary constitutional effects? At present the sequence of symptoms seems to show the first view to be more probable, viz., that it is a general disease, with a special secondary localization.

What is the exact nature of the general disease? The blood is buffed and cupped; *i. e.*, there is increase in the fibrine; that is all that is known of the blood; the peculiar changes in the nervous system and the muscles are quite unknown.

What is the exact nature of the respiratory affection? If we reply, it is a general hyperæmia, this is a mere translation of terms. Of the exact cause of that hyperæmia we have no idea. Is there a partial coagulation of blood in the venous system, or some affection of the vaso-motor nerves leading to general dilatation; and is the altered mucous discharge due to such change, or to some special condition of the nutrient plasma as it comes from the vessels, which strikes deeply at their growth and nutrition? The inflammation, if we are to give it that term, is evidently specific; in what the specific character consists it seems at present vain to inquire.

Morbid Anatomy.—Fatal cases of pure Influenza are rare; they occur chiefly in old persons, with old lung or heart disease, or in consequence of recent inflammatory pulmonic or cardiac complications.

The results of simple Influenza seem to be general congestion of the respiratory tract, amounting sometimes to enormous congestion of the lungs, œdema of the lungs, with more or less collapse. The collapsed portion is smooth, non-crepitating, and is said to be sometimes softer than usual, like gangrene, but without fetor.[1] Sometimes membranous exudations are found in the bronchi, not unlike those of croup.

If pleurisy and pneumonia have occurred, the usual post-mortem appearances of those diseases are present. The pneumonia is sometimes lobular (or possibly this statement has arisen from lobular collapse not being identified) or lobar, and is often double.

Prognosis.—The very young and the very old bear Influenza badly, especially the latter. Persons with chronic bronchitis, emphysema, and fatty heart, are bad subjects.

In persons without such complaints the danger is chiefly connected with the state of the lungs. Great dyspnœa, very weak voice, impossibility of coughing up the tough sputa, and duskiness of the face, are unfavorable signs. If the pulse becomes early very feeble and slow, and then unequal and intermittent, it shows that the heart is not receiving its due supply of blood on account of the lung-congestion.

The fever and the nervous symptoms seldom kill, yet in some epidemics there have been frequent delirium, convulsions,

[1] Especially in very aged persons: Greene, in Graves's Clin. Med. vol. i. p. 438.

and fainting, and these have always been found to be very bad symptoms.

As favorable signs may be noted, copious warm sweats, loose so-called concocted sputa, spontaneous diarrhœa, and urine with copious red lithates.

In the case of pregnant women there is danger of abortion and subsequent hemorrhage.

TREATMENT.—*Preventive Treatment.*—No means are yet known by which Influenza can be prevented. Unfavorable hygienic conditions, and especially overcrowding, heighten its prevalence and its severity; but persons in the most favorable circumstances may be attacked. Perhaps persons in well-warmed and yet ventilated houses escape best. It has sometimes been noticed that persons exposed by work to the weather suffer most: hence it may be a rule that those persons who can do so, should be more within the house during an epidemic; but as bedridden persons are not infrequently attacked, this is no guarantee.

Treatment of the declared Disease.—Regimen.—It is of great importance to have the room cool and well ventilated. Pearson, whose little work on Influenza is one of the most practical which has ever been written, pointed out, in 1803, the difference in this respect between common catarrh and Influenza. In the former case the patient is better in bed in a warm room; in the latter case, if the patient is not too ill, it is better to get him out of bed after the third day, and to place him on a sofa.[1] Draughts or chills must be, however, most carefully avoided, on account of the risk of pneumonia.

As there is usually almost complete anorexia, it is difficult to give much food. The common custom of giving hot beef-tea is an extremely bad one; it invariably increases the headache and languor, and, as Pearson pointed out, any warm food which forces sweating appears not only to be useless, but to do harm. Solid meat also should be abstained from for two or three days in bad cases. Several writers recommend vegetable food for four or five days. Plenty of cold drinks, especially sub-acid fruits, oranges, lemon-juice, cream-of-tartar water, raspberry vinegar, weak citrate of potash, and citric acid flavored with sugar, barley-water with lemon juice, infusion of mallows or althea, and drinks of the like kind, should be given *ad libitum*, and when there is fever they should be iced. Very weak cold white-wine whey is a very grateful drink. Some good writers speak strongly against the practice of stimulants early in the disease, in all young persons; the great languor and weakness often lead to their use, but it seems probable that they do harm. If stimulants seem indispensable, claret or hock, with seltzer water, is the best. In old persons it may be necessary to use stimulants earlier and more freely.

Stimulants must however be given, and often given largely, in the later stages, if the heart fails, and especially if there are symptoms of intense lung congestion and asphyxia. Brandy, with ammonia, must then be freely used.

As soon as the severity of the fever is passing away, patients should be made to eat; the appetite is still bad, but they will generally take food. Care should be taken not to derange the stomach by too great quantity or variety of food, of which there is some danger.

No experiments have yet been made, to my knowledge, on the effects of cold affusion in the stage of fever; but possibly it might be useful. The wet sheet has been used, and apparently with some benefit. For old people, Schönbein used to order warm baths or warm fomentations.

Keeping the air of the room moist, by conducting the steam from a boiling kettle into it by means of a tube, or by putting boiling water into flat shallow vessels, appears to ease the cough. Also, as in common catarrh and bronchitis, the inhalation several times daily of hot steam is most useful. The old inhaler of Mudge, with the hollow handle, and the valve in the cover, or any of the new inhalers, may be used. If they cannot be obtained, breathing through a sponge dipped in hot water is the best way.

Drugs.—Slight cases require almost nothing; a little cooling saline medicine, citrate and acetate of potash, nitrate of potash, &c.

In severer cases treatment must be more active.

Blood-letting seems always hurtful, and this was noticed so long ago as in the epidemics of 1580. Hardly a writer of any note has failed to make the same remark. The fever is not relieved, the nervous depression is increased, and the risk of the lung congestion and paralysis is augmented. Even with supervening pneumonia, in the old days of bleeding, blood was very seldom taken more than once. If cupping or pneumonia come on with severe pain, a few leeches or a pleurisy glass to the painful part are often useful, but depletion should hardly go beyond this.

A dose of calomel, one to three grains, according to circumstance, repeated once, but not oftener, should be given at first, and may be followed by a saline purgative. Pearson strongly recommended this, and

[1] In some of the older epidemics the practice in England was to keep the patient extremely hot in bed, and to give calefacients. 1801.

various writers have endorsed the practice. The calomel generally brings away copious dark-colored motions, after which the patient is much better in spirits, and the fever abates. But neither mercurial nor other purgatives should be too freely or repeatedly given, as the intestinal mucous membrane is irritable. Repeated catharsis is sometimes most injurious. The substitutes for mercury, podophyllin, jalapine, &c., have not yet been tried in Influenza, and it is impossible to say whether they will be more or less useful.

In children, gray powder must be substituted for calomel, or, what seems better for them, clysters of warm water, with a little castor-oil, may be used.

From the good effects of one or two doses of mercury some have proposed to continue to give mercury, stopping just short of salivation. But this is bad practice; there is no evidence that these small repeated doses of mercury are useful, and it is impossible to be sure that salivation will not come on before we are aware, with all its evils.

Emetics, at the onset, have been very strongly recommended, and in the older epidemics an emetic of antimony and ipecacuanha was invariably given, when the patient was first seen. If there is much nausea an emetic is useful, and perhaps may be so in all cases; but there is one disadvantage, it occasionally produces great and permanent irritability of the stomach, so that it is afterwards difficult to check the constant vomiting. Tartar emetic has been chiefly used, but it causes much depression. On the whole it seems undesirable, as a rule, to give emetics.

After the bowels have been well acted upon, the best remedies to give in common cases seems to be nitrate of potash mixed with lemon-juice and sugar. It seems most useful to give it highly diluted, so that it may be taken as drink. From 60 to 120 grains, in twenty-four hours, may be given to an adult.

Supposing the chest symptoms are not urgent, nothing else need be done; but if the lung congestion is considerable and the cough very hard, some expectorants must be used. Of these ipecacuanha seems on the whole the best, and can be combined with conium or henbane, or with the etherial tincture of lobelia. (Blakiston.)

Tartar emetic, as an expectorant, has been strongly recommended, but it appears to be too lowering in many cases. In the epidemic of 1847 I found it to be of little service, and sometimes to cause irritation and congestion of the intestinal mucous membrane. It is, I believe, better avoided altogether.

Opium requires to be used in bad cases with the greatest caution. There has been much discrepant evidence as to its employment, but on the whole it seems, as Pearson pointed out, best to defer its use till the later stages. If given early it increases the tightness across the chest and the difficulty of breathing. At a late stage, when the expectoration is coughed up easily, and all danger of great lung congestion seems passing off, opium with ipecacuanha quiets the paroxysms of cough, and gives great ease.

Sometimes, however, when the cough is extremely violent, and conium and henbane do no good, opium must be given. In fact the cough itself, simply as a mechanical agent, excites an unfavorable effect on the congested lung, and must be stopped. Then Dover's powder, with nitre and lobelia, should be given; if this does not answer, the liquor morphiæ muriatis or the bimeconate of morphia with ipecacuanha, in large doses, must be used.

Squills seem decidedly hurtful till quite the latter stages.

If there is great tightness across the chest, sinapisms and warm bran poultices or warm water fomentations must be constantly used. Sharp stitches in the side, if pleuritic, must be treated with sinapisms and warm poultices, or, if very severe, with a few leeches, followed assiduously by warm fomentations. If no friction-sound can be detected, they are intercostal neuralgic pains, and are soon relieved by warmth, opium, and chloroform, applied externally.

In the latter stages, if the expectoration is profuse, the cough still violent, and the strength failing, senega and serpentaria, mixed with light wines, seem to be very useful. Ammonia must also be used. If the expectoration continue extremely profuse, the acetate of lead, with a little opium is useful.

Some of the older writers thought that cinchona bark was hurtful in the earlier stages, but in some of the late epidemics quinine appears to have been found useful throughout. Whether this be the case or not, it seems clear that immediately the acute stage is passing off quinine should be freely given. It does good service against the neuralgic pains which are often troublesome at the commencement of improvement. [The employment of quinine in Influenza has now become extremely common in America, even in non-malarious districts. Beginning with it upon the occurrence of the first symptoms, doses of from three to five grains every two or three hours, until ten or twelve grains have been taken, will, if not abort, at least mitigate the violence of an attack. Should this, with other palliative treatment, not avert or arrest the disorder at the beginning, quinine will, throughout its course, do the most good in small or moderate doses; not more than eight or ten grains in a day. The early *abortive* practice, however, with quinine seems to

be well worthy of general trial, not only in epidemic Influenza, but in those sporadic catarrhal attacks, which, under the name of "catching cold," are common everywhere.—H.]

Warm plasters between the shoulders have been much praised by some writers (Legendre). Blisters do no good, and add to the patient's sufferings.

Inhalations have been tried both for the cough, sore throat, and nasal soreness. Pearson used the vapor of ether, which he had found very useful in common catarrh; it was not so good in Influenza. Chloroform, in small quantities, may relieve the tightness and the violence of the cough. Inhalation of steam has been already noticed. In future epidemics it would seem very desirable to try various inhalations to act on the membranes of the nose, pharynx, and lungs. It is impossible *à priori* to say whether they could be of any use, but small quantities of chlorine, iodine, carburetted hydrogen, even perhaps sulphurous acid, might be tried. The naso-bronchial mucous membrane is very accessible to such influences, much more so than to medicines introduced into the blood.

It may be a question also whether some local applications could not be made to the membranes of the nose and throat, such as solutions of iron, catechu, or alterative substances of that kind. Possibly the local disease might be thus partly checked.

The use of sulphites of potash and soda may also be suggested as a local application to the throat and nose.

Complications. — It is very doubtful whether pneumonia is benefited by bleeding; the pneumonia has itself a course, and cannot be cut short; it is probably better to persevere with ipecacuanha and nitre, and to apply only a few leeches or a cupping-glass if pleuritic pain be intense.

In double capillary bronchitis bleeding is hurtful; the great danger is suffocation; brandy and ammonia, with valerian, and lobelia inflata, must be freely used. Sometimes, even in cases of exhaustion, it is necessary to give an emetic, as the thick secretion blocks up the tubes; sulphate of zinc and ipecacuanha is then the best emetic.

In obstinate vomiting, hydrocyanic acid, and very small doses of morphia, with effervescing draughts, will generally suffice.

Excessive diarrhœa must be checked, but moderate diarrhœa does good, and is indeed a favorable sign, especially on the third or fourth day.

In suppression of urine, a very hot bath and copious draughts of linseed-tea, with a little liquor potassæ, or chlorate of potash, must be given.

If there be intense headache and stupor, purgatives, cold applications to the head and a few leeches, either to the temples or the Schneiderian membrane, will often give relief.

If there be much coryza and great pain in the nose and frontal sinuses, a few drops of solution of muriate of morphia in a little water, sniffed or injected up the nostrils, will give relief.

If rheumatic symptoms come on, colchicum is said to be useful (Peacock) in small doses (4 or 5 minims of the tincture of the seed), given every 3, 4, or 6 hours, with ammonia and opium. Iodide of potassium with colchicum is also sometimes useful.

Convalescence.—Iron and quinine must be given for some time in small doses.

A very nutritious diet, beer, and wine, must be employed. Milk in large quantities is very useful. Milk and seltzer water is a favorite German remedy.

The skin must be very warmly clothed, as it is very sensitive.

If there is much dyspnœa left behind, the alcoholic or etherial tincture of lobelia should be used. Flying pains of the chest are best treated by opiate fomentations, or a liniment of acetic acid and oil of turpentine, recommended by Dr. Stokes.

If a paroxysmal cough is left behind, with copious and rather viscid expectoration, ammoniacum and opium should be given.

HOOPING-COUGH.

By Edward Smith, M.D., F.R.S.

Definition.—A convulsive cough consisting of a series of forcible expirations, followed by a deep, loud, sonorous inspiration, and repeated more or less frequently during each paroxysm; occurring usually in childhood, and once only during life, and continuing several weeks. Cullen's definition is, "Morbus contagiosus, tussis convulsiva, strangulans, cum inspiratione sonora, iterata, sæpe vomitus."

It is popularly known in England as Whooping-cough, Kink-cough, Chincough; in France, Coqueluche, and in Germany, Keuch-husten and Kik-husten; from the sonorous inspiration which marks it; and technically, as Tussis convulsiva (Willis and Sauvages) and Pertusis (Sydenham and Cullen).

History.—It is difficult to believe that a disease having characters so well and easily defined could have been known to the ancients without a description having been recorded by which we might now recognize it; and, as no writer before the middle of the seventeenth century has described it, we are led to the conclusion that the disease was unknown to the fathers of medicine, or that it has acquired one of its chief characteristics since their day. Diseases having a contagious or epidemic character, and resembling Hooping-cough in its catarrhal symptoms, were known to Hippocrates and others before the Christian era, and have been described by Arabian, Italian, and French authorities down to the sixteenth century; but, lacking the distinctive character of the Hoop, they more nearly resembled influenza than any other disease now known to us. Hence the history of the disease cannot be clearly traced back to a period earlier than that of Willis, from whom we have received not only the first description of it, but one which in all respects is applicable to the disease as it exists at this day; yet, as from his definition "Tussis puerorum convulsiva seu suffocativa, et nostro idiomate, *chincough* vulgo dicta," it is probable that the disease was then commonly known to the people, we may infer that it had existed in England some time before he described it. Dr. Gibb avers, but without citing authorities, that it has been known traditionally among the French Canadians for more than three centuries; and, as they are presumed to have received it from France, he affirms that the disease to which Mezeray gave the name *Coqueluche* in the fifteenth century, was truly Hooping-cough notwithstanding the absence of the distinctive Hoop in that author's description of it.

Causes.—There is no known specific cause to which it can be attributed; but that atmospheric influences are its chief exciting causes may be inferred from the facts that it has often occurred as an epidemic, and is most prevalent at certain seasons of the year. The imperfection of our knowledge in reference to atmospheric influences, other than temperature, and the absence of registration of the prevalence of diseases which do not end fatally, prevent a more minute inquiry into this relation. The fact that Hooping-cough is, without reasonable doubt, a contagious disease, implies that a *materies morbi* generated, or at least acting, within the body, is communicated from one to another person, and that the atmosphere is also the vehicle for its transmission; but as we know nothing of the nature of this *materies morbi* within the body, so are we equally ignorant of its characters when existing without it. Further, we do not know with any precision the period of incubation during which the communicated poison is imperceptibly acting within the body, but it probably does not exceed ten days.

The influence of childhood in the causation of the disease must also be cited. In our analysis of the deaths from Hooping-cough, published in the Medico-Chirurgical Transactions for 1854, it was shown that Hooping-cough was the most mortal of all diseases of children under æt. 1 year; that 68 per cent. of all the deaths from Hooping-cough occurred under æt. 2 years; and that only 6 per cent. of the deaths were recorded after æt. 5 years.

But here again our knowledge is most limited and vague when we attempt to analyze the conditions attending early life which may be presumed to lead to the occurrence of the disease. It is summed up in the phrase "great excitability or impressionability of childhood," by which all influences are asserted to exert special power at that period. But it applies with equal force to the occurrence of other diseases in childhood which have but little

affinity with Hooping-cough in its leading characteristics. Yet it accords well with the generally adopted views as to the immediate cause of the cough, to which we shall presently refer, and is further supported by the fact proved in the paper just quoted, that the disease when fatal prevailed more in females than in males —in the sex in which this special character of childhood is the most marked.

Nature and Seat.—The intimate cause, or the nature and seat, of Hooping-cough is variously regarded, as one of the two leading characters of the disease—the catarrhal or the convulsive—is the more urgent; but with literature rich in authorities the preponderance of opinion is in favor of the essentially nervous nature of the disease. This opinion has been held by Hoffmann, Hufeland, Lobenstein, Löbel, Paldame, Wendt, Jahn, Cullen, Leroy, Guibert, Webster, Pinel, Todd, Gibb, and Copland. The immediate seat of this nervous irritation has been very variously ascribed to the stomach (Chambon and Broussais); to the lungs (Wendt and Paldame); to the diaphragm (Millot); to the pneumogastric nerves (Hufeland and Hoffmann); to the phrenic nerve (Jahn); to the medulla oblongata (Copland); to the brain and its membranes (Webster); and to the general nervous system (Guibert); but several of these authorities included more than one seat in their description.

The most characteristic views of recent date are perhaps those of Guibert and Copland, to be found in the renowned Dictionary of Medicine of the latter. Guibert "considers that a common cough may pass into this affection by having the spasmodic state of the muscles of the larynx and of the diaphragm superadded to it; and, therefore, that spasm superadded to cough constitutes the disease—the state of spasm resulting from the high nervous susceptibility and particular disposition to it existing in children, and from individual idiosyncrasy." "The increased secretion of mucus he refers to an excited state of the mucous membrane of the air passages . . . existing independently of any inflammatory action . . . the nervous symptoms being the result of the spasm, which he considers the chief agent of the morbid phenomena." Dr. Copland writes: "I believe that the disease is chiefly nervous in the simple cases; that it preserves this character more or less throughout, even when inflammatory complications ensue; and that in the uncomplicated state the nervous affection never proceeds beyond irritation. . . . The inflammatory appearances in the medulla oblongata and base of the brain may be owing to the functional relation of these parts to the respiratory order of nerves which receive the first impression of disease."

The writers of high repute who give greater prominence to the catarrhal or even inflammatory nature than to the nervous character of the disease, are Laennec, Dewees, Guersant, Watt, and Badham. Dawson believed that the inflammation was restricted to the glottis, whilst Desruelles, with many others, regarded Hooping-cough as beginning with bronchial inflammation and advancing to cerebral irritation. Many writers, with Guersant, believe that the inflammation is of a specific kind; but the chief distinction which they draw between this and ordinary bronchitis is the marked character of the spasm, and the other evidences of nervous irritation—evidences which coincide more with the views of those who believe in the nervous nature of the disease, than with those who consider the disease to be essentially inflammatory.

In a disease in which these two main characteristics exist, there are doubtless grounds for difference of opinion as to their relative importance, and particularly when their respective influences vary in different cases and in different epidemics, and when observers, by their special studies, are led to regard cases from different aspects, as the nervous, inflammatory, and pathological. Those who adopt the opinion that Hooping-cough is essentially a disease of the blood, and is due to a morbid poison existing in that fluid, regard both the nervous and the catarrhal evidences as of equal importance, but with this difference in their aim—that the former are direct evidence of the action of the poison, whilst the latter are the throes of the system to rid itself of the poison by secretion from the mucous membrane. Without denying the existence of a specific poison, and without admitting that the supposed poison is eliminated by the mucous membrane of the bronchi, we do not doubt that that feature which gives character and importance to the disease is the nervous or spasmodic one, and that in any uncomplicated case, when this has been abated, the disease is shorn of its specific characters and dangers.

SYMPTOMS.—On proceeding to state the symptoms of the disease, it becomes necessary to divide them into two classes: those of the simple and those of the complicated form of the disease.

Simple Hooping-Cough.—The early evidences are those of simple catarrh without any, or with scarcely any, febrile complication. They are coryza, secretion from the nose, cough, more or less severe, but not at this stage spasmodic, with frothy and watery secretion from the bronchi, lassitude, restlessness, and some diminution of appetite. After a period varying from one to two weeks the cough becomes

a more marked symptom—is louder and more prolonged than an ordinary cough, and generally assumes a spasmodic character. When the nature of the disease has become quite clear, the cough is found to occur in paroxysms, during which the body is bent forward, and a series of short, very rapid, and violent expirations occur, and are continued until the face is extremely suffused and the respiration seems almost to have ceased, when a deep, prolonged, loud, and crowing inspiration takes place. This alternation occurs two, three, or more times in each paroxysm. The attack terminates with the emission of a somewhat large quantity of semi-transparent glairy and very tenacious mucus, which hangs about the mouth and lips, and not unfrequently with vomiting. At a yet later period the pertinacity of the expiratory effort is diminished and inspiration occurs more frequently, whilst the secretion, although still abundant, is more opaque and less tenacious, and vomiting less rarely occurs.

During this period the peculiar character of the sounds with the cough somewhat subsides, and in progress of time it is omitted from some of the attacks—the relative frequency gradually diminishing until it altogether disappears, and the cough has no longer any special characters. In mild cases the disease may soon end; but in more severe cases there remains much exhaustion and emaciation, with defective appetite and increased sensibility of the stomach, which leads to vomiting from trivial causes. The rate of pulsation is increased in a most marked manner during the attack, so that in very severe paroxysms it is too great to be counted; yet it is not due to any inflammatory or febrile condition, but to the mechanical interference with respiration. In the intervals it assumes a normal state, except when the system has become much enfeebled. The force of the heart's action is the greatest at the commencement of each paroxysm, and diminishes sensibly when the rapidity of pulsation is the greatest; and it is also lessened when the disease has been prolonged and the system much exhausted.

The skin is usually soft, and at the end of a paroxysm is bathed in perspiration. It is also usually cool and highly sensitive to low temperature.

Bleeding from the nose is a very frequent attendant upon a severe attack of Hooping-cough; and whilst it shows how great is the interference with the circulation, it is often a most valuable remedy.

The period of the occurrence of a paroxysm is uncertain, but it is particularly liable after a meal, when the stomach is full and the action of the diaphragm is impeded, and when food of slow digestibility has been eaten. If the child be very young, the cough is excited when the nurse throws it about; and if older, crying or seeing another in a paroxysm will bring on an attack. The paroxysms are more frequent in the day than in the night. Hence there is usually much interference with nutrition, and consequent loss of flesh, whilst the lassitude extends to exhaustion and prostration of the system.

In ordinary cases the child regains much of its spirits and healthful appearance between the paroxysms, and runs about, plays, and eats almost as in health; but when the paroxysms are severe, the face remains suffused, the eyes injected, and the surrounding parts swollen during the intervals; whilst loss of strength is proportionate to the constitutional feebleness and the early age of the child, the vomiting, and the duration and violence of the disease.

In a typical case the catarrhal symptoms, without spasmodic cough, continue about two or three weeks, and the spasmodic cough three to four weeks; whilst after the spasm has ceased and the cough has become again catarrhal, the duration may be short, if the child have not been too much enfeebled, and prolonged for some weeks, if otherwise.

Complicated Hooping-cough.—The complications are of two classes, viz.: when Hooping-cough supervenes upon another disease and complicates it; and when, the Hooping-cough being primary, other diseases arise in its course. The former class is a somewhat extensive one, and is for the most part limited to diseases which involve bronchial affections in their course; but the latter only will be considered here.

The complications are of four kinds, viz.: disease of the lungs, disease of the brain, infantile remittent fever, and vital exhaustion. The last may by some be regarded as one of the sequels of uncomplicated Hooping-cough; but when it is considered that the almost infinite proportion of the cases of simple Hooping-cough end favorably, with only a moderate state of exhaustion, it will be thought better to regard the very exceptional occurrence of fatal exhaustion as a complication rather than as a sequel to the simple form. The pulmonary complications are, congestion of the lungs, emphysema, atrophy, bronchitis, and bronchopneumonia.

A certain amount of congestion of the lungs is found in all severe cases of Hooping-cough. It is due perhaps exclusively to interference, through the respiration, with the pulmonary circulation, and it is one of the sources of danger attending the disease; but in the degree in which it becomes a complication, the dyspnœa and frequency of respiration are increased and

continue during the intervals, the discoloration and suffusion of the face are more marked, the pulse is feeble and rapid, and the exhaustion of the system is greatly increased. Hæmoptysis of a more or less severe kind sometimes occurs and yields temporary or permanent relief. Physical examination of the chest shows that the respiratory sounds are somewhat more feeble than in simple Hooping-cough, and there may be a shade of dulness on percussion; but unless effusion occurs into the lung-parenchyma, the physical signs are not very marked. There are not any marked signs of fever.

Emphysema, although usually regarded as a sequel of the disease, is a frequent concomitant of the severe forms, and particularly in the children of parents who have been afflicted with chronic bronchitis. Its production is mechanical, as in the case of adults, and occurs from the forcible compression of the air in the lungs, which is effected by the diaphragm and other expiratory muscles, whilst an obstacle is opposed to the egress of the air. This obstacle is most commonly only the ordinary one which exists in the larynx and pharynx, and is a necessary part of the act of coughing, as shown in my paper on the "Closure of the Larynx at its Upper Orifice," in the *Journal de Physiologie*, and as seen at the Rima Glottidis by the laryngoscope; but it may also be produced by the plugging up of a large bronchus after the part of the lung to which it leads has been distended with air. The result of this condition is to increase the dyspnœa and to render it permanent in proportion to its extent, and if it exist in any considerable degree the respiratory sounds will be lessened and the resonance on percussion increased.

Atrophy of a part of the lungs is a not unfrequent complication of Hooping-cough. It results from closure of one or more divisions of the bronchi, by which the ingress of air to a part of the lung is prevented, and the space thus left unoccupied is filled up by the undue expansion of the adjoining cells. When the part thus rendered useless is considerable, the gravity of the complication is great, and it may be detected by the diminished expansion of the intercostal space, and by the absence of respiratory sounds over the part atrophied; but when it is small, the encroachment of the adjoining structure prevents the occurrence of distinct physical signs.

Bronchitis and broncho-pneumonia are, however, the more frequent and fatal lung-complications of this disease. In the paper already quoted from the Medico-Chirurgical Transactions it was shown clearly that deaths from Hooping-cough were almost exclusively due to these diseases, and that they did not correspond at all with the rate of mortality from zymotic or nervous diseases. In both there are evidences of fever in the varying degrees of heat and dryness of the skin, and in the rapid pulse both during the paroxysms and in the intervals. The cough is more frequent and not always spasmodic, and the dyspnœa is more permanent. Discoloration of the face, enlargement of the opening of the alæ nasi, difficulty in speaking, and panting respiration are more perceptible as the complication is severe. The only change in the physical signs is an increase in the moist râles, whilst with broncho-pneumonia there is a more or less persistently localized state of this sign accompanied by some amount of dulness on percussion and lessened respiratory sounds. The general exhaustion and loss of appetite are more apparent.

The brain complications are convulsions and hydrocephalus. It has already been shown that, in the opinion of very able physicians, irritation of the brain and its membranes, and particularly of the medulla oblongata, is so common as to be an integral part of the disease. Usually, however, there are no signs of this state other than the reflex condition which excites the spasm of the glottis; but in no inconsiderable number of complicated cases convulsions occur with or without hydrocephalus.

The occurrence of convulsions cannot usually be predicted; but if the child be teething or suffering from derangement of the bowels, if during the spasm the thumbs be drawn inwards, and during the intervals the discoloration of the face continue without lung-complication, and if there be a marked degree of exhaustion or oppression following the paroxysm, or the eyes be intolerant of light, this complication may be imminent. They are in some cases due to irritation of the membranes of the brain and medulla oblongata, and in others to congestion of the brain, due mainly to interference with the pulmonary circulation.

Hydrocephalus is so frequently a constitutional affection that the relation of Hooping-cough to it is rather that of an excitant of a pre-existent predisposition. The signs are often obscure at first, but in many cases the occurrence of drowsiness, headache and starting during sleep, convulsions, increased heat of skin and rapidity of pulsation, intolerance of light and lessened mobility of the pupils; and in others the persistent disposition to vomiting on being moved, will indicate the advent of this most important complication. The breathing is more irregular and accompanied by sighs, than it is in simple Hooping-cough; and if there be no lung-complication existing at the same time, the diagnosis will not long remain

doubtful. When the convulsions are restricted to one side of the body, or when paralysis of one side occurs either with or without simultaneous convulsions on the other, the evidences are still clearer.

[Occasionally, even in children, *apoplexy* occurs, under the violent disturbance of the circulation of the head during the paroxysms of Hooping-cough. Such a case occurred in the neighborhood of Philadelphia, a few years since, in a child not more than eight years of age, with a fatal result in less than twenty-four hours, the symptoms not having been previously unfavorable.—H.]

The complication with infantile remittent fever is most generally found when the latter disease prevails, and when there have been evidences for some weeks of a disordered state of the bowels. The tongue is coated, the breath foul, the evacuations unhealthy, and the bowels are tender on pressure and tumefied. The patient does not recover health and strength, but, with or without introductory rigors, slowly exhibits signs of fever, having the exacerbations and remissions distinctive of remittent fever, and wanting all the diagnostic signs of bronchitis. Such cases are usually protracted in their recovery, and demand very careful and able supervision.

The complication of excessive exhaustion is most frequent in children of very weak constitutions, or in those which have been enfeebled by previous disease. In such, prostration is a marked feature even during the catarrhal period, but when the spasm has fairly set in it is extreme after every paroxysm. Careful examination into the state of the lungs, brain, and bowels fails to offer any satisfactory reason; and this, with the absence of fever, suffices to indicate the complication to which we refer. Moreover the skin is unusually soft, cool and liable to perspiration, and the appetite is inadequate to the nourishment of the system.

Diagnosis.—The distinguishing feature which marks Hooping-cough is undoubtedly the paroxysm of spasmodic cough, whether the sound accompanying it amount to a distinct whoop or not; whilst the accidental (as opposed to essential) symptoms are the preliminary catarrh, the glairy tenacious secretion from the bronchi, the early age of the patients, and the general course of the increase and decline in the severity of the paroxysms. In the early catarrhal stage, it cannot be distinguished from a common cold.

Pathology.—The pathology of this disease has been already hinted at in our account of its history. In reference to simple Hooping-cough the aim has been to determine the cause of the spasm of the glottis and the closure of the larynx, with the prolonged expiratory action of the diaphragm, which are the prominent features of the disease. All writers have regarded the nervous system as the source of this influence, and have speculated upon the part which was principally involved. Thus the recurrent laryngeal nerve as affecting the larynx, the phrenic as controlling the diaphragm, the medulla oblongata as controlling all the respiratory movements by the pneumogastric nerve, and the whole brain and its membranes, have each in their turn been cited as the seat of this disease. The blood has by others been assumed to contain a zymotic poison which, acting upon the nervous system, excites spasm, and upon the mucous membrane of the bronchi, causes cough, with a secretion whereby it is to be ultimately cast out. We cannot yet, however, arrive at a sound conclusion upon this question, although there are many points of striking similarity between Hooping-cough and some recognized forms of zymotic disease.[1]

The interference with the circulation of the blood which occurs when the respiration is so greatly impeded and the lungs so largely emptied of the residual air as in severe cases of Hooping-cough, is doubtless the cause of the complications both in the head and the lungs to which we have referred; whilst the exhaustion which follows each paroxysm, and the interference with nutrition, are the causes of the remaining complications. The mechanical act of coughing whilst the larynx and some of the bronchi are closed produces emphysema of the lungs and dilatation of the bronchi; and closure of the tubes by secretion may lead to atrophy.

Morbid Anatomy.—In nearly all fatal cases death occurs not from the Hooping-cough, but from its complications; and the morbid signs will therefore be those of the supervening diseases, and must be sought for in other parts of this work. Dr. Copland believes that in all cases there are inflammatory appearances about the medulla oblongata; and it cannot be denied that congestion of the bronchial, laryngeal, and faucial mucous membrane is always present. From what has been already stated, it may be affirmed that there are evidences of bronchitis or of

[1] [The analogy to Hydrophobia is not unimportant; in the combination of a nervous disorder with a respiratory affection; the length of *duration* in Hooping-cough corresponding, also, in some manner to the protracted *incubation* in Hydrophobia. Instead of the cough, in Hydrophobia, we have spasmodic, gasping inhalations, produced by any sudden impression made upon the senses.—H.]

broncho-pneumonia, with atrophy or collapse of lung, in by far the greater number of fatal cases.

Prognosis.—The prognosis depends upon the age and strength of the patient, the severity of the spasm, and the presence of particular complications. In simple Hooping-cough occurring in childhood, with moderate spasm and with an average state of health, it may almost always be regarded as favorable, and the disease may be expected to leave no ill effects behind. When it occurs in an infant under 4 months old it is very liable to induce head symptoms, unless the attack be a very mild one; and when occurring in adult life, it is more likely to lead to chest complications and to leave permanent changes in the structure of the lungs.

When the paroxysms are unusually prolonged and the spasm very severe, complications are almost sure to arise; and if the child be feeble, the prognosis must be given with caution.

The complication of bronchitis renders the prognosis unfavorable only in proportion to its severity. When it occurs in a moderate degree and without inducing much dyspnœa in the intervals between the paroxysms, and at a season of the year when the temperature is not very low (the deaths from both Hooping-cough and bronchitis are inversely as the temperature), the gravity of the case is not seriously increased; but when it involves both lungs, and is attended by much dyspnœa and increased lividity of the countenance, the prognosis becomes unfavorable. Extension of the inflammatory condition to the substance of the lung adds much to the danger, from the fact that broncho-pneumonia is usually less amenable to treatment than bronchitis alone.

When emphysema and enlarged bronchi have already occurred, or when it is probable from the severity of the spasm that they will occur, permanent dyspnœa to a greater or less extent may be apprehended.

The occurrence of head symptoms attended by convulsions or paralysis must always render the prognosis unfavorable; but with convulsions not due to hydrocephalus, many cases recover.

The cases in which disordered secretions and intermittent fever occur will certainly be protracted, and may leave an enfeebled state of system from which the patient will not entirely recover, although life may be continued for many years. It is probably a less fatal complication than the others referred to, but causes much anxiety both to the physician and the friends of the patient.

When extreme exhaustion is present without evident cause, the prognosis should be a guarded one.

It is a fact which is not sufficiently appreciated, that whilst so very large a proportion of cases of Hooping-cough recover, only six other diseases in the London district during the ten years from 1844 to 1853 inclusive, were more fatal than Hooping-cough; viz., phthisis, pneumonia, bronchitis, typhus, convulsions, and scarlatina.[1] Hence at the commencement of any attack of the disease it is well to speak of the future with caution.

Treatment.—There are but few diseases in which so many remedies have been employed as in Hooping-cough, and still fewer in which so much has been confidently asserted of remedies which differ much from each other. This is owing probably to the fact that the disease, running a more or less clearly defined course, usually subsides either with or without medical treatment, and with or in spite of whatever remedies may have been employed, and also that many remedies diverse in name and appearance are closely allied in their modes of action upon the system.

It is highly probable that many have been recommended on empirical grounds only; but others have been intended to effect one of the following objects: namely, to abate inflammatory action, to promote expectoration, and the elimination of the supposed morbid poison, to diminish the bronchial secretion, and to allay the spasm, directly or indirectly, through the invigoration of the general system.

Those to abate inflammatory action have been leeches and antimony; to promote expectoration, antimony, ipecacuanha, squills, and other emetics; to lessen secretion, alum and zinc; and to allay spasm directly, hydrocyanic acid, conium, hyoscyamus, belladonna, opium and morphia, musk, valerian, ether, and chloric ether, with various liniments supplied to the chest and back, and a strong solution of nitrate of silver to the throat; to allay spasm indirectly, iron, zinc, copper, silver, and other metallic salts, mineral acids, with quinine, and other vegetable tonics, and change of air. Some were probably expected to have a specific action, as tar-water and tar inhalations, the air in the neighborhood of gas-works, salts of lead and cantharides.

Simple Hooping-cough.—In the treatment of an uncomplicated case of Hooping-cough, the chief, if not the sole, aim should be to allay the spasm, and thus to prevent complications which result from it, and reduce the disease to a common cough.

As a preliminary step it is needful to

[1] Medico-Chirurgical Transactions, 1854, p. 230.

regulate the functions of the chylopoietic viscera, to prevent or remove fecal accumulation, to promote a proper secretion of bile, to prevent the occurrence of an excess of acid in the secretions of the stomach and œsophagus, and to prohibit the use of indigestible or irritating food. For these purposes, castor oil, or carbonate of magnesia, or Dinneford's or Murray's soluble magnesia, should be given daily or every second day, until the necessity for their use has ceased; and an occasional dose of one or two grains of hydrarg. c. cretâ may be given at bedtime. The state of the nurse's milk should be examined in the case of suckling infants, and at a later period the food should be restricted as far as possible to cooked milk, and given alone or in puddings. Two or three pints of milk may be taken daily, but in such a manner that the quantity given at a time shall not exceed one-quarter of a pint, and the intervals between the supplies be short. Bread should be rarely if ever given, except when cooked with milk. The use of vegetables should be greatly restricted. Meat in small quantities, and cut into very small pieces, may be given to a child of three years of age and upwards; and eggs made into puddings, and beef-tea, may be allowed at any age if there be a deficiency in the supply of milk.

In the general management of the child the body should be kept properly warm by clothing, and the atmospheric air should not have a lower temperature than 64° either by night or day; and so long as the aim is to allay the spasm, the patient should be kept absolutely quiet both in mind and body, or as quiet as may be possible. [When the general symptoms are mild, fever and inflammatory bronchial disorder being absent, there is advantage in the child being taken into the *open air*, in good weather, every day. Often, the paroxysms will be very few and moderate while the patient is out of doors, becoming more frequent and severe under confinement in the house.—H.]

Such being premised, the next step depends upon the view which is taken as to the possibility of shortening the course of the disease. Some of the most eminent physicians of the present day are of opinion that it will run its course, and that the duty of the physician is to conduct it evenly and safely to the end.[1] With this view it is only possible to use palliative remedies, such as small doses of ipecacuanha, with or without rhubarb; but if a belief is entertained that the progress of the disease is amenable to treatment, the proper course is to select that sedative, narcotic, or antispasmodic agent, the action of which is the most uniform, and the dose capable of proper regulation; and the aim should be to administer it with the frequency and dose which will allay the spasm and *keep it under control* without interfering materially with any vital action.

[In American practice, *assafœtida* is used, probably, in mild or moderate cases, as largely as any other antispasmodic medicine. It may be given as soon as the paroxysmal character of the disorder is shown, in combination with ipecacuanha or squills. For children, a much employed preparation is the "milk" of assafœtida, in teaspoonful doses. It is a mild remedy, less powerful as an antispasmodic than musk, belladonna, &c., but, in ordinary cases, it seems to be often serviceable.—H.]

Hydrocyanic acid has been strongly recommended by, amongst others, Drs. Granville, Hamilton Roe, Atlee, Elliotson, and West; and of these Dr. Roe has given the most detailed and judicious directions. He prescribes three-quarters of a minim of Scheele's strength to an infant, 1♏ to a child aged three years, and 1½ to 2♏ to one aged ten or twelve years, to be given every three or four hours, or even more frequently when the effect of the previous dose has abated. He has given 1½♏ every quarter of an hour for twelve hours to a girl aged ten years. He attaches great value to its action in reducing fever—a state of system which in simple cases rarely exists; but he also affirms that it will cure simple Hooping-cough quickly, or at any rate abridge its duration. Laurel-water, which contains hydrocyanic acid, has been given in doses of 6♏ to children and 30♏ to adults every two or three hours. Belladonna has been strongly recommended by Boerhaave, Hufeland, Guersant, Trousseau, Williams, Jackson, Churchill, G. A. Rees, and others. The dose of the extract recommended varies from $\frac{1}{12}$ to $\frac{1}{4}$ grain for a child aged two years, and 1½ grain for a child aged four years and upwards; and Dr. Williams, bearing in mind its action upon the iris, and the desirability of diminishing the irritability of the bronchial and laryngeal muscles, regards the action upon the former as the measure of the action upon the latter, and takes it as his guide.

Conium was introduced as a specific remedy for this disease, and has received the support of many distinguished physicians. The dose of the extract most commonly employed is $\frac{1}{40}$ of a grain for a child aged four months, $\frac{1}{30}$ to $\frac{1}{16}$ for one aged one year, and $\frac{1}{10}$ to 1 grain for older children, repeated every six hours. In the use of this drug, as in that of hyoscyamus and belladonna, it is customary to add small doses of ipecacuanha, and some physicians, as Guersant, combine it with the oxide of zinc or other metallic salts.

[1] Sir Thomas Watson's Lectures.

[The *succus hyoscyami*, in doses of a few drops at a time, will sometimes act very well in quieting the paroxysms of coughing, especially at night.—H.]

Tincture of lobelia is now given by Dr. Sidney Ringer with very good effect. The dose is 5♏ for a child æt. one or two years, increasing to 10♏ as the age advances to ten years, and is repeated every hour.

The preparations of opium which have been most generally used are the tincture of opium and salts of morphia; but Battley's sedative solution and codeia have also been much commended. In a paper published in the Edinburgh Medical Journal, May 1856, we took occasion to express the opinion that morphia was the best remedy in this disease, since it is more certain and uniform in its action than belladonna, conium, hyoscyamus, and digitalis, and exerts a less injurious effect upon the sensorium and the bowels than the tincture of opium. It has already been pointed out that Drs. Roe and Williams, when administering hydrocyanic acid and belladonna, understood the importance of giving them in doses sufficient to allay the spasm; but in the paper referred to we endeavored to explain that an essential part of the treatment was to carry this influence just so far as to be evident to an observer, and to maintain it during the period of treatment. With the view that the essential character of Hooping-cough, and that which leads to dangerous complications, is the spasm, and that the removal of the spasm should be the object of the physician, our aim was to cause the slightest oppression of the sensorium as a measure of the required effect of the drug, and to maintain it from three to six days. With children under one year of age the dose of the hydrochlorate or the acetate of morphia should be $\frac{1}{8}$ to $\frac{1}{4}$ of a grain repeated every four hours; with children between one and three years of age, $\frac{1}{4}$ to $\frac{1}{3}$ of a grain; and with those yet older $\frac{1}{3}$ to $\frac{1}{2}$ of a grain. The dose selected should be repeated three or four times; and if no perceptible drowsiness should be induced, it should be increased a step and repeated in like manner, and again increased if necessary until the dose has been ascertained which produces a very slight oppression of the sensorium. The aim must then be to maintain this effect by repeating the same dose, or by further increasing it from time to time. The cases of simple Hooping-cough are extremely few in which *slight* drowsiness has been produced and uniformly maintained for three or four days without the spasm having subsided, and the cough nearly reduced to that of a common cough. The plan, when intelligently carried out, has been most successful in our hands, and in those of Dr. Müller in Germany, as well as in the practice of many in England. It is rarely necessary to add any other remedy; but in certain cases the exhibition of carbonate of soda in addition to the morphia has further lessened the irritability of the larynx and promoted expectoration.

A very favorite combination is that recommended by Dr. Pearson, consisting of one drop of tinct. opii, five drops of vin. ipecac., and two grains of carbonate of soda, to be given every four hours after the operation of an emetic.

Dr. Eben Watson has proposed an admirable plan of treatment in the application of a strong solution of nitrate of silver to the larynx, by which the spasm is quickly relieved. The strength should be twenty grains to the ounce, and the solution applied not only to the tonsils and uvula but to the back part of the fauces, and if possible to the seat of the epiglottis. This is more difficult to effect in very young children, but in children of eight years of age and in adults it may be readily performed by depressing the back of the tongue until the free edge of the epiglottis is seen, and sweeping the pharynx with the camel's-hair brush or the mop charged with the solution. The application should be repeated every second day, and the spasm may be expected to subside in less than a week.

The external application, to the chest and between the shoulders, of belladonna plasters and of liniments containing belladonna, opium, or oil of amber, has been much employed with advantage; but the effects of belladonna must be watched, and dilatation of the pupil restrained within very moderate limits.

The inhalation of ether and chloroform, when diluted with air with the aid of a proper instrument, is often of great advantage in cases where the spasm is very severe and the patient not very young. It should never be carried so far as to induce anæsthesia.

The employment of metallic salts in the treatment of this disease has been very general and extensive, and those of arsenic, copper, and silver have been regarded as specifics. The proper dose of liq. arsenicalis is one drop daily, divided into four doses, for an infant, and one drop twice or thrice a day for a child of five years of age and upwards, and it may be given with water or decoction of cinchona. Carbonate of iron is prescribed by Dr. Graves after recovery from inflammatory symptoms: but others give the sulphate, and employ it in the early stage of the disease. In Dr. Rees's opinion it is particularly adapted to those of a strumous diathesis. Zinc has been given both to lessen the secretion from the mucous membrane and to allay the spasm. The proper dose of the oxide is one grain

three or four times daily for a child aged one year, and two grains four to six times daily for those of five years of age and upwards. Acetate of lead has been particularly recommended by Dr. Rees, and he affirms that ¼ grain given every six hours removes the spasm on the first day of its exhibition.

Sulphuric, hydrochloric, and nitric acids have all been given in this disease with advantage when no inflammatory complication existed. Of these, nitric acid has received the most recent support, and has been given in remarkably large doses. To a tumblerful of thin syrup as much dilute nitric acid is added as will render it as sour as lemon-juice, and of this a child under one year of age may take a dessert-spoonful every hour, and an adult the whole tumblerful in three or four hours. So much as ℈ij to ʒj of the dilute acid is given to a patient ten years of age and upwards, and 10♏ to a very young infant, when well sweetened with honey or sugar. No evil results are said to have followed, and the beneficial effect has been, it is affirmed, proportioned to the dose. Injury to the teeth is averted by using a gargle with carbonate of soda after each dose. This plan of treatment has received the approbation of Dr. Gibb, and its mode of action is presumed to be that of a tonic, sedative, and antiseptic.

Change of air is a very popular remedy, and it is affirmed that it matters little whether it be to a purer or to a less pure atmosphere, but above all others the air in the vicinity of gas-works and lime-kilns has been, even very recently, commended. Except upon empirical grounds, and in the absence of sufficient proof of the benefit alleged, we can advise only that change which country air may offer to children living in towns; and even this exerts no marked influence in otherwise healthy children until the period of recovery from the exhaustion which follows the disease.

Alum and tannin have been much commended with a view to restrain the secretions and allay spasm. The former, when given in the nervous stage of the disease, was exceedingly efficient in the hands of Dr. Golding Bird. His prescription was, alum twenty-five grains, extract of conium twelve grains, with syrup and dill-water to make a three-ounce mixture, of which a dessert-spoonful was given for a dose to a child two or three years of age. Tannin is given in doses of one-sixth to three-fourths of a grain, and even to three grains, combined with hyoscyamic or benzoic acid every two hours.

Alkalies, as carbonate of potash or soda, sulphuret of potass, liquor potassæ and liquor ammoniæ, are oftentimes of value, when conjoined with other remedies, in rendering the bronchial secretion less tenacious, and in relieving disordered bowels.

Nux vomica and strychnine have been given with advantage in the stage of spasm. Certain antispasmodics, as musk, valerian, and ether, are of value, but they do not so immediately and certainly relieve the spasm as narcotics and sedatives administered so as to slightly affect the sensorium. They are more particularly suited to the stage of recovery from the spasm. [Some physicians, however, have found musk the most potent of all remedies for the violence of the cough in severe cases.—H.]

Emetics may be employed with advantage in those simple cases of Hooping-cough in which there is unusual difficulty in removing the secretion from the bronchi, whilst at the same time it is excessive in quantity and impedes respiration. But with the view which we entertain of the nature and treatment of this disease we do not think that emetics should be the chief remedies employed.

Such are the remedies which have been and may be properly employed in simple cases of Hooping-cough — hydrocyanic acid, belladonna, conium, morphia, arsenic, zinc, alum, nitrate of silver, hydrochloric and nitric acids,—all affirmed at different periods to be specifics, or, at the least, sure and speedy remedies for this disease. With the weight of testimony in their favor we cannot deny that they have been and are very valuable agents; and if we have given the preference to one of them, it is because by it we may the most readily and safely induce that gentle impression of the sensorium by which the spasm subsides. The mode of administration is as essential a part of the treatment as the drug itself, and the plan will succeed only in intelligent and careful hands.

Complicated Hooping-cough. — As the most frequent complication is that of bronchitis, care should be taken to watch the earliest indications of its approach. When it exists, the use of metallic, vegetable, and mineral tonics and astringents should be discontinued, and antimony or ipecacuanha in small doses should be added to the narcotic or sedative in use. If there be much febrile action, the patient should be placed in a warm-water bath up to the neck every night or every second night, and spt. eth. nit. with liq. ammon. acet. added to the medicine. With much oppression of the respiration and difficulty in removing the secretion, the occasional use of an emetic and the constant use of alkalies will be proper, and counter-irritation of the chest by blisters, turpentine, or mustard should be effected. If there are evidences of the extension of the disease to the parenchyma of the lung or to the pleura, the application of leeches and cupping at the root of the lung or over a painful part may be of service; but the

use of mustard or other rubefacients between the shoulders will usually afford the greatest relief. Even during this period it will also be needful to sustain the vital powers by good beef-tea, and as far as possible by milk, given in very small quantities at a time; and great care should be taken not to carry the antiphlogistic treatment so far as to lower the vital powers and induce disgust for food.

When convulsions occur, the first duty should be to ascertain if the teeth cause irritation, or if there be irritating matters accumulated within the stomach and bowels. If the former, the gums, when tender, should be lanced; and if the latter, the bowels should be freely evacuated and the character of the evacuations carefully watched. At the same time the patient should be supplied with beef-tea and milk. When hydrocephalus occurs, with or without convulsions, the treatment of the case must centre in that complication, and be such as will be elsewhere advised in this work. With heat of skin and head it will be proper to apply cooling lotions and even ice; and should the disease be active, leeches to the base of the head should be early used. The free exhibition of mercury in the form of calomel in small doses, or of hyd. c. cretâ, must be persisted in when effusion has been diagnosed. As the bowels are usually constipated in this conditlon, mercurials do not readily purge, and care should be taken to induce a sufficient action. Moreover, a state of inanition from want of food must not be permitted.

In the complication with remittent fever, care should be taken to evacuate the bowels and to correct the secretions, in addition to the use of those remedies referred to under that disease in another part of this work. The cough, which is also then unusually distressing, will require the frequent exhibition of morphia, syrup of poppies, and of tolu. As soon as possible the patient should have a change of air.

When extreme exhaustion is the chief complication, it will become necessary to administer nitric acid with bark, or some of the metallic tonics with quassia, and to supply wine-and-water in small doses every three hours. Much care should also be taken to induce the patient to take food, in small quantities, made from milk, eggs, jelly, and meat every two hours; and if neither the exhaustion be extreme nor the atmospheric temperature low, to obtain change of air and a moderate degree of exposure to it.

The recovery from Hooping-cough always demands the use of tonics [quinine in tonic doses, iron and cod-liver oil.—H.], of abundant animal food, and of change of air, and possibly also of wine administered in small doses. Sea-voyaging is then of great service.

DIPHTHERIA.

By William Squire, L.R.C.P. Lond.

Definition.—An acute specific disease, both epidemic and contagious, characterized by a special inflammation of the mucous membrane, chiefly of the pharynx and first air-passages, attended with enlargement of the lymphatic glands, a rapid exudation either of fibrine or non-organizable lymph, and its deposit within and upon the surfaces affected. Other parts of the mucous membrane and the skin sometimes suffer at the same time, and changes often take place simultaneously in the spleen or kidneys, albuminuria frequently occurring at an early period. The disease is accompanied by great prostration of the vital powers, and is followed by a remarkable series of local lesions of innervation; the tendency to death is by asthenia, either coincident with the disease or gradually induced, or by apnœa from implication of the air-passages, which may happen as early as the second day, or as late as the second week of the disease.

Synonyms.—Cynanche; Angina, Celsus; Synanche, Aëtius Aurelianus; Ulcera Egyptica vel Syriaca, Aretæus; Crustosa et Pestilentia Tonsillarum Ulcera, Aëtius Amidenus; Ulcera Pestifera in Tonsillus, Paulus Ægineta; Morbus Suffocans, Villa Real; Angina Exulcerata Maligna, Nuñes; Faucium Ulcera Anginosa, Mercatus; Angina Maligna, Heredia; Carbunculus Faucium Anginosus, Riolan; Morbus Strangulatorius, Aëtius Cletus; Morbus Gulæ; Aphthæ Malignæ; Angina Puerorum Epidemica, Bartholin;

Prunella Alba, R. James; The Sore-Throat attended with Ulcers, Fothergill; Malignant Ulcerous Sore-Throat, Huxham; Angina Infantum, Wilcke; Angina Polyposa sive Membranacea, Michaelis; Cynanche Pharyngea Epidemica, and Epidemic Croup, Rosen; Angina Suffocativa, or Sore-Throat Distemper, Bard; Mal de Gorge Gangréneux, Chomel; Angine Couënneuse Pharyngienne, and Croup in the Adult, Louis; Pellicular Angina, Diphtheritic Angina, Diphthérite, and Diphthérie, Bretonneau; Pharyngite Pseudo-membraneuse, Rilliet and Barthez; Cynanche Membranacea; Cynanche Maligna, Putrid Sore-Throat, Malignant Quinsy, England; Garotillo, Spain; Strypsiucka, Sweden; Rachen-Croup, Germany.

NAME.—Diphtheria is derived from διφθέρα, a skin, or covering of leather. The multiplicity of synonyms has been occasioned by the undetermined nature of the disease, and its appearance at times and places removed from each other, either by considerable intervals or by imperfect communication. Fothergill has the merit of setting forth, during its prevalence in the last century, the identity of the epidemic with that of the preceding century; while the Spanish and Italian physicians had already recognized its correspondence with the accounts derived from antiquity. Diphthérite, as signifying the special product of a specific disease, was proposed by Bretonneau. To him we owe not only the name but that energetic inquiry into the disease, as it showed itself in France during the present century, which has led to a more perfect definition of its character, and a better comprehension of its relation, than was previously possible. He at first applied the term to the whole disease, as well as to its characteristic morbid product, so soon visible in the fauces of those attacked; but further observation, proving that the local appearance was only one of the manifestations of the more important general affection, induced him, while retaining diphthérite in its more restricted sense, to speak of the general disease itself as diphthérie. We are indebted to Dr. W. Farr for the introduction of Diphtheria into our nomenclature as soon as the epidemic spread of the disease among us rendered it important that it should be designated by one general term.

HISTORY.—There is little doubt that Diphtheria, like the other acute specific diseases, has existed as long as the history of man extends. We have traces of it two thousand years ago, and the description given of it more than a thousand years since applies equally to its appearance in our own day. Its individuality is not difficult to recognize during its epidemic prevalence, at other times its distinctive characters have been merged with those of scarlet fever and erysipelas; these two diseases, though specifically different, approach at many points the nearest to it: they were not discriminated even in Sydenham's time; and though scarlet fever then began to be separated and to receive an increasing share of attention, the intimate connection always observed between it and Diphtheria precludes our astonishment at finding them sometimes confounded.

Unmistakable evidence of the existence of this special form of disease is found wherever medical science has attained any degree of exactness. Hippocrates describes it, and gives us the name of probably its first recorded victim.[1] It attracted the attention of Asclepiades and Celsus.[2] Aretæus[3] is the founder both of our knowledge of and treatment of the disease; and Aëtius of Amida showed an equally familiar acquaintance with it. It is impossible to say which of the many plagues of the dark ages of history may claim this disease as its agent. After the time of Paulus Ægineta only the writings of the Arabian physicians can be appealed to, and it is not until the intellectual revival of the sixteenth century that we again find its traces recorded;—as by Forrestus, in Holland, in 1557; Weir, in Basle, in 1567; by Baillou, in Paris, in 1576; and by Spanish writers from 1581 to the close of the century, whence is dated the first clear account of its epidemic prevalence in modern times. The numerous and graphic accounts of many and able Spanish medical writers of the seventeenth century afford valuable materials for the comparative study of the disease; and though the Sicilian and Italian writings of the same time are less original, we gather from them many important particulars. The epidemic attracted attention in the kingdom of Naples in the year 1618, and is described by Sgambatus, Carnevale, and Nola. The writings of Cortesius, in 1625, and of Alaymus, in 1632, speak of its ravages in Sicily; at the same time it had extended into Central Italy, as witnessed by Aëtius Cletus; Severinus and Bartoline speak of its continuance. The disease, if not epidemic, continued to be extremely prevalent in Spain; and, besides the special works of Fontecha, Villa Real, Herera, Tamayo, and Nuñes, was treated of systematically by the royal physicians, Mercatus and Heredia. Scattered notices of its occur-

[1] Hippoc. Epid. lib. v. tex. 37.
[2] Celsus, lib. iv. cap. 4.
[3] Aretæus, De Causis et Signis Acutorum Morborum, lib. i. cap. 9.

rence elsewhere appear in subsequent publications, but it is not until near the middle of the next century that we find it extensively epidemic, appearing simultaneously in Italy, France, and England, as evidenced by the independent observations of Ghizi, in Cremona, 1747; Arnault, in Orleans, 1748; and Starr,[1] in Cornwall, 1749: it also called forth the noble essay of Fothergill,[2] published in 1748; the epidemic had then hardly attained its height in England, though cases had been observed as early as 1739. It existed at Rouen at the same time, and appeared more extensively in 1748,[3] when the epidemic was at its height in France. Sweden suffered from it in 1755, and occasional outbreaks occurred there till 1778, as we learn from Michaelis. The presence of the disease in North America at this time is described by Dr. Samuel Bard, who having been trained in the University of Edinburgh to the study of medicine, returned to its practice in his native country, and furnished, by his careful observation of the cases under his care, the foundation upon which our knowledge of the disease was to be much further advanced.

That each of these epidemics preserved the essential characteristics of the disease as now observed, two quotations will suffice to show. Of the first, in Italy, Aëtius Cletus, in the introductory chapter to his work,[4] the only part of interest, says, "Morbi facies hæc est. In faucibus rubor apparere incipit, cum dolore, et febre; paulo post præbet se conspiciendam pustula, quam subsequitur cum crusta ulcus cinerei coloris, quod frequentius accidit, subalbicantis nonnunquam, vel nigrantis coloris. Sæpius sine pustula crustosum ulcus; . . . . et in ipsis faucibus sunt, quibus ex ulcere itur in gangrenam, et sphacelum, qui ex œsophago porrigitur ad ventriculum, vel ex aspera arteria ad pulmonem; et hi difficulter respirant, et non nisi recta cervice, illi deglutire nequeunt . . . . ex his nonnulli profundiori somno oppressi moriuntur; alii copiosa cum narium hæmorrhagia vita finiunt, alii absque his symptomatibus exanimantur. Morbus impuberes precipue invadit, tanta cûstrage, ut familiarum multarum omnes emori visi sint. Nulla moriendi est certa dies . . . die quarto decimo elapso non absque delicto quis emoritur—ex iis, qui pristinam valetudinem consequuntur anni, plus minusve spatio, omnes fere mussitant, et verba difficulter efferunt." Of the next, Dr. Starr[1] writes in 1749: "The morbus strangulatorius, with great propriety and justice thus denominated, has within a few years raged in several parts of Cornwall with great severity. Many parishes have felt its cruelty, and whole families of children, whence its contagious nature is but too evident, have, by its successive attacks, been swept off. Few, very few, have escaped." "Many in the first attacks have complained of swelling of the glands, as tonsils, parotids, submaxillary and sublingual glands, but frequently of no great importance. A few, from an internal tumor, have had a large external œdematous swelling of the subcutaneous and cellular tunic, from the chin down to the thyroid gland, and up the side of the face. Not a few early in the disorder have had gangrenous sloughs formed in their mouths, and perhaps so early in some, that the disorder was scarce complained of till the slough was formed, so quick has it been in its progress." And again, "I have not mentioned a fœtor oris, because, though some have had it, others have had it not." The symptoms of its extension to the larynx are then given, and he goes on to say: "I have frequently examined the matter these patients have at times spit; the greatest part was of a jelly-like nature, glairy, and somewhat transparent, mixed with a white opaque thready matter, sometimes more, sometimes less, resembling a rotten membranous body, or slough. Such a slough I have seen generated on the skin of one of these patients in the neck and arm, where blisters had been before applied. . . . . This white surface had the aspect of an oversoaked membrane, which, being oversoaked, had become absolutely rotten. The part blistered, if not quite, was in effect dry, and the flux from the slough was incredibly great. . . . . I scratched the slough with my nail; it separated with ease, and without being felt by the child. What my nail took off afforded the same appearance with the matter of the spittle before mentioned. Hence I thought I saw sufficient reason to convince me that the disorder in the larynx and aspera arteria was similar to this, generating in the same manner, and arising from the same external cause; . . and it is likely, had the anatomical knife been employed, that what was seen on the back of one, might have been discovered in the arteria aspera of the other. There is a circumstance which adds to the probability of this opinion, viz., in one or more instances these different disorders appeared in different subjects in the same family at the same time."

A century again elapsed before the disease attained to epidemic intensity;

[1] Phil. Transactions, vol. xlvi. p. 435.

[2] An Account of the Sore-Throat attended with Ulcers, by John Fothergill, M.D. 8vo. Lond. 1748. 2d Edition.

[3] Phil. Transactions, vol. xlix. pt. 1.

[4] De Morbo Strangulatorio Opus, Ætii Cleti, Signini. 8vo. Rome, 1636.

[1] Loc. cit.

during the interval, outbreaks of more or less extent and severity occurred in various parts of France, Germany, North America, and in England, Scotland, and Ireland; sometimes sufficiently serious to receive full comment in the medical periodicals, sometimes so isolated and rare as almost to escape notice.

Paris, at the beginning of the present century, was a frequent seat of its appearance, and the death from this cause of a promising member of the Imperial family of France in that city, in the year 1807, was the occasion of a large share of medical attention being directed to its elucidation. No real progress however was made towards that object until the epidemic at Tours, in 1818, received a thorough investigation under Bretonneau. From this time the *Archives Générales de Médecine* contain frequent reports of its appearance in various parts of France, and, by recording its period of greater or less prevalence in Paris, and the details observed in the Hôpital des Enfants Malades, afford valuable contributions to the study of the disease: most of these notices are to be found under the heads of Croup and Epidemic Croup.

The records of the disease in this country become less frequent after the close of the last century.

Dr. Mackenzie, of Glasgow, describes two cases that came under his notice in 1813, of throat-disease ending in Croup, and says that this kind of disease was very prevalent in Glasgow in 1819; he gives a careful description of it, agreeing very closely with the results arrived at by Bretonneau: these, made known in England by the publication of his work in 1826,[1] rendered the differentiation of the two diseases possible, and henceforth they are described apart. Dr. Abercrombie[2] alludes to cases of this kind in Dublin; he says, though not a common affection in Scotland, yet that it was very frequent and fatal among children in Edinburgh in the year 1826, and that "it is evidently an affection quite distinct from the idiopathic inflammation of the membrane of the larynx, to which we commonly apply the name of Croup." Dr. Webster, of Dulwich,[3] records the deaths of two children in one family, in a house near London, in 1824, and of four other cases of ulcerated sore-throat, involving the larynx, coming under his observation shortly after: they "all had," he says, "an assemblage of similar symptoms, and the common point of danger was the windpipe." Mr. Ryland[4] describes cases of the kind as epidemic in Birmingham, in the year 1837. Dr. Humphry, of Cambridge, observed a case in the Norwich Hospital in the same year, and Sir William Jenner[5] remarks, in his Lectures on Diphtheria, "I have seen cases of it every now and then as long as I have practised medicine."

Though isolated cases were from time to time observed, any memory of its epidemic violence in this country had almost passed away when its appearance in South Wales was announced by Mr. J. D. Brown,[6] of Haverfordwest; about two hundred cases occurred there in 1849–50, of which forty were fatal. Some general conditions, which at present we are unable sufficiently to appreciate, including the effects of the two cold winters of 1853 and 1854, and the intense cold of January, 1855, seem at this time to have checked its epidemic progress, and also to have stayed the ravages of cholera in this country. Traces of it meanwhile are to be found in other countries: Denmark,[7] Germany, France, the North of Africa, Madeira, Teneriffe, America, and Hindostan. Dr. Jackson[8] reports two cases occurring in Calcutta in 1853, one at a later period, and also thirteen cases at the Martinière school, five of which died. We have accounts of it at Lyons in 1851, at Avignon[9] in 1853, of its great increase in Paris[10] in 1852–53, and of its reigning simultaneously there and in many parts of France from 1855 to 1857, as a most fatal epidemic. In Boulogne alone it was the cause of 366[11] deaths in that period. It was present, though not extensively, in the armies in the Crimea[12] in 1855, and also at Moscow[13] in the same year, and in

[1] Des Inflammations Spéciales du Tissu Muqueux, et en particulier de la Diphthérite, ou Inflammation pelliculaire, connue sous le nom de Croup, d'Angine Maligne, d'Angine Gangréneuse, &c. 8vo. Paris, 1826.

[2] Abercrombie, John, M.D. Pathological and Practical Researches on Diseases of the Stomach, Intestinal Canal, Liver, and other Viscera of the Abdomen. 8vo. Edin. 1828. Pp. 53–56.

[3] The Institute, vol. i. p. 100. These cases were published in an Appendix to Mr. Higginbottom's Essay on the Use of Nitrate of Silver. 8vo. Lond. 1829. Pp. 185–196.

[4] Treatise on the Diseases and Injuries of the Larynx and Trachea, by Fredk. Ryland. 8vo. Lond. 1837.

[5] Jenner, Sir W., M.D., F.R.S. Diphtheria, its Symptoms and Treatment. Lond. 1861. P. 3.

[6] Med. Times and Gazette, 1850, vol. i. p. 670.

[7] Beck in Oppenheim's Zeitschrift, b. xliv. s. 200.

[8] British Medical Journal, 1859, p. 373.

[9] Archives Générales de Médecine, s. 5, t. viii. p. 338.

[10] Ibid. s. 5, t. v. p. 260.

[11] On Diphtheria, by E. H. Greenhow, M.D. Lond. 1860. P. 68.

[12] Haspel, Gazette Médicale, 1855, p. 829.

[13] Tarassenkoff, Diphtheritis Epidemica, Med. Zeit. Russlands, p. 92.

Algeria[1] the year following. It was epidemic in California[2] in 1856–7, and somewhat later in the Northern States of America.[3]

The epidemic attained its height in England in the year 1858 and 1859, and during these two years we may estimate that 20,000 deaths were occasioned by it. In 1860 the number of deaths had fallen from near 10,000 to 5202, and though they have not since reached the latter figure, yet the mortality from this cause in London was almost as great, and in the northwestern counties quite as great, in 1862 as in 1859.

In Scotland, where the disease prevailed at the same time as in England, the mortality from this cause did not reach its highest until 1861. It is somewhat remarkable that though Diphtheria existed both in India and California, we have no history of any outbreak of it in Australia until 1859, when Mr. James Moore[4] records nine deaths from this cause, and the occurrence of 275 cases at the same time in New Norfolk, Tasmania. This part of the world is perhaps more exclusively in communication with England than any other. The appearance of the disease there is not until after it had attained in this country to its full epidemic development: moreover, though epidemic in North America, both in California and the United States, as early as 1857, it was not till 1859 that it made its appearance in Nova Scotia, the part of that continent in closest communication with this country.

The severity of the outbreak at Boulogne, the constant communication from our own shores to that place, and the number of English visitors and residents there, many of whom fell victims to the epidemic, excited reasonable alarm, not only of the advance of the disease, but of its direct introduction into this country through the medium of those affected. Individual cases were imported, both at Folkestone[5] and Dover, without the disease spreading in those localities; its independent appearance in Wales, Lincolnshire, Cornwall, and Staffordshire precludes Boulogne from being considered its origin; yet sources of infection imported thence may have afforded new centres for the propagation of the disease, and have contributed in some degree to the determination of its type.

ETIOLOGY.—The records of the first half of the epidemic in England are imperfect; many deaths from this cause were included in the registration under the heads of scarlet fever, croup, and cynanche maligna. A table,[1] published by Dr. Farr, in his letter to the Registrar-General, shows that, while in 1855–57 there are 1846 deaths registered as cynanche maligna, and only 725 as Diphtheria, in the year 1858, upon the first introduction of the new general term,[2] the numbers are 1770 as cynanche maligna, and 4836 as Diphtheria; and that in the year following, when not only the name, but also the nature of the disease was more generally understood, 9587 deaths are registered as Diphtheria, and only 597 as cynanche maligna. In looking through the returns under croup and scarlet fever, the two diseases most likely to be mistaken for Diphtheria, it is not difficult to infer, both from the unprecedented numbers returned under these heads, as well as from the usual number of deaths of the two sexes from these causes being reversed, that some other disease has been included, and this variation occurs in the very districts in which we have other evidence of Diphtheria being present: thus in Wales, while the deaths from croup in 1853 were 207 males and 197 females, total 404; for the year 1854 they were 215 males and 298 females, total 513; they then continue at or near 500, till Diphtheria is introduced into the registration nomenclature, when they suddenly fall to 424. The same exceptional proportion is first noticed in the Cornwall district in 1854; in the three following years the whole number is doubled, and in 1859 undergoes the same sudden diminution. The west-midland, north-midland, and eastern districts, from which at this time we have the most frequent accounts of Diphtheria, show the same gradual increase, and the same sudden fall; and it cannot be doubted that of the 6220 deaths returned as croup in the year 1858, at least 1000 were Diphtheria. In scarlet fever this is still more conspicuous; the number of deaths mounts up from 14,229 in 1857 to 30,317 in 1858; and in the latter year, for the only time that I am aware of, the number of deaths of females from that cause exceeds the number of deaths of males. An epidemic of scarlet fever certainly accompanied that of Diphtheria, and culminated in the following year, when the deaths from it, Diphtheria being excluded, reached 19,907; so that we may reasonably suppose that of the 30,317 deaths registered under this head in 1858, near upon 10,000 were really owing to Diphtheria. The probability is that some deaths from this cause in previous years were so regis-

[1] Notes sur la Diphthérite. Recueil de Mémoires de Médecine Militaires, s. 2, t. xvii. p. 392.

[2] Fourgeaud, Diphtheria. 8vo. Sacramento, 1858.

[3] Boston Medical Journal, vol. lix. p. 252.

[4] Australian Medical Journal, July, 1859.

[5] E. H. Greenhow, M.D., op. cit. p. 69.

[1] Twenty-fifth Annual Report, p. 178.

[2] This was in the first Quarterly Report of the Registrar-General, No. 37.

tered: on turning to South Wales in 1857, we find that of 267 deaths from croup, 177 were of females; and of 349 from scarlet fever, 185 were of females; so that in 600 deaths, chiefly of children, the majority of which ought to be among males, we find an excess of 40 among females.

In the second half of the epidemic, taking the four years 1859-62, the number of deaths registered from Diphtheria is 24,219; of these 11,229 are of males, 12,990 of females. Half these occur in the first five years of childhood, and show a nearly uniform fatality in each of these years, the first year only being somewhat below 10 per cent. of the whole number; half the remaining number, or 25 per cent. of the whole, take place between the ages of five and ten years, and about 10 per cent. during the next five years; in the next ten years, that is, from the ages of fifteen to twenty-five, the proportion of deaths does not exceed 6 per cent. of the whole; from twenty-five to thirty-five it hardly reaches 2 per cent. and is below 1 per cent. in decreasing ratio for each succeeding decade. The proportional mortality from Diphtheria for each age, to the deaths from all causes, is:—

	1st Year.	2d Year.	3d Year.	4th Year.	5th Year.	5—10 Years.	10—15 Years.	15—25 Years.	25—35 Years.
Both Sexes	·5	2·	3·7	6·	8·	9·	6·3	2·5	·32
Females	·5	2·1	4·	6·4	8·8	10·4	7·3	1·8	·45
Males	·5	1·9	3·4	5·6	7·3	7·7	5·3	1·2	·2

These results are opposed to some statements with respect to the liability of sex, but fully bear out Fothergill's remarks on this subject. He says:[1] "Children and young people are more exposed to it than adults; a greater number of girls have it than boys; more women than men; and the infirm of either sex are more likely to have the disease and suffer from it than the healthy and vigorous. If it break out in a family, all the children are commonly affected by it, if the healthy are not kept apart from the sick; and such adults as are frequently with them, and receive their breath near at hand, often undergo the same disease." He also remarks that "this disease has now been with us several years, and has consequently survived the different seasons and all the variety of weather to which we are subject."

Climate and season do not influence the nature of the disease; it presents the same features in Sweden as in Spain; nor is any difference observable in its appearance in our own humid climate from that which it presented to Aretæus, when he attributed its causation to the peculiarly dry air of Egypt. Hot and dry climates or seasons, the germ being present, seem to favor its development. Yet difference of season has no marked effect on the course of the disease, nor on its prevalence[2] over limited areas; nor has a sudden change of weather any effect in originating or arresting its epidemic appearance.[3]

The London Quarterly Reports illustrate, not the influence of season upon the disease, but the variableness of its prevalence in the metropolis. On the commencement of the registration of Diphtheria in the second quarter of 1859, the number of deaths from this cause was 173, in the third quarter 190, and in the winter quarter 141. In the next year the third quarter shows the lowest, and the winter quarter the highest mortality. For 1861 the numbers are: 1st quarter 139; 2d, 159; 3d, 168; 4th, 231[1]—a progressive increase rarely met with even in other epidemic diseases. The extreme degree of cold reached in the winter of 1860 had no influence in checking the second advance of the disease in London: that it was not the occasion of the increase is shown by the comparatively low number for the first quarter of 1861, and the very high return for the last quarter of that year, when the season was usually mild.

The influence of contagion is established; not the exact mode in which it operates. The matter of infection would seem not to be capable of any wide diffusion in the air, but to cling to particular places, houses, and even chambers; hence it not only becomes endemic, but might also be distinguished as hypodemic, as well as epidemic. When epidemic, no kind of soil or situation influences its occurrence; of places in close proximity some escape, while others suffer, and th n frequently some direct communication is to be traced with the districts previously affected: isolated cases are, however, frequently met with without any widely-spread consequences. The earlier part of any local epidemic is generally attended with the largest proportion of fatal results: during the period of greatest epi-

[1] Op. cit. p. 30.

[2] Boudet, Arch. Gén. de Médecine, 1842, s. 3, t. xiii. pp. 144, 446.

[3] Gazette Médicale de Paris, 1848.

[1] Some deaths from this cause are included under Croup. *Vide* Vol. III.

demic prevalence the disease is found to be subsiding in one district while advancing in another; the height of the epidemic coincides with the greatest number of local outbreaks. No general atmospheric condition can then be its cause, and whatever the unknown assemblage of circumstances that favor the progress of the disease, they would not occasion it without the presence of its own special reproductive gem; so that the idea of the epidemic constitution must always include that of contagion.

The theory of direct contagion, as set forth by Bretonneau, requires considerable modification; the possibility even of inoculation is rendered doubtful by the experiments of Trousseau upon himself, and of Dr. Harley[1] upon animals; nor is there reason to suppose that greater power of propagation attaches itself to the fibrinous exudation, or special product of the disease, than to some of the secretions and exhalations from the sick. I know of no instance of the disease being carried from one house to another by the passage to and fro of those who were themselves unaffected: the presence of one sick person in a house is sufficient for its communication to the susceptible, however carefully kept apart, so that the infectious matter must, to some extent, be diffusible in the air, and the danger of infection has seemed to me to be greater from this source than from direct contagion. Most of our brethren can speak of having come into disagreeable contact with the secretion from the throat of sufferers, no bad effects following: very many can refer to dangerous illness in themselves as the result of too long or too close attendance in the sick-room. It is probably to this influence rather than to direct inoculation that is to be attributed the death of Valleix, and of Dr. Adams of Boston, U. S.[2] The case of M. Herpin, adduced by Bretonneau, admits of the same explanation; it is not clear that a very limited denudation of the skin, or of the mucous membrane, renders the system more exposed to the contagion; but the disease once contracted, those parts of the mucous membrane exposed to the air are specially liable to be affected by the disease; or if there be any abrasion of the skin, it is likely to suffer.[3] The existence of excoriation, as at the edge of the nostril, of unhealthy gums, of enlarged tonsils, possibly of catarrh, may determine the local manifestation of the disease without these lesions having been the channel of its admission.

It has been noticed that the more abundant the secretion in particular cases of Diphtheria, the greater has been the consequent extension of the disease, especially where the greatest care has not been given to cleanliness; even where this has received every attention infection will sometimes cling with surprising tenacity to particular houses or apartments. A single case, in itself not severe, occurring in a house, is sufficient to originate the severer form of the disease in the susceptible. It is not necessary that there should be much secretion from the mouth and nares to originate infection; a man named Sheppard, under Dr. Reynolds's care in University College Hospital, 1865, for paralysis subsequent to Diphtheria, contracted the disease from his child whom he held constantly in his arms during a short illness, the nature of which was not evident until after the child's death from asphyxia. A very short time suffices for the development of the disease after exposure to infection; the boy in the Hospital for Sick Children whose case is reported by Sir W. Jenner,[1] was observed to have advanced symptoms of Diphtheria within thirty hours of the commencement of the affection in a child occupying the next bed, and it is probable that a time considerably shorter is sufficient.

Evidence of infection is not always shown immediately after exposure; of two children removed from an infected house to a village where no case of Diphtheria had occurred, the elder showed the first symptoms of the disease on the third day, having been very cheerful and well the whole of the two intervening days. A young lady going from this village to nurse her sister in the infected house, became ill four days after her arrival. Eight days is the longest interval that has been known to occur before the disease has shown itself where prompt removal from the source of infection has been carried out.[2] Mr. W. Adams, of Harrington Square, has kindly furnished me with the clearest particulars of the case in point, mentioned at page 46 of Sir W. Jenner's book. A much longer interval may occur where the cause of infection is acting more continuously. Dr. Hughes,[3] of Denbigh, records a case where a servant was taken ill twenty-two days after the occurrence of Diphtheria in the house

[1] Transactions of Path. Soc. of London, vol. x. p. 315.

[2] Med. Times and Gazette, 1861, vol. i. p. 375.

[3] Mr. Lister, while House Surgeon at University College, received a scratch on the finger while performing tracheotomy in a case of diphtheria. Signs of the disease subsequently appeared in the injured part.

[1] Loc. cit. p. 83.

[2] Exactly this interval has been observed under the same circumstances in scarlet fever, as the longest that has come under my notice.

[3] British Medical Journal, 1859, p. 80.

where she was residing. In a vicarage in the country a servant was ill with Diphtheria, and remained one month in the house till convalescence enabled her to be removed; an interval of ten days was then allowed for cleansing and ventilating the house, when the family, consisting of mother, a weaned infant, and two other children, the eldest under four years of age, who had left the house on the first appearance of the disease, returned home; three weeks afterwards the infant had symptoms of Diphtheria, and before the death of this child, which took place on the tenth day, the mother who had been constantly in attendance was taken ill, and subsequently the two other children.

Infection may be disseminated for an uncertain time by those convalescent from Diphtheria; a girl ten years of age was removed from home at the outbreak of Diphtheria, from which three children, two younger and one older than herself, afterwards died; two other sisters convalescent from the disease went to the seaside, the one five weeks from the commencement of her illness, the other only three weeks, but from whom the last trace of deposit had cleared away; they left together and seemed to be equally well: they were joined by other members of the family, and at the end of the week by the little girl, æt. 10; early in the following week symptoms of the disease appeared in her, and became rapidly fatal. She was the only one of the family that had been isolated during the whole period of the illness. Sir W. Jenner[1] gives two similar instances: in the first, only a fortnight elapsed from the commencement of the disease to the removal into the country, and within a fortnight from the date of arrival, another member of the family, some time resident there, was attacked; in the second a little boy, after three weeks' separation from his family, joined his sisters, convalescent from Diphtheria, at a country residence to which on their recovery they had been removed. "Ten days after his arrival there, the boy sickened with Diphtheria. In this case, either the poison was in the child's system when he left London, and remained latent for a month, a supposition highly improbable, or he caught the disease from his sister, after they met in the country."[2]

Constitutional predisposition has a great influence in increasing the liability of families and individuals to receive the disease: of two families residing in the same house several members of the one have suffered, while all of the other have escaped; a difference of susceptibility is also observed in members of the same family, and this not always in favor of the seemingly more robust. Allowing for the effects of similarity of diet, occupation, general management and hygiene, and chiefly for the degree of exposure to the same sources of infection, it is yet impossible to contest the effect of family constitution, both in favoring the occurrence of the disease and in disposing to its fatal termination.

Great mental activity, and a high degree of nervous susceptibility, would seem to increase the liability to become affected; bodily fatigue, and exhaustion from any causes, predispose. There appears to be but little difference in the liability of the different classes of society.

The presence of other diseases, as of typhoid fever, pneumonia, measles, erysipelas, and scarlet fever, has a great effect in exposing the system to the attacks of this one. The special poison of this disease sometimes produces effects short of general Diphtheria, which, however, predispose to the occurrence of the fully-developed disease, either from the first local evidence of it being unchecked, or upon some accidental source of weakness arising, as from the effects of aperient medicine, or of menstruation. The disease is especially likely to be established, if there be repeated or continual exposure to the exciting cause.

The recurrence of Diphtheria more than once in the same subject is not settled so conclusively in the affirmative as has been supposed. That the same person may repeatedly suffer from the slighter forms of the malady, and that some do so upon the slightest exposure, is frequently observed; but when the fully-formed disease has been undergone, though relapses are to be feared in convalescence, even during the whole of the subsequent period of debility, which may be prolonged for two or three months, independent recurrence is rare. Of the children that recovered from the disease at the commencement of the epidemic, and who have been constantly under my observation since, no instance of a second visitation has occurred. Such instances are recorded, one by Dr. Gull, eleven months after the first attack, and three others by Dr. Greenhow,[1] one of which was fatal, but no particulars are given in this case as to the intensity of the first attack. The development of Diphtheria agrees more closely with that of erysipelas than of scarlet fever; the closeness with which many of its pathological effects coincide with those of the latter disease makes it necessary to remark here, that the occurrence of scarlet fever offers no protection against the attacks of Diphtheria.[2] So with respect to croup: two

[1] Loc. cit. p. 52.

[2] Loc. cit. p. 53.

[1] Loc. cit. p. 111.

[2] Dr. Ballard, in Medical Times and Gazette, 1859, vol. ii. p. 78.

instances have occurred to me, wherein children that had suffered severe attacks of croup, fell victims to Diphtheria after intervals of four and six years. Hygienic conditions influence the frequency, and even the fatality of Diphtheria, less directly than they do the permanence and diffusion of the infectious principle; want of cleanliness allows the infectious particles to accumulate; the presence of decomposing organic matters shields them from destructive oxidation in the atmosphere; thus they linger in districts naturally malarious, and in those artificially so from an improper drain system, as well as in the close, dark, and dirty districts of large towns, and evils thus matured rest not in their cradles, but come forth and often exert their most fatal effects in homes the most unlike those where negligence had allowed them to lurk. [The influence of unsanitary local conditions in promoting the occurrence and mortality of Diphtheria has been marked of late years in many places in America; amongst others, in certain quarters of the city of New York. Tenement houses, containing an excessively crowded population, have often been subjected to great loss of life from this as well as other "enthetic" diseases. Foul privies, badly drained cellars and streets, and leaky connections of dwelling houses with sewers, favor Diphtheria very positively, notwithstanding the specific nature of its immediate causation.—H.]

Symptoms.—Some general constitutional symptoms precede those occasioned by the concomitant local changes; both may co-exist in every degree of severity, sometimes the one, sometimes the other attaining a deceptive prominence; at ether times both are so obscure as to make the inroad of the disease remarkably insidious. Among the earlier symptoms are yawning or sighing, shallow and infrequent respiration, great lassitude and debility, some aching of the back and legs, either a distinct rigor, or chilliness, pallor, a sense of nausea or rising in the throat, anorexia, sometimes vomiting, or diarrhœa, and in children convulsions, a sense of constriction across the forehead, or intense headache, vertigo, extreme muscular weakness, some anomalous fixed pain, an altered mental state, slowness of recollection, an indifference of manner, and an obtuseness of the mental faculties; this latter gives place to a short period of excitement, during which it may be thought that no illness is impending. There is, however, a marked elevation of temperature, which is increased at night, when wakefulness or restlessness always occurs. The pulse is accelerated, and in children or young persons may rise to 130 or even to 140 in the minute: this frequency soon subsides, generally before the end of the second day; and though the pulse continues to be quick, it is either feeble or easily compressible. The respiration is never proportionately accelerated at this period. The tongue is moist, with a thin creamy fur; the urine is pale in color, at first free, soon rather less in quantity, with scanty deposit of lithates, but still pale, and at this time it may be albuminous. The lymphatic glands at the angle of the jaw are already preceptible; there is always some enlargement of the cervical glands, and redness with a little swelling of the posterior part of the soft palate, of the fauces, of the back of the pharynx, and of the tonsils. The throat is sore, deglutition is difficult if not painful, sometimes the pain is felt in the ear, and there is frequently stiffness of the neck; very little external swelling is at this time noticeable, and there is no diffused redness of the skin; sometimes little isolated red spots are found in different parts of the surface, as over the neck or behind the ears; there may be suffusion of the eyes and slight injection of the conjunctival vessels, and a little obstruction to the nasal passage from a similar vascular state of its lining membrane. Sometimes this is seen at the nares to be simply red, sometimes an opaque white spot may be noticed beneath the epithelium, or one nostril may be completely obstructed, and some fibrinous exudation already observable; sometimes the back of the pharynx will show the first indication of this in lines of opaque tenacious secretion, or commencing deposit is seen there, in the apices of its enlarged follicles. The earliest evidence of the disease is, however, most frequently found within the follicles of the tonsils or deposited on its inflamed and turgid surface.

The first general symptoms are transient, and may be so trifling in degree as to escape notice, until they are intensified by the progress of the local lesion; this will give rise to pain, heat, and soreness of the throat, with impeded function: it also excites some sympathetic febrile disturbances of its own, and always increases that proper to the general disease; where both are severe, the throat, though covered with exudation, is often the least part of the patient's complaint; where both are slight, there may be an interval in which little complaint is made. During this interval, two days, there may be no visible exudation in the fauces; the tonsils continue to be enlarged and their surfaces irregular, and they, in common with the whole of the pharynx, the arches of the palate, the velum, and the uvula, are of a deep-red color and unequally turgid; one side is generally the most affected, the uvula is enlarged, red, and glistening, and a mottled redness extends forwards from

it over the soft palate, but the rest of the buccal membrane is pale. Some of these parts soon appear more tumid and glistening than others, and spots at first semi-transparent and afterwards opaque, rapidly form and coalesce, so that in a few hours a large surface may be covered with a continuous layer of exudation; or the exudation is limited to one or more centres, the mucous membrane around being elevated and of a violet tinge of redness, until it is invaded by the same change. The raised edge is then extended, the centre being occupied by a flattened, yellowish-white, leather-like deposit, increasing in thickness by additions to its under-surface; this is accompanied by the pouring out of a considerable amount of a more fluid secretion, while that from the surrounding mucous follicles is also increased and altered in quality, so that a tenacious fibrinous matter is mingled with the mucus. The cervical glands increase in volume, the submaxillary lymphatic glands especially; a large amount of serous infiltration takes place in the surrounding cellular tissue, and the whole of the front of the neck becomes greatly swollen; deglutition is involuntarily suspended, secretions escape in some quantity from the mouth, and even from the nostrils, by which the lips are excoriated. The superficial layers of the localized deposit, partially separated and exposed to the passage and re-passage of the air and the warmth and moisture of the mouth, undergo decomposition and give rise to great fetor; extravasated blood mingles with the decomposing exudation, or escapes freely from its under-surface, or occasions epistaxis. The voice is muffled or nasal in tone; impeded respiration may occur from the physical obstacles in the fauces without laryngeal symptoms arising; or these being slightly marked, symptoms of pulmonary obstruction come on insidiously. These processes may be gone through with great rapidity, and are accompanied by the most marked adynamia; the pain in the head is intense, the restlessness and agitation extreme, vomiting or diarrhœa may occur, the lips and tongue become dry and brown, the pulse very rapid, feeble, and irregular; the skin becomes cold, the face pallid, the whole attitude is indicative of powerless exhaustion; there is impaired consciousness, slight delirium, or deep somnolency, and some repeated attack of syncope terminates life.

The elevation of temperature is always marked, and characteristic; it may be as high as 103° very early in the disease; it will generally reach this point by the end of the third day, and is highest at night, when it may amount to 104°, then probably with delirium. This temperature has not been exceeded in uncomplicated cases. The days of highest temperature observed from the commencement of the illness have been from the third to the ninth or tenth. Free exudation is attended with temporary depression of temperature. Defervescence in the severer cases recovered from has been on the twelfth and fourteenth days; in the milder cases this has not occurred before the sixth day, though some precedent subsidence of temperature may have been noticed. In fatal cases an earlier decline of temperature has sometimes been met with. A girl, aged 8, had a surface temperature of 103° on the third day, 101° on the fourth, then barely 100°, with swelling of glands, fœtorosis, and hæmorrhagic tendency, death on the eighth day; the day before the temperature in the axilla was below the normal. In cases markedly asthenic the temperature of the surface falls, while that of the interior of the body continues to be high: in a boy, aged 2, with a temperature of 103° in the rectum on the fifth, sixth, and seventh days of the disease, only 100° could be obtained in the axilla, and sometimes only 99½°; the temperature after subsiding to 100° was suddenly raised to 102° by acute kidney complication; continued there two days, and was again below 100° the day before death. Another boy, aged 4, with nasal diphtheria and slight tracheal implication, had a temperature of 103° in the rectum on the sixth day, suddenly falling to 99·5° on the seventh, then continuing at or near 100° until death on the fifteenth day. In the apparent interval between the first illness and the distress occasioned by the throat symptoms, the high temperature is an indication of value: a child has been taken out for a walk on the fifth day with a temperature *in recto* of 103·7°. A young man was found dressed and walking about on the ninth day of illness, with a surface temperature of 102°; on being kept in bed the temperature fell 2° in two days; the exudation then ceased to extend, but the tonsil last affected had become turgid and more inflamed coincidently with the fall of temperature: albuminuria appearing here during defervescence was considered to be rather hæmic than renal.

During convalescence the temperature is low, and readily depressed: but any intercurrent affection may keep up the temperature indefinitely.

The advancing asthenia is always accompanied by a considerable increase of exudation; it continues while the process of exudation is going on, and is at its greatest when the separation of the diseased products is being accomplished; this may be completed by the ninth or tenth day of the disease. The exhaustion at this period, if not fatal, is often so great as to suspend for some days the hope of recovery, to delay the process of repair,

and to prolong the diseased action, so that there may be a reappearance of exudation in the fauces, or it may at this time invade the larynx. If there be any injury of the skin, the diphtheritic action, probably already established there, will be continued so as to become an additional source of exhaustion. Besides the hemorrhagic tendency at any affected surface, spots of purpura sometimes make their appearance either widely distributed or grouped together: sometimes an earthy pallor pervades the whole surface of the skin. Where there are none of these unfavorable complications it is not rare to find that, when the exudation is clearing off from the fauces, a remarkable increase of albumen occurs in the urine. Nocturnal delirium may occur, not only at the commencement, but at the height of the disease, when the asthenia is considerable. A more violent symptomatic disturbance at the commencement of the disease, occasioned either by local or by constitutional reaction, may mask the tendency to asthenia without making it less; and should the course of the disease not be interrupted by the implication of the air-tubes, asthenia becomes the prevailing condition throughout all the subsequent illness, and may bring on a fatal result, not only at the end of the second week, but during any of the subsequent weeks of convalescence, even to the fifth or sixth from the commencement of the disease.

Extension of the disease to the larynx and trachea may be the cause of death at a much earlier period; the air-passages are specially liable to become the seat of the peculiar exudation of Diphtheria; it may commence there either by independent centres of deposit, or by the spread of exudation from the pharynx to the epiglottis, over the arytæno-epiglottidean fold, and thence downwards, even to the remotest bronchi, and be fatal, with symptoms of sudden or of slow suffocation, in the earliest stages of the disease, or at the period of greatest exudation, towards the end of the first or commencement of the second week. This extension often proceeds insidiously when the general symptoms are of great intensity, and is only indicated by hoarseness, or weakness of the voice, and by some laryngeal quality in the breath sounds, faintly audible during both inspiration and expiration, by signs of impeded respiration. evidenced in the œdema of the face and livid hue of the lips, sometimes only by pallor, and gradually deepening unconsciousness, and by the retraction of the softer parts of the thoracic parietes. In other cases the first invasion of the mucous membrane of the upper part of the air-tube is announced by highly characteristic phenomena, similar to those produced by acute laryngitis in adults, or by idiopathic croup in children, differing only in the less sharp and sonorous clang of the cough, in the more husky tone of the voice, and in the antecedent and concomitant symptoms. Besides the appearance in the fauces, and the dysphagia, the sudden change from the asthenic character of the prevalent symptoms to the excitement, arterial and general, now occasioned is very striking. In other cases, chiefly among children, the disease may have set in with no great severity, the power of swallowing and even the desire for food may be returning, and the child, no longer confined to bed, is resuming its amusements, when a hoarseness of voice and a noise in breathing are the only precursors of paroxysmal dyspnœa, differing only from that of croup in the time of the day in which the first attack may occur, and in the readiness with which the child will return to play on its subsidence, but soon becoming more terrible in the unintermitting violence which it rapidly assumes, or in the suddenness of the fatal result.

The mortality from this complication alone is very great; it has been estimated that one-half of the fatal cases of Diphtheria die from this accident; nor is this estimate excessive when children are the sufferers; an extension to the air-tubes, necessarily fatal to children, will sometimes be survived by adults; in the man Sheppard a cough of unequalled violence with expectoration of false membrane continued into the third week of illness. In a small proportion of cases the larynx or trachea has been the first seat of the disease; in these cases and in those where the faucial deposit is small or already disappearing, the urine is more frequently found to contain albumen at an early period than in those cases attended with abundant exudation on the pharynx. In a female, aged 35, named Bowra, under Dr. Hare's care in University College Hospital for Diphtheria, in April 1865, who died of pneumonia, coincident with slight deposit in the larynx and fauces, there was a considerable amount of albumen in the urine on her admission, the second day of her illness; the next day the temperature of the surface rose to 104°, pulse 144, respiration 36; the albumen increased to one-sixth, and the respiration to 57; death took place on the sixth day. The first evidence of deposit was in the larynx, and there was no extension of it into the bronchi. Soreness in the front of the neck, and pain or sense of tightness over the sternum, are frequently complained of in these cases, and sometimes slight œdema is noticeable in front of the trachea.

Albumen is found in the urine in the great majority of cases; its presence either in large or small quantity does not

necessarily affect the excretion of urea: this is always increased during the whole period of illness, and when the disease is at its height is frequently double. In one case (J. B., a young man, aged 18), on the seventh day of illness, 606 grains of urea were excreted, or more than treble the normal quantity: the urine has an acid reaction, a high specific gravity, and deposits a furfuraceous sediment in which uric acid, urates, oxalates, and sometimes phosphates, are detected by the microscope, and not infrequently casts of the renal tubules, either waxy or granular; blood-corpuscles are rarely found, and never in large quantity. Albuminuria generally occurs early in the illness; it may be absent and reappear more than once in its course. In a man named Walker, aged 42, admitted to University College Hospital, under Sir W. Jenner's care, Nov. 14th, 1864, for Diphtheria, at the end of the first week of illness, there was no albumen found till the end of the third week; it then continued till death, which took place in the fifth week of the disease. In the case of J. B., albumen was present throughout the illness; on the twelfth day it had increased to one-third, on the twenty-fourth day it was one-eighth, and then gradually lessened during the ingress of paralytic symptoms, and finally disappeared at the end of the fifth week, while the nervous disorder was at its greatest, and three weeks before convalescence was established. Albuminuria is not persistent after recovery, and does not often result in anasarca.

Hemorrhage is not infrequent, and may be so profuse as to cause sudden exhaustion, either on the separation of some morbid deposit of unusual depth, or from some dyscrasia of the blood favoring its ready transudation. Vomiting and diarrhœa, generally absent after the first ingress of the disease, may set in with gastralgia and great depression. Exhaustion may come on gradually without these symptoms, the pulse increases in rapidity and feebleness, and death takes place without distress of breathing or impairment of the mental faculties. In some of these cases, and of those yet to be described, fibrinous coagula form before death in the cavities of the heart.

The liability of other mucous membranes and of the skin, especially when denuded, to become the seat of the changes characteristic of Diphtheria, has often a great influence on the progress of the disease; eczema behind the ears, or in any fold of integument, abrasions or fissures at the juncture of skin and mucous membrane, leech-bites, blistered surfaces, and even those irritated by a mere rubefacient application, may become affected, and add greatly to the amount of disease against which the patient has to contend; a cutaneous eruption, occasioned by the disease itself, is sometimes, though rarely, the seat of these ulterior changes; the cuticle is raised, a white surface is exposed—this is identified with the upper layer of the cutis and does not implicate its whole depth—the skin immediately around is red and tumid; new vesicles, sometimes sanious, are raised upon it, they burst and coalesce; the white layer extends its surface, which is still somewhat below the level of the surrounding skin; it increases in thickness by additions from below, and the upper layers soften, decay, and disintegrate; it is accompanied by an irritant fluid secretion, both increase proportionally, and the extension of false membrane takes place most readily in the direction where the secretion is most in contact with the skin; where these changes take place in some portions of the skin, towards the end of the disease, there is but little secretion formed, and no great extension occurs. At the height of the disease the extension is sometimes very rapid; but this is less remarkable, because less constant, than the tendency then observed to repetition. Not only will every abraded or irritated surface take on this action, but isolated patches of deposit may occur on the edge of the lip, on the eyelid, in the meatus auditorius, and elsewhere. Open wounds undergo a similar change, the superficial granulations are converted into this pseudo-membranous layer, cicatrization stops, the wound becomes painful, and a copious ichorous secretion is poured out; a fissured nipple has been the starting-point of the morbid process, and much of the surface of the breast has suffered; the disease readily establishes itself in the vagina, where layer upon layer of false membrane may accumulate. An abraded surface of integument is sometimes covered with a granular or pulpy exudation only, attended with but little fluid secretion; the affected surfaces then continue to be irritable, but do not always extend. Diphtheria has generally first appeared in the fauces, and made some progress, before other parts of the mucous membrane, or of the exposed skin, have been attacked; instances are not, however, wanting of the disease commencing elsewhere, and inducing in the pharynx, larynx, and trachea, the characteristic changes that soon become fatal.

A remarkable train of symptoms often make their appearance after the disease has reached its height, and become a new source of danger. These are the limited and varying series of paralyses, which are some of the characteristic effects of the special diphtheritic poison. An altered tone of voice and the regurgitation of fluids through the nose are frequent evidences of this, and have long attracted

notice; an inability to swallow, first pointed out by Ghizi, was noticed by M. Guersant to occur about the ninth day of the disease, and to become an embarrassing obstacle to recovery in cases where tracheotomy had been performed. This is frequently observed only with respect to fluids; there is at this time, as shown by M. Trousseau, a loss of sensibility in the velum pendulum palati, and probably in the pharynx and glottis. Complete inability to swallow from loss of power in the muscles of deglutition seldom occurs before the third or fourth week of the illness; at this time on inspection of the throat, the uvula is often found to be lax, and neither it nor the pillars of the fauces act upon stimulation; difficulty in the ingestion of sufficient nutriment adds to the dangers of this period; vomiting, moreover, is possible when the power of swallowing is gone. Loss of power and of sensibility in the parts supplied by the par vagum occur at a somewhat earlier period than the paralytic affections of other parts of the body, and hence arise special sources of danger; the slow weak pulse observed in the second and third weeks of illness is from this cause, and is frequently a fatal symptom. The pulse may be reduced in children to sixty or even forty beats in the minute, and the child seeming otherwise well, death by syncope has occurred suddenly, on some undue exertion; or the heart's pulsations have gradually failed to thirty or, shortly before death, even to sixteen beats in the minute.[1] I have seen a similar failure of respiration occur to a boy, aged 9, in the second week of illness, where no extension of the disease to the larynx had taken place; inspiratory efforts had to be artificially excited at frequent intervals during many hours; every few minutes the respiratory movements became more and more shallow and inefficient, until they were again stimulated into action, and even then the ribs were not always elevated; at the end of twenty-four hours respiration was more satisfactorily performed; during the second day the voice regained its clearness and force, the intellectual activity seemed to be more than usually quickened, the respiratory murmur was perfect in every part of the lung, both food and stimulant were swallowed, but the action of the heart became at first slow and weak, afterwards very feeble and rapid, and death took place on the third day from the commencement of these symptoms. Paralysis of the muscles of respiration occurring at a later period, in an adult, threatened to be fatal but for the stimulus of galvanism. Dr. Gull reports the case of a boy, aged 11, who, five weeks from the commencement of Diphtheria, was unable to prevent the head falling forward, or to either side, owing to paralysis of the muscles of the neck; he suffered from dysphagia, aphonia, and paroxysmal dyspnœa; a few days afterwards "the breathing became entirely thoracic. The diaphragm was unmoved in inspiration and depressed in expiration, indicating a loss of power in the phrenic nerves."[1] Death approached rapidly by apnœa.

Loss of power and irregular action of the pharyngeal muscles is not only the earliest, but the most frequent form of disordered innervation. It sometimes rapidly disappears, and is not followed by other symptoms of this kind; in other cases the power of swallowing may continue to be impaired for three or four weeks; at first the difficulty is with liquids, afterwards it is now and then found that solids cannot be passed down, and are in danger of remaining in the lower part of the pharynx, or of being forced up towards the posterior nares, while liquids find their way into the stomach, and these conditions may alternate. At this time articulation is sometimes defective from imperfect movement of the tongue; tingling sensations in the tongue and lips are also felt, and may continue during the fifth and sixth weeks. Paralysis of the nerves of the special senses was first observed as a consequence of Diphtheria by J. F. Hoffman.[2] Taste is sometimes lost, more rarely hearing; defective vision is not infrequent; it begins with an immovable and sluggish pupil, and an inability to read or to distinguish near objects; soon more distant objects become indistinct, double vision or strabismus may occur; sometimes one eye only is affected; these changes of vision take place from the fourth to the seventh week, and when sight is perfect the pupils again act freely. As these alterations of sense improve, numbness and tingling commence in the fingers and toes, extending gradually to the hands and arms, and to the feet and back of the legs, or even to the hips. Some degree of these sensations, with formication or a sense of coldness in the extremities, various degrees of anæsthesia, or even some hyperæsthesia, may occur earlier, and before any great loss of muscular power. These conditions vary very much, disappearing at some parts, increasing and extending at others. They may pass off for a time, and reappear with the increasing muscular debility; the sense of touch may continue to be impaired; anæsthesia is seldom complete except in the severest cases. The lower

[1] Sir W. Jenner, op. cit. p. 44. Mr. Adams has given valuable details of a case of this kind in the Second Report of the Medical Officer of the Privy Council, p. 327.

[1] Lancet, 1858, vol. ii. p. 5.

[2] Rust's Magazin, 1831, b. xxxiii. s. 241.

extremities suffer most; it may be impossible not only to stand but to move the legs in bed. When this degree of paralysis is not reached, the gait is often unsteady, and walking may be impossible from a loss of the muscular sense; this sometimes interferes with the use of the hands, and the power of co-ordinating muscular movements. Loss of power is not to the same degree or so persistent in the upper extremity as in the lower, though here also remarkable alternations are observed, one limb or one set of muscles being free to act on one day, and powerless on another; when power is regained, one muscle, or part of a muscle, may remain for a time the subject of inaction or of spasm. Reflex action is often diminished, but not increased. Paralysis of the bladder has occurred, not of the rectum. Constipation, from paralysis of the abdominal muscles, is a frequent condition, demanding attention. The first symptoms of paralysis will almost always appear before the end of the fourth week; they generally attain their greatest degree of intensity by the seventh or eighth week; they may not do so until the tenth or twelfth, as in Sheppard's case; all traces of the affection may not have entirely disappeared after five or six months. Lesions of innervation are not in proportion to the extent and persistence of the local lesions, nor always the consequences of the more severe and prolonged attacks of Diphtheria only, though it is after these that they are most likely to occur; they may conduce not only indirectly, but directly, to a fatal result, from progressive loss of nervous power, apart from muscular weakness; in these cases the period most dangerous to life is reached in the seventh or eighth week. Trousseau[1] has detailed a case where delirium and convulsions at this time appeared, with ultimate recovery. More frequently death by asthenia, unattended by symptoms of other disease, occurs within this period. A peculiar pallor and opacity of the skin is often a concomitant of even the more slightly marked cases of paralysis consequent on Diphtheria. In the more prolonged cases there is always some evidence of anæmia.

Diagnosis.—A careful inspection of the fauces will, in the majority of cases, be conclusive as to the presence of the disease: where a yellowish patch of exudation moulded to the surface it has invaded is thus brought into view, the nature of the serious illness, which may have been obscure, is at once revealed: where this anatomical character of Diphtheria is not obvious, the appearances in the fauces, taken in conjunction with the associated phenomena, will still be of primary diagnostic value. It is important that the earliest stages of Diphtheria should be distinguished from catarrhal affections. The redness of the fauces in Diphtheria is more intense but less uniformly diffused than in catarrhal inflammation; the tonsils are more tumid, and one side is more affected than the other; the lymphatic glands at the angle of the jaw and beneath the sterno-mastoid are always enlarged: in children coryza may be present, the vascularity of the conjunctiva is then more like that observed in measles, but there is less secretion; afterwards, the defluxion from the nares becomes more considerable, it is not simply mucous, but sero-purulent or sanious; or the nares not being so much affected, a quantity of unequally opaque and tenacious mucus bubbles in the gullet, and prevents the view of the posterior part of the pharynx; there will be pain in deglutition, as shown by the infrequency of the effort, or the grimace that accompanies it; in either case the exudation soon extends to the larynx, and the cough, hitherto infrequent and moist, becomes frequent, dry, and croupy, and the disease is set down as catarrhal croup. For the further diagnosis of croup from Diphtheria, see the article on Croup.

Tonsillitis resembles Diphtheria in the two sides of the throat being unequally affected, and in the occurrence of external swelling at the angle of the jaw; the constitutional symptoms are, however, symptomatic, dependent on, and in proportion to, the local complaint; the lymphatic glands are not enlarged at the commencement of the attack, nor those beneath the sterno-mastoid at any time. A yellowish soft secretion appears at the orifices of the tonsil; the mucous surface preserves a smooth, glistening appearance, and any exudation of lymph upon it is semi-transparent, very thin, and limited in extent; the tendency of the inflammation is to resolution or suppuration, the other tonsil often becomes affected in the same way, but without any similar change taking place in the intermediate mucous membrane.

In Herpetic sore throat the highest temperature is on the second day, when it may rise to 102°, and then rapidly subside.

In scarlet fever, the throat affection is always preceded by symptoms of severe febrile disturbance, which are persistent, and in proportion, not to the throat affection, but to the severity of the attack of fever which follows; the chilliness and headache may not be so marked, but the heat of skin is greater, and the pulse at once attains a high degree of frequency which it maintains during the first days of the illness, and until after the appearance of the rash, or the condition of the

[1] Gazette des Hôpitaux, 1860.

throat, has removed all doubt as to the nature of the disease. The premonitory symptoms in Diphtheria are sometimes not noticed in the severer cases, and when well marked do not always indicate the approach of the graver symptoms; should these follow, the frequency of the pulse during the first few days is not maintained, the respiration is shallow and not proportionally accelerated, and there is neither the continued high temperature nor the same pungent heat of skin. The diminished frequency and fulness of respiration at the outset of Diphtheria is often an indication of value when the disease is not yet fully developed. The cervical lymphatic glands are enlarged in both diseases. The redness of the throat in scarlet fever is uniformly diffused; on the second or third day it becomes very intense, appearing simultaneously upon all parts of the throat and palate, and affecting the papillæ of the tongue; both tonsils are equally enlarged. In Diphtheria the redness and turgescence are greatest in certain parts about to become the seat of exudation, and at the edges of the exudation already formed, so that it and the surrounding redness gradually advance upon the contiguous portions of the mucous membrane; the papillæ of the tongue are neither red nor enlarged, and the tonsils are unequally affected. In scarlet fever exudation, both tonsils are covered with a milk-white layer applied equally to the surfaces of both, and the soft palate and tongue may be covered with a similar layer; this undergoes no great increase in substance, and at a certain period is detached in shreds; it is not capable of absorption, and on its separation leaves a red and sensitive surface. In Diphtheria the process of exudation continues to be active for some time; on its cessation, there is a separation of membranous layers of considerable density and extent, which may represent a cast of the surface on which they formed; a re-absorption is now possible of some of the products still imbedded in the mucous tissue; this is accomplished either without loss of substance, or with superficial ulceration only, and the sensibility of the surface is diminished. In scarlet fever there may be sloughing of the tonsil, and there is a tendency to suppuration of the cervical glands. There is no tendency in the inflammation of the throat in scarlet fever to be propagated to the air passages; the nasal tone of voice, and the regurgitation of fluid through the nose, cease on the subsidence of the swelling of the tonsils and lymphatic glands. In Diphtheria these symptoms often undergo a remarkable increase subsequently, from the paralytic sequels of the disease. Scarlet fever has a definite course, modified only in degree of severity; Diphtheria may either be arrested or modified in its course, so that the period of its duration is less definite. In scarlet fever there is a greater liability to inflammations of the serous membranes than in Diphtheria. Albuminuria, as a sequel to scarlet fever, interferes with the excreting power of the kidneys, is attended with hæmaturia, and more frequently results in dropsy and anasarca: it is an early symptom in Diphtheria, is rarely attended with hæmaturia, seldom interferes with the excretion of urea, and does not result in dropsy.

Both diseases are contagious, but while the contagion of Diphtheria has not been shown to give rise to scarlet fever, that of scarlet fever has apparently been followed by Diphtheria. They are not prophylactic of each other: in the late epidemic of Diphtheria, children who had gone through scarlet fever were equally liable to suffer;[1] and though in some cases a severe attack of Diphtheria has seemed to give an immunity from scarlet fever, yet instances are not wanting of those who have recovered from Diphtheria being attacked with scarlet fever. Dr. Buchanan,[2] of Glasgow, records the appearance of scarlet fever in a boy six years old, four days after the performance of tracheotomy, and in the second week of his illness from Diphtheria; anasarca appeared in the third week; in the sixth week convalescence was complete. A patient suffering from paralytic symptoms, consequent upon Diphtheria, while under the care of Dr. Stewart, in the Middlesex Hospital, contracted scarlet fever, during the febrile stage of which, and the full appearance of the rash, the paralytic symptoms cleared away. This modification of a symptom peculiar to the one disease upon the establishment of a condition peculiar to the other, though an instance of the distinct nature of the two, does not show any necessary antagonism between them; not only may the one succeed to the other at very short intervals, but it would seem to be possible for them to coexist. The appearance in the throat characteristic of Diphtheria may commence after the subsidence of the redness occasioned by scarlet fever, and the disappearance of the rash; they may, however, come on at any period of its course, and more rarely the two diseases may seem to be coincident, so that the aspect of the throat on the first day may be indicative of Diphtheria, and with the characteristic rash of scarlatina appearing on the second or third day there may be a fall in the frequency of the pulse. In some epidemics of scarlet fever undoubted cases of the disease occur without the character-

[1] Dr. Ballard, loc. cit. p. 78.

[2] British Medical Journal, September 1864, p. 324.

istic rash. A redness of the skin, and more or less marked rash or eruption, readily distinguishable from that of scarlet fever, has sometimes accompanied the early stages of Diphtheria. Mercatus[1] mentions a rash like flea-bites in some of the Spanish epidemics, and a redness of the whole face and neck, with loss of voice and dyspnœa. Fothergill describes a rash, in the first edition of his treatise, which he says, in a foot-note[2] in the second edition, did not regularly accompany the disease, and which is certainly not that of scarlet fever; it agrees more nearly with that lately observed, and described by Dr. Babington, as rubeola notha, which was sometimes, though rarely, seen in cases of Diphtheria, appearing on the first day. Dr. Fuller[3] communicated to the Harveian Medical Society, February 1858, the case of a child ill with sore throat and a rash, like scarlet fever, from the commencement; on the third day there were great dyspnœa and excitement with an increase of the rash and of the redness of the face; by night a membranous cast of the pharynx was expelled, with immediate relief to the dyspnœa and a rapid subsidence of the redness and rash, so that by the next day no trace of either remained; recovery was rapid, there were none of the sequelæ of scarlet fever, and no desquamation. Desquamation of the cuticle from the hands and feet has occurred after prolonged illness from Diphtheria, when there has been no preceding rash.

Erysipelas comes on with rigors, or chills, and headache; there is pain in deglutition, often extreme, and some enlargement of the cervical glands; the throat redness, though intense, is diffused; there is no secretion, and none of the fibrinous exudation characteristic of Diphtheria: this form of sore throat may occur either before or after the appearance of the erysipelas on the face or head. Erysipelas, smallpox, and measles are liable to be followed or complicated by Diphtheria.

Diphtheria may follow upon typhoid fever or any prolonged and exhausting disease; it is important, therefore, to distinguish the sordes that collect in the fauces, and the special product of thrush, or muguet, to which such cases are liable, from the exudation of Diphtheria. The matter of thrush is closely attached to the mucous membrane when it is first exuded, but it becomes more and more easily separable; it occurs in little rounded masses, is whitish, and soon projects beyond the level of the surface: if artificially removed, the membrane beneath looks slightly hollowed, and either red or gray, but it is neither completely abraded nor ulcerated; the buccal membrane and not the throat is specially the seat of this formation; it acquires no great extent, nor considerable tenacity. Chemically, it has an acid reaction, is not acted upon either by acetic acid or by alkalies, and is only dissolved or destroyed by sulphuric acid; these qualities are owing to the large parasitic vegetable growth of *Oïdium albicans*, which also give to it special microscopical characters. Sordes occur in patches of unequal thickness, very little coherence, and no great extent, and are removable without injury to the subjacent tissue, however red and tender it may be; they chiefly accumulate in front of the arch of the palate, acquire an acid reaction, and then also become the seat of the *Oïdium albicans*. It is only when the diphtheritic exudation approaches to these local and chemical conditions that it becomes the seat of similar parasitic growths.

Pathology.—The general disease and the local lesions that arise during its continuance have an interdependence and mutual reaction. The latter are not confined to one period of the disease only; they occur throughout its course, sometimes proving fatal by the vital importance of their site, at others adding to its force, and prolonging its continuance. The general disease impresses a special character, not only upon the local lesions which it occasions, but upon any concomitant morbid action: it is marked throughout by an elevation of the normal temperature of the body, by enlargement of the lymphatic glands and the spleen, by a varying amount of congestive action of the liver and kidneys, and of various parts of the mucous surfaces, as well as that of the fauces and first air-passages, where the speciality of the diseased action is most marked, and where it sometimes expends its whole violence.

The local lesion peculiar to Diphtheria is most readily induced in surfaces exposed to the free access of air, and though not restricted to them, it is there that it assumes its most characteristic development; there is a state of blood in the capillaries, a destruction of the red corpuscles, and a formation of fibrin, as shown by the spontaneous coagulation of part of the exudation. These changes take place in close contact with the bloodvessels, and commence in the mucous membrane beneath the epithelium, transforming the cells of the sub-epithelial layer or of the epithelium itself, or altogether replacing

[1] Consultationes, p. 136.

[2] Op. cit. 2d edition, pp. 32, 33. It is not until the publication of the fifth edition, twenty years after this, that an error in the alteration of this foot-note, and an added paragraph to the preface, led to the confusion that has since existed between Fothergill's sore throat and scarlet fever.

[3] British Medical Journal, 1858, p. 173.

them by the fibrinous exudation or false membrane. The false membrane cannot be detached without leaving a bleeding surface, which is rapidly covered with a new layer: it neither assists the cicatrization of the surface on which it forms, nor ever becomes itself organized. Superficial ulceration results from the interstitial necrosis of some parts of the tissue invaded by it, but there is no gangrene or mortification of its substance. Changes of decomposition rapidly take place in the deposit itself, with injurious consequences, both to the lymphatic glands near, and to the system at large, from the absorption of effete matters; the site of these changes may occasion other ill effects, by contaminating the air of respiration on its passage to the lungs. The disappearance of the false membrane is effected partly by this superficial destruction, and partly by absorption from its under surface and edges, and by return to healthy action in the vessels below. It is seldom entirely separated in this way; a thin layer is often left, through which pink points gradually appear; soon only isolated spots of exudation remain, which are finally removed by absorption, gradually effected as the process of reparation proceeds. This is sometimes much retarded by the constitutional debility induced by the disease; and is sometimes interfered with by a retrograde process of ulceration.

The extension of false membrane proceeds, primarily, from the wide dissemination of original centres of its deposit; secondarily, from the invasion of contiguous surfaces; the morbid action may thus extend gradually in every direction, or advance with great rapidity on the parts irritated by the accompanying serous exudation. This may be one cause of implication of the air tubes,[1] and of that progress of the disease from above downwards which Louis was induced to consider as almost the law of its extension. An illustration of the opposite mode of extension is sometimes seen when Diphtheria extends from the nose to the lachrymal duct, and thence to the conjunctiva; or in its progress from the lower edge of the palatine arch to the uvula; the turgescence preceding the exudation causes the uvula to be recurved upon the side already affected; soon the false membrane has not only invested the whole uvula, but extends upwards behind the velum. Wherever false membrane is formed, some degree of inflammatory action is excited; this may either approach very nearly, both in its products and symptoms, to the type of ordinary inflammation, or be entirely subordinated to the influence of the general disease, in which case this peculiarity is remarked—that the less marked the inflammatory condition, the more extensive is the pseudo-membranous formation.

The evidence of the general disease next in importance is the existence of albumen in the urine. Dr. Copland, in his Dictionary, first mentions "albuminous urine" in his account of the pathology of croup, which is framed to include the croupal complications of this disease. We are indebted to Dr. Wade, of Birmingham, for demonstrating the dependence of this system upon Diphtheria. Bouchut and Empis[1] soon after called attention to its importance, and Dr. Sanderson[2] considerably advanced our knowledge of its relation to the general course of the disease, by showing that the presence of a considerable amount of albumen did not interfere with the large excretion of urea, which accompanies the progress of the general disease. I possess notes of three cases confirmatory of his observation; in that of J. B. before referred to, thirty-one determinations of the quantities of urea and albumen were made from the sixth to the thirty-seventh day of the disease, by Mr. W. Dunnett Spanton, now of the North Staffordshire Infirmary, at that time (1861) residing with me. On the tenth day of the disease, when the albumen was estimated at one-third, the quantity of urea was twice as much as is normally excreted, the specific gravity being 1016; it was not until the thirty-seventh day that the urea fell to its normal quantity, and albumen was then for the first time absent; the specific gravity had fallen from 1015 to 1010. Subsequent observations on the forty-seventh, fifty-fourth, and sixty-second days, agreed very closely with the last result. The albuminuria is not to be considered as solely dependent on an original change in the blood, but chiefly upon a morbid process in the kidney, which is one of the disseminated lesions of structure occasioned by the general disease. Congestion of the Malpighian tufts is an early lesion, followed by further change in the tubercular structure of the kidney; a relation is found between these changes and the amount of albumen, but no constant relation between the albumen and the amount of blood change. In the case of Walker, where albumen did not occur till late in the disease, the blood change was extreme, as evinced by hemorrhagic oozing from the palate, petechiæ, and purpura; there was no hæmaturia at

[1] Not only may noxious matters thus reach the trachea, but detached shreds of secretion from it may be drawn into the smaller bronchi, and mechanically block up, by a kind of embolism, some lobules of the lung, as pointed out by Sir W. Jenner.

[1] L'Union Médicale, No. 132, 1858.

[2] Brit. and For. Med.-Chirurg. Review, January 1860, p. 196.

any period, and recent disease of the kidney was found *post mortem*. That the function of the kidney may be seriously interfered with, and even suppressed, is shown by a case mentioned by Dr. Gull,[1] and by one reported at the Pathological Society, February, 1865, by Dr. Greenhow.[2] Dr. Humphry gives one instance of the occurrence of anasarca,[3] but these cases are rare: albuminuria does not persist after convalescence. Blood changes may be concerned in these symptoms; that these changes are considerable during the progress of the disease is shown by the hemorrhagic tendency, by the occurrence of fibrinous coagula in the heart and great vessels after death, by the remarkable pallor during the illness, and the anæmia of convalescence, while the frequent affection of the spleen would point to a cause of this aglobulosis of the blood. Whether the lesions of innervation are owing to this cause, to a failure of nutrition in the nerve textures, or to a more special effect of the diphtheritic poison, is doubtful; from the instability of the disordered innervation, and the variety of conditions which it assumes, we may conclude with M. Trousseau, "that the lesion of the nervous centres is not of a very grave character." The impairment of vision is generally, as remarked by Mr. Dixon,[4] due to loss of adjusting power, and there is inaction of the ciliary muscles; he has not found any important change in the retina. Some of the more serious and persistent muscular paralyses are owing to wasting or degeneration of the muscular tissue itself. There are no good reasons for supposing that either special deterioration of the blood, or alteration of its quality, precedes the development of Diphtheria; a state of hyperinosis, if it were possible, is not that induced by many of the diseases to which Diphtheria readily succeeds; the alterations in the properties of the blood, physical, vital, or chemical, are rather the consequences than the cause of the disease; and it would seem that the first influence of the disease, as well as its later effects, are exerted upon the nervous system.

Morbid Anatomy.—The special product of Diphtheria has affinities with some deposits formed in other diseases, with the products of some forms of inflammation, and with the buffy coat of the blood, which it often closely resembles in appearance and some of its physical properties. It has an alkaline reaction, swells, and becomes transparent in strong acetic acid, and is disintegrated or dissolved by caustic alkalies. It is unaltered by maceration in water, and yields no gelatine to it, as tested by tannin; in this respect it differs from the buffy coat, and from coagulable lymph, and also in giving no evidence of albumen on being boiled; it is stained brown by tincture of iodine, and assumes entirely the character of pure fibrin. It is possible to obtain solution of some specimens in the same way that Denis[1] dissolved recent fibrin, and to find them unmixed with other protein matters; other specimens less fibrillated, or less recent, are not acted upon in this way. The tubes of exudation found in the bronchi will often lose their coherence on maceration in water only, and afford evidence of albumen on boiling, which will not always be the case with the denser membrane from the trachea.

Microscopically the superficial part of the exudation is made up of the epithelium of the membrane on which it occurs, entangled in the upper layers of a transparent homogeneous substance, throughout which are found some altered epithelium cells, granular corpuscles, and nucleolar bodies, in varying proportion; they become less numerous in the lower layer of the exudation, in which blood-corpuscles and pus cells frequently occur. Fibrillation, similar to that of other fibrinous exudations, is sometimes observed on the under-surface of the diphtheritic false membrane, from the fauces. The false membrane from the trachea is corpuscular throughout, as is also the pulpy and granular deposit which is sometimes found in the fauces, and is more frequent in the larynx; these deposits rapidly pass into granular degeneration. In the bronchial exudation the corpuscular element differs little from that observed in the product of ordinary inflammation there; sometimes little flakes of fibrin are found attached to the bronchial membrane, but the only other evidence of its presence is the coherence of the bronchial casts. The laminated, or fibrillar, the pultaceous and the granular, or corpuscular, are the leading forms of this exudation; they may present themselves in any combination under the same morbid influence; in the trachea they are always associated, and either may predominate; the fibrillated may be deposited in the mucous and submucous tissue of the larynx,[2] and the granular may be seen in

[1] Second Report of the Medical Officer of the Privy Council, p. 304.

[2] Transactions of the Pathological Society of London, vol. xvi. p. 47.

[3] British Medical Journal, July, 1863, p. 4.

[4] Holmes, System of Surgery, vol. ii. Diseases of the Eye, p. 766.

[1] Arch. Gén. de Méd. s. 3, tom. i. p. 171. Half a drachm of nitre to an ounce of water will effect this at a mean temperature. The solution is imperfect, viscid, coagulates in flakes at 163° Fahr., and is strongly precipitated by acetic acid.

[2] Dr. Bristowe, Trans. Path. Soc. Lond. vol. x. p. 323.

the fauces together with the laminated, the less coherent deposit, misnominated croupous, being as truly diphtheritic as the more tough, leather-like formation. The one point common to both is their intimate relation to the structure of the membrane on which they are formed. In the case[1] of Bowra I saw deposit in the contiguous edges of the true and false vocal cords, the sacculus laryngis being free, and the commencing granular deposit in the trachea firmly imbedded in the mucous membrane. Dr. Wilson Fox has demonstrated, in all stages of tracheal exudations, lesions of the membrane sometimes exposing the fibrous tissue beneath, both in adults and in children. M. Hache observes[1] the resistance to the separation of the false membrane from the trachea at a certain stage, and calls attention[2] to the sanguineous points found imbedded in its under-surface when removed.

The condition of the tissue in which the morbid action is exerted, though of less importance than the general course of the disease, affords better means of characterizing it than the exudation to which it gives rise: the unsatisfactory nature of a distinction founded upon the two forms of exudation in this disease is thus admitted by the most advanced pathologists of Germany:[3]—"The difference which I formerly established between the croupous and diphtheritic forms, is often lost in particular cases, so that the true croup of the larynx and trachea invades the tissue of the mucous membrane, ay, very often coexists with Diphtherite at the back of the pharynx and of the fauces; and not till the deeper bronchi are reached, or the pulmonary cells, does it become a free exudation. This is found not only in the epidemic croup of children, but also in Diphtherite occurring in typhoid conditions, and in hospital gangrene; or simultaneously with diphtheritic necrosis of the vaginal and intestinal mucous surfaces." This interstitial necrosis, or ulceration of the surfaces in relation with the exudation, is the anatomical character on which the distinction between the diseases here mentioned and Croup, in our acceptation of the term, must be based.

The diphtheritic deposit may occur in isolated patches, or extend continuously from the nares to the bronchi; it may be moulded on to any anfractuosity of the nasal passages; it may line the whole pharyngeal cavity, adhere to both surfaces of the epiglottis, cover the interior of the larynx, form a cylindrical cast of the trachea, and may thus be removable from even the smaller bronchi. More rarely it is found to extend in this manner throughout the œsophagus; in one of the two cases reported by Bretonneau,[1] it was strongly adherent to the upper part of the tube. In two of seventy-four cases tabulated in the British Medical Journal,[2] the œsophagus was partially affected, one with, one without extension to the air-passages. In a case reported by Dr. Morley Harrison, of Manchester,[3] the false membrane extended forwards to the mouth, and a patch existed upon the frænum linguæ. Dr. Bristowe gives an instance of the upper part of the œsophagus being covered. Virchow alludes to one case; he has seen the occurrence of false membrane in the gall-bladder.[4]

The tonsils are frequently the seat of this formation; it is often detached early, but may accumulate to a surprising thickness, the outer surface retaining for a time the exact appearance of the enlarged tonsils, and marked with the opening of its follicles. I have met with one specimen, which it was difficult to believe was not the organ itself under examination; every part of it was soluble in Denis' solution, and no trace of organized tissue occurred throughout its substance; this exudation has attained to the thickness of two-thirds of an inch.[5] The decomposition of such concrete deposits, their varied color from admixture of altered blood, and their detachment in offensive shreds, has often given a false idea of the gangrene of the tissue beneath, which really remains almost intact. Bretonneau called attention to the importance of this fact, but while he correctly described the "ecchymoses of no great extent, as well as the slight erosion of the surfaces on which the disease had existed longest,"[6] insisted too much upon the integrity of the mucous membrane being always preserved; but though much may be repaired before death, it is rare not to find some evidence of lesion of the mucous membrane. Sometimes the submucous layer is bare and granular, without defined ulceration;[7] at other times a defined ulcer exists, exposing the muscular fibres.[8] M. Louis noticed erosion of cartilage at the posterior nares. The uvula and part of the soft palate have

[1] Barthez and Rilliet, tome i. p. 318.
[2] Ibid. p. 319.
[3] Virchow, Path. und Therapie, vol. i. p. 292.

[1] These cases, and two others occurring in children with implications of the larynx, are mentioned by Barthez and Rilliet, tome i. p. 322.
[2] Brit. Med. Journ. 1859, pp. 305–6.
[3] Ibid. 1863, vol. i. p. 306. A complete cast of the tube was ejected.
[4] Op. cit. vol. i. p. 292.
[5] Memoirs on Diphtheria. New Sydenham Society, 1859, p. 98.
[6] Traité de la Diphthérite, p. 33.
[7] Dr. J. R. Hughes, Brit. Med. Journal, 1859, p. 80.
[8] Barthez et Rilliet, tome i. pp. 259 and 287.

been lost by ulceration.[1] Dr. Sanderson[2] mentions the division of the left half of the soft palate by a penetrating ulcer. Mr. Simon[3] describes a circular ulcer of the pharynx and other signs of ulceration. Dr. Ashley[4] observed phagedæna commence at one tonsil and extend to the carotid. Ulcerations on either surface of the epiglottis, and on the mucous membrane of the larynx and trachea, are frequently met with. The submucous tissue may be infiltrated with blood, with serum, or with inflammatory products. Dr. Newman[5] reports the formation of an abscess in the palate soon after the disappearance of the superficial exudation. In the case described by Sir T. Watson,[6] pus was found in one tonsil, and this has since been not infrequently observed. Mr. Pound,[7] of Odiham, reports a case of abscess in the pharynx, and Greenhow[8] gives one of post-pharyngeal abscess. Suppuration rarely, if ever, occurs in the cervical glands; they are large and red, or paler, and brittle, and sometimes present a spleniform disorganization; the swelling around them is from infiltration of serum in the cellular tissue. Dr. Bristowe[9] reports a remarkable effusion of blood among all the tissues of the neck. Various forms of petechiæ, purpura and superficial eschars are found upon the skin, and spots of purpura, petechiæ, and ecchymotic staining are frequent in other parts of the body, as in the muscular tissue and under the serous membranes, as on the lung, upon or within the heart and the peritoneum. I have met with no instance of inflammation of the serous membrane. Fatty degeneration of the muscular tissue of the heart occurred in the case reported by Dr. Bristowe.

Fibrinous coagula are very frequently met with in the cavities of the heart, extending into the great vessels. This occurrence is not limited to cases where there has been obstruction of the air-passages, though they are at least as frequent in the right cavities as in the left. Dr. Barry,[10] of Tunbridge Wells, reports three cases, all in the right side of the heart; the coagulum existed in the right auricle only in one case, and in another extended into the pulmonary artery. Dr. Rollo[1] gives the case of a soldier where, with the characteristic exudation of Diphtheria, fibrous polypi were found in the right ventricle. The lungs are frequently congested, and the seat of lobular hepatization in various stages, often sufficiently extensive to be the cause of death, even where no exudation has occurred in the air-passages. Pulmonary apoplexy is sometimes found. Where the air-passages are the seat of the exudation, lobar and lobular pneumonia, the latter often secondary to embolism of the smaller bronchi, with collapse of lung tissue, and acute vesicular emphysema, have generally been induced. The vessels of the brain or of the pia mater have presented fine injection in some cases; in others there has been fulness of the sinuses, and even transudation of blood; and Dr. Gull reports[2] suppurative inflammation of the membranes of the brain and cord, with soft, purulent lymph in the sub-arachnoid space. Dr. Humphry[3] has met with a small spot of suppuration on the under-surface of the left cerebral hemisphere, with softening of the adjacent brain substance, attributed to Diphtheria. The liver is frequently found full, sometimes greatly congested, but is seldom the seat of disease. The stomach often presents serious changes, its mucous membrane being softened, unequally thickened, and red from small patches of deep-colored congestion, or from extravasated blood; sometimes these changes are found in connection with isolated patches of opaque deposit at its cardiac end, and the œsophagus presents abrasions of its mucous membrane when it has been the seat of deposit. Vascular injection and minute ecchymoses have been found in the small intestines, the follicular orifices are often conspicuous, more rarely Peyer's patches in the lower part of the ileum are very distinct and prominent; sometimes deposit and ulceration occur in the large intestines, hardly distinguishable from that of dysentery. The spleen is generally found full and soft, more frequently paler in color than redder, and often presenting a cloudy or opaque appearance on section.

The kidneys may often appear healthy to the naked eye, but are very rarely found so under microscopic investigation; they generally present well-marked, and sometimes extreme evidence of special change: this is chiefly in the intra-tubular structure, and though congestion of the Malpighian tufts is often very conspi-

[1] Greenhow, op. cit. p. 201.
[2] Loc. cit. p. 191.
[3] Trans. Path. Soc. Lond. vol. x. p. 317.
[4] Brit. Med. Journ. 1859, p. 490, case 60.
[5] Ibid. 1863, vol. i. p. 215.
[6] Lectures on the Practice of Physic, vol. i. p. 865. 4th edit.
[7] Brit. Med. Journal, 1858, p. 750. Report of Reading Path. Soc.
[8] Op. cit. p. 237.
[9] Trans. Path. Soc. of London, vol. x. p. 328.
[10] British Medical Journal, 1858, p. 623.

[1] Essay on Cynanche Trachealis, by John Cheyne, M.D. 4to. Edin. 1801. P. 68.
[2] Loc. cit. p. 299.
[3] Brit. Med. Journal, July, 1863, p. 4.

cuous, the general aspect, sometimes mottled from an irregular blending of anæmia and congestion, is more often of a pale than of a deep color; they have not been found in the extreme state of disorganization sometimes seen after scarlet fever. The tubules appear opaque, from the epithelial cells being numerous, easily detached, and filled with an unusual amount of finely-granular material, in which oily globules are sometimes abundant. Some tubes are devoid of epithelium, and present transparent, fibrinous casts, or these are found separated from the tubes to which they correspond in diameter; occasionally blood-corpuscles are found in the tubules; more frequently they are filled with masses of epithelial cells and fibrinous exudation. These changes are as noticeable in the medullary as in the cortical structure; granular exudation is also found between the Malpighian capillaries and their capsule. The supra-renal capsules have been found intensely vascular.

Prognosis.—No case of Diphtheria is to be regarded without anxiety; every danger incident to the disease may result, though the early symptoms are but slightly marked. The successive appearance of fresh patches of deposit, of albuminuria, or of other signs of the disease, excites alarm, lest its next local manifestation should be in the larynx: the simultaneous occurrence of many of these signs, and the increase of any of them, as of the enlargement of the lymphatic glands, and of the amount of exudation, indicate an intensity of the general disease which the young or enfeebled will hardly withstand, and which, with implication of the larynx, will be rapidly fatal. The least laryngeal quality of the voice or cough, and especially of the respiration, is a sign of the greatest danger; it may, in the strongest person, soon end in fatal obstruction of the glottis from the occurrence of exudation there, or it may be the first indication of the equally fatal and more insidious extension of it to the bronchi. Much exudation in the nasal passage is unfavorable. If both nares are occluded, respiration must be carried on by the mouth with great discomfort; this may be a cause of death in young children, as they are thus unable to suck. The occurrence of hemorrhages is a bad and dangerous symptom; so is repeated vomiting and purging at the commencement of the attack. These symptoms occurring somewhat later, bring on death by exhaustion, and indicate either serious alteration in the blood, or that the stomach has become the seat of the disease. A very rapid pulse, except at the commencement, is indicative of danger; so also if the pulse fall below the normal frequency. Albuminuria, if unattended by any rise in temperature, is in itself not serious as long as the urine is in normal quantity, without blood corpuscles or casts of tubes, and while the specific gravity continues to be high. Any increase of the temperature of the body after the first five days, or a persistence of high temperature after the first ten days, is unfavorable; a sudden rise may indicate danger from some intercurrent disease. There is also the danger of relapses from the slightest debilitating causes, either in the earliest or at the latest periods of the illness. The liability to laryngeal obstruction is greatest toward's the end of the first week; it begins with the commencement of the disease, and probably continues throughout; this complication has happened as late as the fourteenth day,[1] and possibly later.[2] These dangers are especially treacherous, as they may occur in cases where there has been but little exudation on the larynx, and as a period of comparative convalescence may intervene between either the first symptoms and the laryngeal complication, or between those of the more advanced disease and its asthenic accidents. The first impression of the general disease may be so profound as to make recovery impossible; on the other hand, cases commencing with marked constitutional reaction, and with symptoms generally unfavorable, such as vomiting, epistaxis, or nocturnal delirium, may recover without experiencing the more serious consequences of the disease.

Certain epidemics and certain periods of them are marked by a great fatality, and instances have occurred in which all or nearly all of those seized have died. Age and family constitution have the greatest influence; where one member of a family has suffered severely from this disease, there is the more reason to dread its effects upon the younger members of the same family. The proportion of deaths to seizures in 1,321 cases reported in the *British Medical Journal* for 1858–59, is one in seven; among the severer cases, and at the height of any local epidemic outbreak, it was as high as one in three; in other cases it was less than one in ten. Of the seventy-four cases collected by the *British Medical Journal*,[3] twenty-six died,—four-

[1] Etude de la Diphthérite, par G. S. Empis. Arch. Gén. de Méd., 1850, s. 4, tome xxii. p. 298.

[2] Cases of Diphtheria, by G. M. Humphry, M.D. Case 7. Brit. Med. Journal, July 4, 1863. Dr. Ballard tabulates one case, loc. cit. p. 55.

[3] Brit. Med. Journal, 1859, p. 498. Of eighty fatal cases occurring in Islington in 1858–59, Dr. Ballard found that in fifty-eight

teen from asthenia, eight from implication of the larynx, three from syncope, and one from subsequent bronchitis. These cases were mostly severe, and seldom under treatment at their commencement. One of the most important conditions of a favorable result is the early recognition and treatment of the disease.

THERAPEUTICS.—In Diphtheria both local and general means of treatment are required; the cure of particular cases may sometimes be attributable to the one, sometimes to the other, but in no case can either be safely disregarded. The general therapeutical indications are of primary importance throughout; they consist neither in attempts to nullify a poison by specifics, nor to expel it by elimination, but in withstanding the encroachment of the disease, and in sustaining the vital powers.

Complete rest and purity of air are essential. Alcoholic stimulants are required throughout;[1] they are often as serviceable early in the disease as in that part of its course when they become indispensable. A rapid pulse indicates their employment, and heat of skin is no counter-indication. The limit to their administration should be calculated according to the age of the patient and the amount of bland liquid representing water that can be taken at the same time. In certain cases one or two full doses of quinine may be given, often with good effect when there is either vertigo, headache, or vomiting; soon afterwards beef-tea, eggs, or even more solid food, can be taken, as well as the brandy or wine; milk, in any form, is always suitable. The night must not pass without either nourishment or stimulant being given; wakefulness or nocturnal delirium is often thus obviated; sometimes a dose of morphia or opium has to be combined with the stimulant, after the second or third night, if restlessness then persist.

The condition of the fauces must be alleviated by the free use of ice sucked or swallowed, and by any of the soothing means hereafter mentioned internally, and by moderately warm applications externally. [One of the most comfortable and often really serviceable applications is ice, taken into the mouth in small pieces and swallowed slowly. Some practitioners assert this to have a more beneficial effect than any other local remedy.—H.] When the patient first comes under observation, a patch of exudation may be apparent, such as to require energetic topical measures. For this purpose a solution of nitrate of silver should be applied so as thoroughly to come into contact both with the patch and the turgid mucous membrane surrounding it. The strength of the solution should not exceed the proportion of one part of nitrate of silver to three of distilled water; the superficial whiteness left by it will clear off in twenty-four hours, and is easily distinguishable from the points of exudation. A mixture of hydrochloric acid and honey in equal proportions, or with one or two parts of water, is as effectual in checking the progress of the exudation, but leaves a more persistent white mark. These applications, when seen to be efficient, need not be repeated; care is to be taken that no excess of them reach beyond where they are required. The strong acid and solid nitrate of silver are both objectionable.[1] Where there is much redness and pain a weaker solution of nitrate of silver, one part to eight or twelve of water, pencilled over the whole surface, tends to prevent further exudation, and affords relief to the local discomfort. Hydrochloric acid, diluted with five parts of water, may be applied in the same way with the same effect.

As soon as nourishment, however light, can be retained by the stomach, five to ten grains of the perchloride of iron, the equivalent of twenty or forty minims of the tincture, should be given, with not less than half an ounce of water and half a drachm of glycerin; this should be repeated every three or four hours, or still more frequently, so that as much as a drachm of the iron, or half an ounce of the tincture, be taken in the course of twenty-four hours. This quantity may be reached even in children, during severe attacks. It should be commenced on the first day of the illness, or as soon as the

---

deaths twenty-seven were from laryngeal affection, and twenty-three from exhaustion; thus distributed as to age and period of illness :—

	Under 5 Years.	Under 10 Years.	10 Years and upwards.	1st Week.	2d Week.	3d Week.	4th Week.
Laryngeal affection . .	17	7	3	18	8	1	...
Exhaustion	13	5	5	8	8	3	4

[1] [Not, however, in every case.—H.]

[1] Extreme dysphagia has at once subsided on the use of solid nitrate to a foul or irritable ulcer consequent upon Diphtheria.

patient comes under notice, and continued till the tongue becomes red and the throat improves; when deposit has already taken place, the good effect of the remedy will be shown, not by any alteration in the dimensions of the patch, but by a diminution in the accompanying secretion, and by an improvement of the general symptoms; it is not to be discontinued for some days, and may require energetic repetition if improvement is slow in appearing. Dr. Druitt[1] reports a case where great benefit followed upon the quantity of the tincture of the muriate of iron, administered under the direction of one of the physicians of the Middlesex Hospital, amounting to one ounce and a half in the twelve hours. Mr. Hamilton Bell,[2] of Edinburgh, first proved the utility of iron in this form and quantity in erysipelas, and his brother, Dr. C. Bell, advocated its employment in analogous diseases; hence its application in Diphtheria. Dr. Godfrey,[3] of Enfield, used it in this way, and its use was further recommended by Dr. Heslop, of Birmingham; it was employed with advantage in the Boulogne epidemic, and M. Aubrun[4] gives the result of three years' favorable experience. The solutions of the pernitrate of iron, or of the acetate, answer equally well when given in proportionate doses, the latter being stronger, the former of less strength, than the tincture of the perchloride. A local as well as a general influence is exerted by these agents; they have a constringing effect on the vessels, and their action on the decomposing exudation is antiseptic; their general effect is as much owing to a topical action on the whole gastro-intestinal canal, as to absorption, for when the prescribed quantity of the persalt of iron is reduced by soda in the presence of citric acid, and so administered in a soluble form little liable to decomposition, and readily available for absorption, no favorable effect is produced; indeed, none of the protosalts of iron are equally efficacious at the outset of the disease. [Chlorate of potassium (mentioned further on) is regarded by many physicians in America as the most valuable of medicines in Diphtheria. It may be used early and freely; with adults, twenty grains every three hours; with children five years old, five grains, every two or three hours, will not be excessive.—H.]

The local action upon the surfaces affected is certainly important; and when the nasal passages are implicated, syringing them with perchloride in a solution slightly weaker than that prescribed for a draught, is essential; half a drachm each of the tincture of the perchloride of iron and glycerine with six or seven drachms of water, may be injected into the nares by an ordinary glass syringe, the point of which is shielded by a piece of india-rubber tubing, or into the upper part of the pharynx by means of the laryngeal syringe directed upwards. These parts may be gently and efficiently irrigated by placing a vessel of any fit liquid at an elevation, and inserting an india-rubber tube from it into either nostril. When the patch is already formed, and the secretion in the fauces considerable, it is well to apply a stronger solution of the perchloride, not exceeding the strength of the tincture, by means of a full-sized camel's-hair brush, both to the patch and to the adjacent surfaces. A patch of considerable thickness, and intimately adherent to the membrane, may be touched with a preparation twice the strength of the tincture, made by mixing equal parts of the liquor ferri perchloridi and of glycerine; this application is strongly styptic, and should be confined to the surface of the exudation, which it readily penetrates: and exerts its influence upon the vessels beneath. Where the use of the persalts of iron is commenced early, and persisted in, the necessity for local application to the throat is often obviated, and it is better to encounter a little difficulty in the administration of the medicine, which may be given in any degree of dilution, than to be obliged to resort to the always disagreeable, though often indispensable, performance of topical medication. The medicine is better given at the time of taking nourishment, the pain of swallowing diminishes under its use, and its application in any way to the whole pharynx affords relief.

Difficulty of deglutition is often a serious aggravation of the illness. Much benefit is derived from the application of the perchloride of iron diluted with water to the strength of the tincture, but with a further addition of glycerine, to the pharynx by means of a camel's-hair brush two or three times in the first twenty-four hours. Pure glycerine[1] applied in this way is very soothing, and the frequent use of more simple means conduces to relief. A lotion of acetate of lead, gr. ij ad ℥j, relieves. A weak solution of tannin, with a little chlorinated soda, is useful. Lime water as a wash, or gargle if the effort be not too painful, alleviates; so does the injection of cold water into the pharynx. The most grateful, and in all cases one of the most useful appliances, is ice in small pieces, dissolving in the mouth. Weak solutions of borax, or of

[1] Brit. Med. Journ. 1861, vol. i. p. 208.
[2] Edin. Monthly Journal, June 1851.
[3] Lancet, October 17, 1857.
[4] Gazette Méd. de Paris, December 8, 1860, f. 764.

[1] Dr. Mayer, American Journal of Med. Sciences, April 1858.

alum, with honey or glycerine, or a strong solution of chlorate of potash, are useful where there is much tenacious secretion; the latter salt in coarse powder, or small crystals of it, may be taken into the mouth from time to time with benefit. Any of these may be used either cold or tepid as may be most grateful to the patient, and when there is fetor, or the glands are much swollen, Condy's fluid (permanganate of potash), largely diluted, or chlorinated soda, one part of the strong solution to twelve or fifteen parts of water, is to be used so as to wash away any detached exudation, and this by means of a syringe if not otherwise easily manageable. The strong solution of chlorinated soda may be directly applied to any foul surface not yet detached, or to any foul ulceration when there is induration of the external glands. The Pharmacopœial glycerine of carbolic acid is a convenient local remedy when decomposing matters are tainting the breath. The similar glycerine of gallic acid checks hemorrhage from parts whence the slough is separating. The glycerine of tannin is most useful in the earlier stages of the disease, and is a necessary application to the fauces where bark with a mineral acid in the place of iron and quinine is the general medication adopted. Semi-detached shreds of decomposing matter are to be carefully removed; such matters cannot be allowed to remain with safety where they are; they must be reduced to the smallest amount possible, and antiseptics employed until they disappear. When exudation ceases, the necessity for local application has passed; some portions of deposit will be slow in clearing away, and may remain without interference; abrasions or ulcerations of the mucous membrane heal most quickly when left alone. A tender surface is never to be exposed by the forcible removal of any adherent exudation.[1]

All the remedies of special utility in Diphtheria are antiseptic: the stronger forms above indicated are limited in their application to parts that can be brought within the range of sight or touch. This range may in some states of the disease be extended by the use of the laryngoscope not only in the direction of the larynx, but also towards the posterior nares.

Many of these agents can be most advantageously used in all stages of the treatment by atomizing their dilute aqueous solutions in the manner first suggested by M. de Sales Giron. Any of the spray-producers now in use will effect this, and supply a ready means of diffusing the remedies over every part of the mucous surfaces obnoxious to the disease. Dr. Dewar, of Kirkcaldy, has shown that the strong sulphurous acid of the Pharmacopœia may be used at short intervals with benefit, and has devised a portable instrument with a vulcanite tube for the purpose. In one case where this was used without resort to the iron treatment in a child, no laryngeal extension of the disease occurred. A mixture of sulphurous acid and honey for frequent deglutition was useful in clearing the posterior nares; the fauces required an occasional application of glycerine of tannin; on the fourteenth day acute kidney complication began, and terminated fatally on the eighteenth day. When the tincture of perchloride of iron is given (generally combined with quinine), very little local treatment other than the spray is resorted to. Besides the weak Condy's fluid, a still weaker solution of carbolic acid, a teaspoonful to a pint of water, may be used in this way; a few spoonfuls of the latter solution is advantageously given occasionally as a drink. Both these liquids should be kept in the sick-room in a stronger form, both for diffusion into the air of the room by means of the vaporizer, and ready for disinfecting any secretions or soiled clothes.

There are other remedies that sometimes are necessary in checking the spread of Diphtherite, or in modifying its character. The salts of copper, as used by Aretæus, are very effective, but their use is not free from danger. Alum in powder (the *poudre croupale* of Pommier) is as effective, and both safe and simple in use; it may either be used alone, or mixed with one-third of its bulk of finely-powdered nitrate or chlorate of potash, or with a less proportion of sugar or gum; it may be applied to the throat by insufflation, as practised by Bretonneau,[1] or by making it into a paste with a drop of water, and carrying it to the part by means of a rod or spatula, or on the handle of a tablespoon. When required in the upper or lower part of the pharynx, or indeed

[1] Ætii Amid. tetrab. 2, serm. 8, cap. 46: "Nam inscii ad quos in rebus dubiis præcipuè homines confugiunt, vehementius illiniunt, simùlque inflammatum locum comprimunt, simùlque crustam detrahunt; quod minimè facere convenit, priusquam elevatam et vix innitentem crustam conspiciamus; quod si enim adhærentem adhuc crustam avellere aggrediamur ulcerationes magis in profundum procedunt et inflammationes consequuntur, augentur dolores, et in ulcera serpentia proficiunt: itaque sicca remedia insufflare oportet, liquida vero cum pinnula illinito, ita ut quantum licuerit, pinnulam penitissimè immittamus." "Ex quo præcepto docent medici, quod cùm pueri nequeunt gargarismatis uti, injiciantur cum siringâ medicamenta liquida." (Heredia op. Med. tom. iii. sec. 3, cap. xii.)

[1] Archiv. Gén. de Méd. tome xiii. p. 5, and tome xvii. p. 508.

on any part from the gums to the glottis, that best and safest of all instruments, the end of the finger, may be used for this purpose; in the same way, with the protection of a little cotton-wool wrapped around it, the other local remedies can be carried to any spot determined upon, without at the moment requiring the aid of sight. Calomel has an undoubtedly useful topical effect, as is seen in its application to cutaneous Diphtherite. Any sore on the skin should receive careful attention, as, however trivial at first, it may seriously compromise the prospect of recovery, either by spreading or by deepening so as to lay bare the subjacent structures. In addition to the requisite escharotics, cotton-wool should be used as a dressing; greasy applications are injurious.

The care given to the local treatment will fail of success, the diseased action checked at one part will reappear, or again extend rapidly, if the general condition necessary for a safe conduct through the illness be disregarded. Children, when improving, must continue in bed; it is the only place where they get rest of limb. There must also be precautions against fatigue from over-amusement or excitement. The period during which rest is to be enjoined is not merely while exudation remains, but while any symptom of the disease continues, such as slightly elevated temperature, excess in the excretion of urea or urates, or albumen in the urine; care is still more necessary in providing against any undue expenditure of strength during some of the after effects of the disease.

As long as solid food cannot be taken, it is to be noted with the greatest exactitude that the quantity of the liquid nourishment and of stimulant administered in the twenty-four hours is equal to the estimated requirement of the patient; the less the quantity of nourishment, the greater must be the dependence upon the stimulant. The youngest children may require a teaspoonful of brandy every two hours; a child of three years old two teaspoonfuls. This may be given diluted in any way and in very small quantities frequently repeated. Older children take it best mixed with iced water or soda-water. Champagne is often a good substitute; port wine requires dilution, except for adults, who also find good claret, red hock, or some of the stronger Hungarian wines, suitable. The quantity of stimulant ordered must be considered in relation not only to the immediate necessity that may exist for its employment, but also to the probable course of the disease and the strength of the patient; a moderate quantity, repeated at regular intervals, is of most service in maintaining the strength of the patient where all the symptoms are well marked and likely to go through their full stages; at certain crises of the disease, or where some symptoms only have attained great prominence, the benefit that results from a bold resort to stimulants is surprising, and the effect is the more marked, if their use had up to that time been neglected. Where the exudation is checked and is first separating, I have known the determined deglutition of four pints of beef tea and nearly two pints of port wine, in little more than twelve hours, by a temperate young man, teacher in a school, put a stop to further illness. Mr. M'Donald,[1] of Bristol, relates a case where bottled bitter-beer being the stimulant ordered, nearly eight pints were taken (twelve pint bottles were emptied) in the course of one night, with a like good effect. At a further period of the disease, when the separation of the deposit is completed, extra stimulants are required to combat the restlessness and depression then sometimes extreme. Sleep, at all times necessary, is at this latter period of the illness to be carefully conciliated. Opiates are well borne, and are now more likely to procure sleep than when the first symptoms in the throat are most troublesome; their use for several nights, when the diminished exudation reveals injury of the mucous membrane, may do much to prevent the exhaustion at this time so dangerous, and upon which stimulants, beyond what is necessary for support, seem to have but little power. Quinine and bark are particularly serviceable; the former may be combined with iron if its use be still indicated, the latter with the mineral acids. Quinine can be given to children in powder, and in solution is often advantageously added to the nutrient enemata that must so frequently supplement the inadequate amount of support otherwise received. Brandy is occasionally to be administered in this way, care being taken not to set up an irritability of bowel that might interfere with the continued employment of a means of sustaining life that so often becomes a necessity in the treatment of this disease.

Aperients are seldom required at the commencement of the illness, and during the earlier stages their effects are injurious; for then the waste of tissue is most active, and the impediment to the reception of nutrition the greatest. It has been a matter of frequent remark that exudation in the throat, already stationary, or even diminishing, has at once extended after the moderate action of aperient medicine. The only indication for their employment would be occasioned by intercurrent disease, which would give rise to

[1] Lancet, 1858, vol. ii. p. 539.

a greater elevation of temperature than usually obtains in uncomplicated Diphtheria; otherwise several days may be allowed to pass without action of the bowels.[1] The appearance of albuminuria does not call for their administration. Mr. Spanton found an increased excretion of urea follow the action of a purgative. Diarrhœa frequently demands attention; opiate injections, or small doses of opium with bismuth, will generally suffice; alum in boluses is mentioned by Bretonneau; small doses of sulphate of copper with opium are best in severe cases. The constipation of the latter stages of the illness requires stimulating enemata, friction to the abdomen, and galvanism.

Salines are contra-indicated. I have seen no good result from the administration of the alkaline remedies recommended by some French authors in the earlier stages, nor from the use of the iodides or bromides in the later. Ammonia, in continued doses, is injurious;[2] as a stimulant it is useless. The diaphoretic action of salines is not required, and their diuretic action has an unfavorable influence upon the specific irritation of the kidney.

Diluents and acid drinks should be continued while albuminuria persists; and though stimulants are then not always to be withdrawn, they are not to be injudiciously augmented. The hot air bath applied to the body only, without removal from bed, or warm packing to the loins, is serviceable in this complication; the appearance of blood-corpuscles or of tube casts in the urine indicates the administration of iron with the mineral acids, and the addition of cod-liver oil.

Mercurials, if continued till their general effect upon the system is produced, are productive of much mischief; Bretonneau's second memoir gives evidence of this. The carefully regulated action of them may be usefully opposed, when desirable, to some of the inflammatory complications of the disease. Salivation rather favors than checks the rapid extension of the special exudation.[3]

Bleeding is specially to be avoided; the worse effects of the disease seem to have been more extensively developed in many of the cases where this was practised.[4] Antimony also is prejudicial.

When the progress of the disease under the influence of appropriate general treatment seems most encouraging, death may suddenly threaten from impeded access of air to the lungs; relief then depends upon energetic local treatment, the same general means not being neglected. Symptoms that, did we not know their cause, would suggest the free employment of bleeding or antimony for their relief, may, if their cause is patent upon inspection of the throat, be efficiently obviated by a styptic application to the extending exudation, or to the orifice of the glottis itself, and their increase or return prevented by the free use of iron and wine. Nor are these to be entirely suspended if the mischief be altogether within the glottis. Where this is the case the symptoms are more gradual in their appearance, and may sometimes be alleviated by giving chlorate of potash, four grains,[1] to a dessertspoonful of water, by the insufflation of alum, or by the application of it to either surface of the epiglottis, or of glycerine to the edge of the glottis: the diffusion into the throat of a strong solution of chlorate of potash, or of sulphurous acid, by means of a vaporizer, may also be useful. It will depend upon the type of the general disease whether one or more doses of calomel are to be administered; the laryngeal symptoms once established, the resort to an aperient may be advisable, and calomel is the best that can be employed; where calomel will bear repetition emetics are also admissible. Senega may be used with this object. If ipecacuanha is chosen, ten grains of sulphate of zinc should be added. When secretion is abundant, alum, as elsewhere directed,[2] or sulphate of copper (five grains to the ounce of water), given in divided doses (a teaspoonful only for young children), is the best and most effectual. Emesis is not to be frequently repeated, and it is only when readily induced that it can be otherwise than prejudicial. Real and obvious relief, with increasing fulness of the chest expansion, is not only the encouragement for giving these means a trial, but the sole warrant for trusting to them for the relief of the temporary exacerbations. They must not be trusted to implicitly, as at any moment it may be evident that the obstruction is beyond their influence.

Tracheotomy should be performed whenever the increasing recession of the softer parts of the thoracic parietes shows that the cause of obstruction to the entrance of air is increasing. In the greater number of cases, if the local medication of the glottis and larynx do not suffice to obviate the danger, tracheotomy, performed early,

[1] Nuñes gives a caution against aperients at the latter stages of the illness, lest the death of the patient should be attributed to their use. Heredia, tom. iii. s. 3, cap. xi.

[2] See also Huxham's "Dissertation on the Malignant Ulcerous Sore Throat." Lond. 8vo. 1759, p. 53 et seq.

[3] See Lancet, 1838–39, vol. i. pp. 726, 728, for two cases illustrative of this.

[4] Memoirs on Diphtheria, pp. 50, 97. Arch. Gén. de Méd., tome xvii. pp. 494–7.

[1] [Twenty grains, in a tablespoonful of water, every three hours, will be better.—H.]

[2] *Vide* Treatment of Secondary Croup, Vol. III.

is much more likely to be successful than after the use of remedies that in any way impair the vital powers. A delay that admits of secretions accumulating in the bronchi is dangerous, and extension of the disease to the lung is the one insurmountable obstacle to success. Where the effects of the obstruction are more suddenly induced, tracheotomy, performed at the very last moment of apparent life, may save it. No degree of severity in the general disease should interfere with this means of averting threatened death from asphyxia, unless the presence of some other complication, necessarily fatal, can be demonstrated. I recently saw a case in consultation with Mr. Adams, in which, had it occurred at the commencement of the epidemic instead of towards the end, I should have decided against tracheotomy, concluding that it must end fatally; although unconsciousness had set in before commencing to operate, the child, six years old, recovered. At some periods of an epidemic, success less frequently attends this operation than at others; at the Hôpital des Enfants Malades,[1] in 1851, there were twelve recoveries in thirty-one operations; in 1853 only seven recovered in sixty-one cases; in 1856 tracheotomy was performed there fifty-four times, with success in fifteen cases.[2] Of 466 operations at this hospital in eight years there were 126 recoveries; these were generally operated upon as soon as the laryngeal symptoms were decided.[3] The result of M. Trousseau's 200 operations gives a similar success of more than one in four, while in the cases operated on by him in private practice one-half have recovered;[4] of nine such cases in 1854 seven were cured.[1] Of forty-two cases reported by Professor Rosen of Tubingen,[2] nineteen recovered. In six of the cases asphyxia had advanced too far before the operation; and of the subsequent deaths, one took place from pneumonia, fifteen days after, and one from albuminuria in the third week.[3] Dr. Buchanan writes, "I have performed tracheotomy twenty-one times with the result of seven recoveries; and if it be remembered that the patients were all on the point of death from suffocation, it cannot but be regarded as an encouragement to the surgeon to endeavor to save life by operative interference in the later stages of this most fatal disease." After the operation the chief condition of success is efficient support. The tube must remain in the trachea for at least a week; liquids are to be given in the form of sop; and nutrient enemata with brandy will generally be required. A little steam should be diffused in the apartment, and the tube lightly covered with gauze or porous woollen material. In adults, laryngotomy is often sufficient; in one case that occurred to me, the admission of air in this way sufficed to expel the exudation so speedily, that no tube was required.

The after-treatment of Diphtheria requires great care in proportioning the amount of exertion to the degree of strength existing. Good food, good air, and tonics are necessary. Galvanism may be required when an important function is impeded from muscular inaction. [Strychnia, used with proper caution, will be as likely to be beneficial in post-diphtherial paralysis as in any other form of paralytic disease.—H.]

[1] Arch. Gén. de Méd., s. 5, tome v. p. 360.
[2] M. André. See art. Croup, Vol. III.
[3] Edin. Med. Journal, vol. v. p. 417.
[4] Clinique Médicale. Paris, 1861, p. 414.

[1] Trousseau, De la Trachéotomie dans la Période extréme du Croup, &c. Arch. Gén. de Méd., s. 5, tome v. p. 360.
[2] Sydenham Society's Year-book, 1863, p. 278.
[3] On Tracheotomy in Diphtheria. By George Buchanan, A.M., M.D. Glasgow, 1865.

SCARLET FEVER.

By Samuel Jones Gee, M.D. Lond.

Definition.—An acute pyretic disease, specific in its cause and course, and best characterized by a peculiar exanthem hereafter to be described.

The earliest record which we possess of the existence of Scarlet Fever bears no more ancient date than A.D. 1556, the year wherein Ph. Ingrassias published a description of a malady which had been previously recognized by the common people, and named by them Rossalia. It is

possible that examples of a severer type of the same disease formed part of the famous epidemic of malignant angina which raged, sixty years later, in the south of Europe; in Germany, about this time, Sennertus witnessed what he considered to be the rossalia of the Italians, and what we may readily admit to have been our Scarlet Fever. In 1676 appeared Sydenham's short chapter on "Febris Scarlatina," and by the end of that century the disease had been described in every part of civilized Europe.

Causes.—I. Contagion is the only known cause of Scarlet Fever. In degree of contagiousness, the disease takes a place between measles and hooping-cough above, and typhus fever below; diphtheria being very far below. The contagious material may be taken up by clothes, and retained by them for a great length of time. Sir Thomas Watson narrates an instance in which a strip of flannel remained contagious for at least a year; and Hildenbrand was infected by a cloak which, after exposure to the disease, had been put aside for eighteen months; hence we may assume that the morbific principle of Scarlet Fever is anything but volatile or unstable. Which being so, affords an answer to the important question: When does a person who has recovered from an attack of the disease cease to be contagious? To speak strictly, not until those natural fomites, the epithelial scales, which were existing at the time of the fever, have been removed; or, what is nearly the same, not until desquamation has ceased. And in the fact that, under ordinary circumstances, these epithelial scales are all but permanently contagious we have an explanation of the tenacity with which danger clings to materials of any but the closest texture. Uncovering a scarlet fever patient in the direct rays of the sun, a cloud of fine dust may be seen to rise from the body; contagious dust, which, no doubt, subsides into every crevice near the bed. The distance at which the disease may be communicated is commonly said to be not more than a few feet; yet considering the slight volatility of the poison, one is quite prepared to admit the possibility of what is said to have occurred,—namely, of the contagion having been conveyed hundreds of miles by letter, or similar means. In like manner a person, himself not liable to the disease, may become a travelling disseminator of contagion: a trite observation, the application of which to practice is easily overstrained.

[The contagion clings long to rooms and houses; sometimes even for many months.—H.]

II. Such being the exciting cause, it is required that the subject be apt or predisposed. 1. This aptitude does not exist, as a rule, in those who have passed through one attack of the disease; still the possibility of recurrence and relapse is admitted on all hands. 2. Of persons not protected by a previous attack, whether the liability to contract the disease diminishes with increase of years or not, is not easy to say. The affirmative is probably true, yet no age is exempt. The percentage of deaths at different ages, according to Dr. Richardson, is,—

Under 5.	From 5 to 10.	10 to 20.	20 to 40.	40 and upwards.
67·63	24·43	5·52	1·73	0·66

Indeed all experience goes to prove that the majority of scarlet fever patients are of an age between eighteen months and six years. The relative proportion of cases of Scarlet Fever to all cases of illness, is greatest from four to nine years inclusive, and is, moreover, within those years, nearly stationary. 3. The two sexes are equally liable to Scarlet Fever; women after puberty suffer more frequently than men, because more exposed to contagion.

Scarlet Fever appears at any season; but in England especially prevails, as Sydenham says, at the end of summer; or, more strictly speaking, from the middle of September to the middle of November.

Incubation Period.—The duration of the incubation period partakes of that irregularity which attends most of the points which go to make up the history of Scarlet Fever. Trousseau relates a case which seems to prove that the incubation occasionally lasts no longer than twenty-four hours; this we may accept as a minimum. Positively to fix the maximum is impossible: probably seven days are rarely exceeded; but I have myself been witness to facts which did not admit of explanation otherwise than by presuming that the incubation period may sometimes last three weeks. Rilliet and Barthez are of the same opinion.

Symptoms.—Scarlet Fever is sometimes so mild as to escape the observation of both patient and physician, or, again, is sometimes so severe as to kill inevitably within twelve hours: another illustration of the wonted irregularity of the disease. Between these extremes lies a mean or typical form (here first to be described), in which all the characteristic symptoms are well developed, and none excessively: a description of the abnormal forms will follow. In the last place will be noticed the complications and sequelæ which may supervene upon any form of the disease.

Ordinary Scarlet Fever.

I. *Invasion Period.*—That stage which precedes the eruption of the rash.

The onset (except in mild cases) is sudden, so that it is often easy to fix the hour, almost the very minute, at which the disease began.

The symptoms are: 1. Sore throat (the first symptom noticed by most adults); tenderness at the angles of the lower jaw and stiffness of the neck soon follow. 2. Vomiting, the warning children give, but not so common with adults; it may be repeated many times, becoming ultimately bilious, yet not prognosticate, as a necessity, severity of the ensuing disease: active diarrhœa sometimes concurs. 3. Pyrexia. The first febrile symptom is often, not always, a sensation of chilliness; never a rigor; the face is pale. Flushing of the face and great heat rapidly succeed; a temperature of 104·75° may be reached on the first day. Pulse remarkably frequent, and frequent out of proportion to the height of the fever; for the pulse of a child to reach 160, or even more, on the first day of the disease, in prognosis, signifies nothing; in diagnosis, Trousseau thinks the symptom might be of some value. Respiration in proportion to pulse; no cough; no lachrymation: coryza before the appearance of the rash is very unusual. The tongue, mostly covered with a light white fur, except at the tip and edges, which are red, in some cases remains quite pale, clean, and moist; there are loss of appetite and thirst in marked instances of the disease. Skin hot, not necessarily dry. 4. Nervous symptoms are present, languor, sleepiness by day (especially in children), disturbed sleep at night, nocturnal delirium; headache, not severe, frontal; aching of the limbs. Convulsions even may precede the rash in non-malignant Scarlet Fever; coma, according to Valleix, is a frequent precursor of the rash of the normal disease, and Sydenham makes a similar observation. Lastly, in mild cases, mere poorliness the day before the eruption is often the only premonitory symptom.

The duration of this stage is, as a rule, from twelve to thirty hours. In trivial cases the rash is sometimes the first symptom of the disease. Not rarely the duration seems to be longer than the maximum given above; but proof is difficult; and the difficulty lies in fixing the time of occurrence, not of the first invasion symptom, but of the eruption; sometimes the rash recedes for a short time, and then comes out again. In a case observed by Trousseau, the rash did not appear before the eighth day, upon the cessation of most unusual prodromata, squinting, infrequent pulse and stupor, with headache, and vomiting.

II. *Eruptive Period.* — 1. *The Rash.* The normal exanthem consists of small dots, in color bright scarlet, most intense at the centre of the dot, fading towards the periphery; confluent by their margins, so as not to leave any skin of normal appearance between; not elevated to the touch; completely disappearing under pressure, and rapidly reappearing when the pressure is removed. The rash sometimes comes out over the whole body at once, but is mostly at first limited to a much smaller area, especially to the sides of the neck and the upper part of the chest. Occasionally the eruption appears first on the legs. The most common departures from the type are the following: The color may be deep, purplish, dusky (this being, alone, no sign of malignancy): in such a case add numerous flea-bites, and the disease may be (and has been) mistaken for typhus fever. On the other hand, the color may be very pale. The puncta are sometimes set so closely as to produce a uniform blush, in larger or smaller patches: this condition is constant indeed on the cheeks, and often seen on other parts of the face, and below the knees. Again the puncta may be perfectly discrete. Wherever there is hyperæmia there must be swelling of the cutis, not commonly perceptible, however, simply because the spots are confluent, but when they are discrete, it is not impossible to feel their elevation (sc. papulosa); in the latter case, should the color be at the same time more purple than usual, the rash of measles will be closely simulated. This swelling of the cutis must not be confounded with other occasional accompaniments of the rash,—namely, a cutis anserina, and a subcutaneous swelling, which is sometimes present to a notable degree in the eyelids, hands, and feet, and which may persist for a day or two after the rash has gone.

The eruption reaches its maximum extent and intensity on the third or fourth day of the illness (occasionally as early as the second); begins to fade on the fourth, fifth, or sixth day; and lasts altogether from five to ten days.

When the nail is firmly drawn over the skin in which the rash is present (over the belly or thigh is best) a white streak soon follows, lasts a minute, and then disappears; a very firm stroke brings out a middle red mark, and two lateral white streaks. The phenomena have been thought to be pathognomonic. But are they really peculiar to scarlet fever? Does the "white streak" show more than that the skin is injected, whereby the effect of a contraction of the small vessels in the cutis is heightened, and of a dilatation, obscured?

Miliaria are frequently, but far from constantly present. They are most com-

monly met with about the sides of the neck and upper part of the chest, but may be nearly universal. Occasionally they are so thickly set that the epidermis is detached by a rough touch. They are not connected with unusual sweating, but rather with the age of the patient and the amount of eruptive swelling of the cutis; the rash rarely being intense in an adult without the presence of miliaria. They dry up in a day or two and desquamate.

Sc. pemphigoidea seu pustulosa is described by continental writers. Large patches of urticaria sometimes come out while the proper rash is still present.

When the skin is pulled upon, the rash does not always altogether disappear; a yellow stain may be left, or all grades between this condition and actual petechiæ. Like a dusky rash, petechiæ alone do not indicate malignity.

2. *The Sore-throat* is always present to some degree. In a typical case, examination of the throat detects increased redness of the soft palate, uvula, and tonsils, sometimes of the posterior wall of the pharynx, and of the tip of the epiglottis. These parts are swelled also, but the amount of redness and swelling is often disproportionate; the latter may be so great that the tonsils meet in the middle line, and thrust the uvula forwards. Such great swelling is due to causes over and above the hyperæmia, namely, (1) Œdema; this is most notable in the uvula, which from gravitation of the serosity becomes club-shaped. (2) Excess of secretion: this enlarges the tonsils; *post mortem* they are found to be surcharged with thick yellow matter; during life the same secretion often appears at the mouths of the tonsillar crypts, or is exuded so as to cover the surface with a uniform layer. Superficial ulceration of the tonsil sometimes coincides. More rarely the matter does not escape, and, liquefying, causes an abscess in the tonsil, as early, it may be, as the sixth day. The mucous membrane is either dry and shining, or coated with thick mucus, which clogs the fauces. Ulceration of any part of the throat, other than the tonsils, before the fifth day, is very uncommon in cases which are not, for other reasons, classed under the gravest forms of the disease. There can be no question that the majority of "ulcers" and "sloughs" observed during the first four or five days of Scarlet Fever are nothing but excess of the secretion of the part smeared over the surface; and likewise no question that the dire sloughing and gangrene described so well by the older writers (*e. g.* Huxham, Heberden), and on their authority still attributed at times to Scarlet Fever, appertain altogether to diphtheria. To quote the words of Armstrong: "The first four or five days there will be seldom either sloughs or specks about the tonsils; but merely an increased secretion of mucus, some of which often adheres to the part and looks like an ulcer. But when the fever continues longer, or runs higher, specks generally form about the tonsils, which are finally converted into superficial ash-colored sloughs." Ulcerative angina, then, either occurs in cases conspicuous far more by the symptoms called malignant, or is a sequela, an epiphenomenon, supervening after the cessation of the specific disease.

The lymphatic glands at the angles of the lower jaw become enlarged and tender; in fact, inflamed. In severe cases the connective tissue around is involved, and puffy; but neither brawniness nor suppuration occurs before the rash has begun to fade.

Difficult deglutition, snoring, and alteration of the quality of the voice (it is thick and nasal), are results of the swelling of the throat. Kennedy once saw a child carried off by œdema glottidis within thirty hours from the onset of the disease.

In favorable cases the angina will recede with the eruption, and have disappeared by the seventh day, the tonsils perhaps being left rather swollen, but pale. The lymphatic glands often remain large for some time.

3. *The Pyrexia.*—The height attained by the fever has been subject to singular exaggeration; the fiery hue of the eruption, and the great dryness of skin often present, conspiring to mislead the observer. The mercury of a thermometer placed in the axilla never rises above 106° Fahr., rarely above 105°. The fever certainly runs higher than in measles, and very much higher than in diphtheria, but on the other hand does not reach the degrees which are often observed in ague, typhoid fever, rheumatic fever, and pyæmia. On the day when the eruption begins to fade the fever frequently submits to a complete crisis, as indicated by the temperature not rising above the normal for twenty-four hours; should this crisis not occur, the pyrexia is prolonged for an indefinite period. (The temperature of a healthy child may reach, but does not rise above, 99°.) In two cases the thermometric crisis occurred on the fourth day, in four on the fifth, and in three on the seventh. So, out of the cases observed by Dr. Ringer, the temperature of two became normal on the fourth day, of seven on the fifth day, and of one on the sixth day. In the rest the pyrexia was prolonged beyond the eruptive period. "The pyrexia slightly remits in the morning, and, rising in the course of the day, reaches its highest point about bedtime." (Armstrong.) A similar forenoon remission and afternoon exacerba-

tion takes place in health. Yet the Scarlatinal Fever is continual; the remission is not at any time complete until (in uncomplicated cases) the final crisis. The severer the fever the more trifling the remission.

4. *The Alimentary Canal.*—(1) Tongue. The fur present during the invasion period becomes thicker on the second and third days, and then begins to clear off from before backwards, so as to leave the tongue deep-red, broad, smooth-looking, and dotted over with elevated papillæ; a condition sometimes met with in other diseases (especially those attended with suppuration), when a thick fur clears off quickly. Both the earlier furred and later papillated condition are often absent. (2) Vomiting during the eruptive period is rare, except in the severer forms of the disease. Constipation, easily overcome, is the rule, but diarrhœa a not infrequent exception. Tympanites is sometimes present. Thirst and anorexia occur in well-marked cases.

5. *The Respiratory Organs.*—(1) Coryza, thin or mucopurulent, may supervene at any period of this stage, and is not necessarily a grave symptom. (2) It is rare to detect the physical signs of bronchitis or pneumonia before the rash begins to fade. Cough due to the condition of the fauces is common. The frequency of the respirations is proportionate to the pyrexia.

6. *The Pulse* maintains the frequency of the invasion period, and may reach 144 in an adult, 160 in a child, or even more, without any serious import. The frequency does not increase as the disease progresses, and it falls with the temperature.

7. *The Urine.*—(1) The water is diminished in quantity. (2) Urea. In an individual case of Scarlet Fever the most trustworthy standard of the amount of urea excreted by the person in health is derived from estimations made while he is kept in bed, and upon unaltered diet, for a week or ten days after the crisis. We must exclude all observations made upon cases in which the crisis of the pyrexia did not occur within the first week; and hence, in order to obtain sufficient data, the observations should be begun upon the urine of the third day at the latest. The following cases fulfilled these conditions:—

Sex.	Age.	Weight. Kilogrammes.	Days inclusive.		Water, cub. cents.	Urea, grammes.	Chl. Sod. grammes.
Boy	7 yrs.	22¼	1st to 4th	pyrexia	377	14·28	2·21
			5th to 12th	apyrexia	450	15·02	4·59
Boy	5 yrs.	16½	2d to 4th	pyrexia	551	18·83	0·97
			5th to 9th	apyrexia	709	13·33	4·69
Girl	10½ yrs.	27½	2d to 5th	pyrexia	316	16·17	0·67
			6th to 13th (minus 1 day)	apyrexia	567	19·64	4·38
					per 24 hours.		

These figures suffice to show that there is no necessary increase in the quantity of urea excreted during the pyrexia. (3) Chloride of sodium is more or less diminished, sometimes very much, sometimes very little. This diminution cannot be connected with any known concurrent condition. The salt is not subsequently passed in abnormal quantity. The diminution mostly ceases suddenly on the fourth, fifth, or sixth day. (4) Phosphoric acid undergoes very decided changes in quantity, which, normal, or a little more than normal, during the first three or four days of the disease, on the fourth or fifth day is notably diminished, for the four subsequent days remains at a half or a third of the normal, and then assumes the standard of health.[1] These changes bear no relation to the temperature or any other condition yet discovered; the same phenomenon of diminished excretion of phosphoric acid at the climax of the pyrexia, or soon after, appears in ague, measles, and probably in other febrile diseases. (5) Uric acid. The only suitable case in which I was able to make a daily estimation by weight of the uric acid manifested a great diminution (almost a suppression) in the quantity of the acid on the second and third days, a return to the normal on the fourth, and a great excess on the fifth day (the last day of pyrexia); after this the normal was resumed. It is highly probable that similar changes always occur in Scarlet Fever of any severity, as is judged from less exact observations upon several other cases. Sediments of uric acid and of urate of soda are common. (6) Pigment is not necessarily increased in quantity. (7) The occurrence of albumen during this period is considered under the head of Renal Dropsy.

8. *The Skin.*—Dryness is a frequent but by no means a constant condition: the patient may sweat; but for a sweat to follow a trivial exertion, say the move-

[1] These remarks are based upon daily observations by Neubauer's process in eight cases of Scarlet Fever.

ments of delirium, is a bad sign. Dryness signifies, not that the secretion of the skin is diminished, but that it is rapidly evaporated.

9. Yellowness of the conjunctivæ, tenderness over the liver, and evidences of bile in the urine, are occasionally present; and when present, devoid of prognostic value. It is rare that the spleen can be felt enlarged.

10. *Nervous System.*—An agitated manner, moderate frontal headache, and delirium, are common enough in ordinary Scarlet Fever. The delirium ("noctibus aliena loqui") which occurs during the evening exacerbation is not a grave prognostic, yet delirium is a prominent symptom of some of the worst cases of the disease.

The *duration* of Scarlet Fever is said by Valleix to be from ten to forty days. But what is the necessary duration of the specific disease, the Scarlet Fever, as distinguished from the possible duration of the illness, common sequelæ included? If we bear in mind that, in regular cases, the pyrexia often wholly ceases while the eruption still stands out well, I think we may agree with Heberden that the disease rapidly recedes after the seventh day, nay, I would add, that the specific disease even ceases then. The sore-throat may abate with the eruption, or be prolonged beyond, just as the catarrh of measles may increase on the eighth day of that disease.

III. *Desquamation Period.*—After the rash has receded, certain deviations from health are observed in all but the mildest cases.

1. If the pyrexia cease by the seventh day, the pulse falls in frequency often to below the normal: irregularity is common when the nervous symptoms of the prior periods have been well marked. The temperature often remains for a week or ten days below the point which it reaches subsequently. The urine is dilute, deficient in phosphoric acid, and often deficient in acidity; earthy phosphates precipitate, and vibriones form. All these conditions of pulse, animal heat, and urine, indicate an exhaustion of the vital energy. The tongue, if it have been characteristic (as is said), becomes, in the second week, more natural, and often assumes a delicately furred, silvery appearance.

2. *Desquamation.*—With the disappearance of the rash, the skin does not regain the characters of health; the epidermis is dry and shining, has lost its pliability, and is easily thrown into fine wrinkles. Desquamation begins in a day or two: the chin, sides of the neck, and upper part of the chest are mostly the parts first to desquamate. Where the cuticle is delicate, it becomes everywhere scurfy, and is so thrown off: where thicker, it is raised and removed in small isolated patches, which increase in size by a continuous desquamation of their margin, until they meet, and so leave the whole surface desquamated; and lastly, where the epidermis is very thick, it is undermined in large flakes before it is cast off. The amount of the desquamation depends very much upon the previous intensity of the rash: where the latter has been slight, there the former may be almost absent. Miliaria cause an abundant and early desquamation. The duration of desquamation is from a few days to a month or two.

3. The throat, in favorable cases, loses its redness; but the tonsils may remain swelled for some time: in like manner the lymphatic glands cease to be tender, and gradually diminish in size.

Such is normal or regular Scarlet Fever; it remains to trace the disease to its extremes, of severity on the one hand, and of mildness on the other. And first of Scarlet Fever of abnormal severity, commonly called

Malignant Scarlet Fever.

But before going further I must enter my protest against the manner in which the epithet "malignant" is used, or rather abused, with regard especially to Scarlet Fever, but also more or less to many other diseases. Malignity has almost ceased to have any real meaning, and all that some of the greatest physicians have written upon the subject seems to have been written in vain. This is not the place for me to discuss what malignity really intends, however much I may feel inclined to do so: in what follows I shall simply strive not to misapply the term in the case of Scarlet Fever.

The severer forms of Ordinary Scarlet Fever merge into a type of malignity characterized by excitement, followed by exhaustion. There are all grades of severity between this, the least grave form of malignity, and that which places Scarlet Fever almost on a footing with Asiatic cholera and the plague, that form in which a preliminary period of excitement is hardly to be perceived, so rapidly does collapse follow upon the onset of the disease.

Type I.—The malignity is deuteropathic, secondary to a state of ataxia or excessive disorder of the functions of the nervous system; in the nomenclature of Armstrong the malignity is inflammatory. The invasion symptoms are all well marked, yet even from the first, those which are to be referred to a disturbance of the nervous system predominate,—delirium, agitation, sleeplessness. The

premonitory stage over, the rash comes out well, the sore throat is considerable, the fever runs high. Delirium remains, exists at all times, and is active; the patient throws himself about, gets out of bed, sings loudly; his mind is confused; his eyes bloodshot; pulse frequent, full and soft; extremities warm; vomiting after food is a prominent symptom; there may be diarrhœa or coryza. But depression follows in a day or two, the delirium is much less active, the person lies muttering, the pulse becomes small, weak, and still more frequent, the face gets dusky, the cutaneous circulation is remarkably sluggish, swallowing is imperfectly performed, vomiting may persist, the belly is tympanitic. Lastly, stupor supersedes delirium, and death ensues. Sometimes the fatal issue is accelerated by a sudden attack of convulsions, the pulse ceases to be perceptible, the whole surface is blue, coma follows.

Type II.—The malignity is protopathic (congestive, in the phrase of Armstrong), the vital debility is primary and spontaneous, the disease goes straight to the source of life. The patient becomes suddenly pale and faint, vomits; the vomiting is repeated with short intervals, the bowels are relaxed, the hands twitch, there are great anxiety and feeble delirium. In a very few hours the depression is extreme, the whole exposed surface feels cold, yet the temperature of the armpit is febrile; the pulse is very small and frequent. Perhaps, for a time, the patient seems to rally; he sits up in bed, but his pulse remains very frequent, and his feet cold; a faint rash may appear. Depression returns, the face becomes pale livid, and perfect stupor (interrupted by convulsions) supervenes; pulse is excessively weak and frequent, respiration remarkably irregular, sweats break out, the skin is cold and mottled; these symptoms precede death. The duration of the whole illness may be less than twenty-four or even less than twelve hours.

The temperature of the body in the rectum and axilla is maintained at a tolerably high fever standard, but I have not yet observed that extreme elevation of temperature which is to be found in some cases of rheumatic fever, tubercular meningitis, sun-stroke, and injury to the brain. It is not given to any physician to observe with sufficient closeness many cases of protopathic malignity in Scarlet Fever; by far the most uncommon form of the disease. A child who died in this manner twenty hours from the onset of illness, afforded a temperature in the axilla of only 104° an hour and a quarter before death. Half an hour previously the temperature had been 105°: this was the highest degree observed, though the temperature was taken at intervals of about two hours. It is manifestly untrue to say that the vital debility is due to hyperpyrexia; they would be merely concomitant conditions in any case in which they should both occur.

Excluding the examples of these well-marked types of truly malignant Scarlet Fever from the whole medley of cases which are loosely called malignant, there is left a large and heterogeneous caput mortuum which consists in part of cases attended by unusual or unusually severe local lesions, and in part of cases which certainly may lay a sort of claim to be regarded as instances of a prolonged malignity. The latter class of cases runs a course more or less of the following kind. For the first day or two the attack does not seem to be unusually grave; but about the third day the patient is alternately restless and heavy, pulse frequent and feeble, expression vacant, face pale and thick-looking, mouth very tender, sweats follow a slight exertion; coryza, diarrhœa, tympanites, ulceration of the tonsils, and dysphagia, supervene, some or all; delirium is frequent, emaciation proceeds rapidly, and the patient dies exhausted, it may be during the first week, or not till far on in the third; the prolongation being due partly to the local processes set up and partly to inability to rally.

The rash in Malignant Scarlet Fever is of small prognostic value; it is often abundant and of a bright color; in the worst cases no rash appears, but none would be expected, death occurring before the second day. The tonsils mostly ulcerate early, even on the first day. The pyrexia lasts to the very end.

Latent Scarlet Fever.

The symptoms are so ill-developed as to be not characteristic or not observed. Examples of Sydenham's Scarlet Fever—disease by name alone—hold a mid place between this latent and the regular forms. How mild soever the primary disease, the gravest sequelæ may ensue.

[Popularly, *scarlatina* is, in some places, supposed to be the proper name for an unusually mild form of Scarlet Fever. It is important for physicians to correct this error, as it induces a want of care, not only in regard to the patient, but as to exposure of others to the contagion. There seems to be no doubt that the mildest cases may, by contagion, give rise to others of the greatest severity; and, also, that very light cases may be followed by considerable dropsical effusions.—H.]

Sequelæ.

As symptoms of the desquamation period, I have described sundry sequelæ of

Ordinary Scarlet Fever which are constant, and the necessary results of normal precedent conditions: there remains for consideration a long list of sequelæ which are inconstant in their occurrence, and therefore mostly looked upon as complications of the disease. The distinction may be somewhat artificial, but it is in the present state of knowledge convenient.

I. *Sore-throat.*—Any sore-throat which may be present during the decline of the rash, or after it, is due either to a simple persistence of the ulcerated condition of the tonsils, which has been described as an occasional symptom of the eruptive period, or to the establishment of a new form of lesion. In the former kind of sore-throat, the ulceration tends to progress deeply rather than widely, and bubo is a frequent concomitant. The latter kind supervenes most frequently on the sixth day, sometimes a day or two later, but never after the end of the second week: in very many cases the fauces have previously escaped grave implications; then, at the time specified, one or several small grayish patches, surrounded by a vivid red margin, appear on the tonsils, uvula, or arches of the soft palate, sometimes on the hard palate or the tip of the epiglottis. At the same time the fauces are swelled and reddened, deglutition gives pain, the lymphatic glands at the angles of the jaw become larger and tender, the connective tissue is puffy. The epithelium of the tongue is sometimes raised in patches. The excoriations go on to heal or to ulcerate. If to heal, the pellicle becomes thicker, so as to look like a false membrane, the surrounding redness abates, and in a short time, when the scab is thrown off, the mucous membrane is left uniform in surface. In the other alternative, when the pellicle is removed, the deeper layer of the mucous membrane is left exposed, ulceration ensues at this spot, and thence may spread far and wide; in the worst cases the tonsils are destroyed, the cartilage and bone in the vicinity are laid bare, dysphagia ensues, the fluids return through the nose. The state of the sides of the neck corresponds: in the favorable cases the swelling gradually disappears; in the severe, all distinction between gland and connective tissue becomes lost, a "collar of brawn" extends around the neck (scarlatinal bubo), softening ensues at places, and here the skin will slough in order to give exit to a shreddy sero-pus; yet the swelling is not diminished until large sloughs of the connective tissue come away, and the skin (it may be of the whole neck) is left floating over the parts beneath, and perforated in numerous holes. The suppuration reaches downwards into the mediastinum, or, spreading upwards, invades the parotid glands, passing along the interstitial connective membrane, and dissecting out the salivary lobules. Hemorrhage from the large vessels of the neck sometimes occurs to increase the complication. The rapidity with which the hard brawny swelling will establish itself is remarkable: a neck which was only puffy one day may be hard the next; a semi-comatose condition is often produced: the veins of the eyelids are turgid. The majority of cases of extensive sloughing of the connective tissue die; symptoms not directly connected with the throat are apt to supervene, particularly vomiting and diarrhœa. Simple suppuration is a much less grave result; and indeed a very considerable amount of firm swelling of the neck may be resolved without suppuration.

II. *Coryza.*—Coryza, which persists into or supervenes during the second week, is an important symptom, indicative of a morbid state of the mucous membranes in general. The direct consequences of coryza are often serious, and especially the extension of disease along the Eustachian trumpet into the tympanum; when this cavity is filled with pus, the membrane bursts or sloughs, and an obstinate otorrhœa results. Fistula lacrymalis is much less common. Worst of all is the coryza, which is indicative of nasal diphtheria, a complication comparatively infrequent.

III. *Otorrhœa.* — Otorrhœa is of two kinds. The first is that otorrhœa which begins in the external meatus; the discharge, which is whitish at the onset, soon becomes purulent, while the lining membrane everywhere thickens. The second kind of otorrhœa is that which follows coryza; ulceration of the lining membrane of the tympanum, necrosis of the bone, facial paralysis, incurable deafness, and abscess of the brain, these are consequences not uncommon: the discharge is purulent from the first; when injections are used, they pass from the nose out at the ears, and the reverse. Hemorrhage from the internal carotid artery is a rare sequel.

IV. *Diarrhœa.*—This sometimes coincides with the decline of the rash. Bloody stools and excoriations of the anus and buttocks may be the consequence of this, as of other kinds of diarrhœa.

V. *Bronchitis and Pneumonia.*—Complications more common in the second than the first week, and mostly preceded by coryza. Severe bronchitis is not necessarily fatal; when it is fatal, we always find lobular pneumonia superadded. Primary lobar pneumonia sometimes occurs.

VI. *Abscesses.*—Convalescence is often put back by the formation of one or more abscesses; if they are very large or numerous, they may be the immediate cause of death, and even when they are small and solitary the local results are sometimes grave. For example, on the back of the

hand, an abscess as it closes leaves the tendons glued to the neighboring parts. Or again, behind the pharynx, an abscess may cause death, especially in infants. The symptoms of post-pharyngeal abscess are dysphagia, return of fluids through the nose, some stiffness of the neck, one side of which may be fuller than natural, and dyspnœa. Any one, or even all of these symptoms, may be so slight as not to attract attention. The finger passed into the pharynx feels a soft tumor projecting in the middle line or somewhat to one side. Should the abscess burst spontaneously, sudden suffocation may ensue. Abscesses about the neck are nearly always, if not always, the result of internal ulceration.

VII. *Rheumatism.*—Towards the end of the second or beginning of the third week, we occasionally observe a considerable increase or a re-establishment of the pyrexia, and at the same time an affection of the joints; the latter consisting in great tenderness and elastic swelling (more around than in the joint), with or without redness of the skin; the patient sweats at times, perhaps profusely; the serous membranes are prone to inflammation. There are all grades of severity between transitory pain in a single joint and painful swelling of nearly all the joints of the body. A similar complication may attend the eruptive period, but is less common; it is common enough for adults to complain of great tenderness of the muscles while the rash is present. The swellings are mostly resolved, and then the patient recovers; but sometimes suppuration occurs around and in the joint. Suppurative rheumatism is fatal. The nature of this complication must, at present, be left an open question, whether the disease be really rheumatic, or whether it be pyæmial.

VIII. *Renal Dropsy.*—Scarlet Fever is very often followed by albuminuria, dropsy, and a form of nephritis; three accidents which it is convenient to group together and to view as collateral symptoms of a more general condition.

1. *Condition of the Urine.*—The proportion of cases of albuminuria seems to differ with different epidemics: albumen was at no time present in the urine of six out of twelve children, the whole of whose urine was saved and examined daily by myself, from the decline of the rash to the fourth week; Abeille found albuminuria in one-third of the cases examined by him; in the experience of Begbie, Newbigging, and Holder, the occurrence of albuminuria was a rule without an exception. In twelve cases I tested the urine daily throughout the fever and the convalescence; in one, albumen appeared on the fourth day, in one on the eighth, and in none was albumen detected for the first time after the twenty-first day; four cases occurred during the second week, seven during the third. In eight out of twenty-one cases observed by Abeille, the urine became albuminous before the sixth day; Jaccoud has noticed albuminuria as early as the second day. In the two above-mentioned instances of the occurrence of albuminuria on the fourth and eighth days, the abnormal condition of the urine was present for one day only; in the ten other cases the duration of the albuminuria was at least a week. In most of the examples of the more enduring form of the disease it is easy to distinguish three stages, each characterized by a peculiar condition of the urine.

Stage 1.—The quantity is diminished (total suppression for several days has been observed by others now and then), the specific gravity is increased in proportion to the concentration, urate of soda is precipitated, the color is unchanged, and, when any saline deposit is dissolved, however turbid the urine be with organic matters, it is not at all smoky: the urea is diminished in quantity beyond the proportion of the diminution of the water; the relative lack of chloride of sodium is greater still; albumen is present; when the diminution in the quantity of urine is considerable, the microscope discovers casts, some clear and perfectly free from granules or epithelium, some finely granular, but hardly any blood disks or renal epithelium; when the disease is less severe (and in all cases as this stage is passing away), there are renal epithelium and epitheliated casts, with a few blood disks. The amount and the duration of the diminution of the quantity of the urine in this stage are to a great extent prognostic of the future course of the disease.

Stage 2.—The quantity of urine regains the normal, and soon exceeds it; there is a perfect diuresis; the specific gravity falls low; the urea and chlorides return to the standard, or nearly so; the urine becomes bloody (from slight smokiness up to the deepest brown); the amount of albumen is relatively less, though it may be absolutely greater than in the earlier stage, and is not proportionate to the depth of the color of the urine; the microscope shows blood disks, hæmatoidin crystals(?), renal epithelium, epitheliated and granulated casts. As this stage passes off the urine becomes clearer, less red, and more yellow (passing through brown and green); the albumen lessens.

Stage 3.—The diuresis continues, but the smokiness disappears; though dull with excess of organic flocculence, the urine is normal in color; the albumen gradually disappears. Even after albumen has ceased to be present, it mostly happens that for some time the quantity of urine secreted is above the normal.

In mild cases the distinctions of the stages are necessarily ill-marked; sometimes the urine is at no time diminished in quantity; sometimes, although the first stage is well marked, the urine is hardly at any time smoky; sometimes the third stage never comes to an end, or in other words the urine remains permanently albuminous; a sudden increase in the depth of the bloody color of the second stage often occurs, without the quantity of albumen in the urine being proportionally increased; the urine returns to its prior condition in two or three days. A class of cases, by far the worst of any in a prognostic point of view, has been exemplified by several children who, when first they came under observation, brought a history to the effect that two or three months previously they had had Scarlet Fever (perhaps so mildly that the nature of the illness was ignored at the time) and that after the lapse of several weeks dropsy had supervened, not suddenly, but gradually, increasing from day to day. The urine is normal in color and specific gravity, clear or with some excess of flocculence, not necessarily diminished in quantity; but the amount of albumen present is very large; very little is to be seen by the microscope, and that little is not characteristic. The steps by which this stage is reached have not yet been observed; so far as the writer has seen the condition is permanent and resists all treatment.[1]

2. *The Dropsy.*—Dropsy or hæmaturia is the first symptom which arrests the attention of the inexpert. The former is very uncertain in occurrence, and is often almost wholly absent from first to last; when it is the first symptom noticed, it supervenes rather suddenly in the third week, and especially towards the end of that week. Dropsy is mostly preceded by albuminuria for a day or two; occasionally the dropsy precedes the albuminuria very rarely, when both symptoms have been present, the albuminuria nearly wholly (possibly wholly) ceases and yet the dropsy remains. In some epidemics, dropsy without albuminuria at any period is common. Philippe of Berlin did not once find the urine albuminous in more than sixty cases of scarlatinal anasarca (quoted by Jaccoud). The dropsy assumes the forms of—(1) Anasarca, which may be general or limited to the eyelids, backs of hands, and bottom of back: œdema glottidis is a very rare result of scarlatinal dropsy. (2) Serous dropsies, which occasionally precede the anasarca: when peritoneal, the effusion is of no great practical moment: the diagnosis of pericardial effusion depends upon physical examination; dyspnœa, intermitting lividity, indistinctness of the pulse, and a tendency to syncope, may or may not be present: pleural dropsy, hydrothorax, is a more common and formidable complication. A child whose anæmia and anasarca have shown a tendency to increase, and who has a slight cough and some sonorous rhonchi over the lungs, suddenly becomes much worse, vomits repeatedly, dyspnœa and lividity ensue and rapidly increase, very little urine is passed; the distress is painful to behold; and death rapidly follows, mostly within twenty-four, sometimes within six hours from the first exacerbation of symptoms. *Post mortem* both the pleural cavities are found to contain a great excess of serum, which is perfectly clear, or floats the network of a delicate coagulum; sometimes lymph is present, the evidence of pleurisy hereafter to be described. The lungs are collapsed but œdematous also, dark iron-gray on section, and capable of imperfect insufflation.

3. *General Symptoms.*—(1) Pyrexia accompanies the onset of renal dropsy, and is high in proportion to the severity of the symptoms; in mild cases pyrexia is almost absent: it is rarely prolonged into the second stage of albuminuria. All the more chronic cases of renal dropsy are, when uncomplicated, apyretic. The attendant symptoms are heaviness, dry skin, troublesome vomiting, parched lips, and constipated bowels. (2) Anæmia is mostly a marked symptom after the pyrexia has passed off. Children who are dropsical look more pale than those who are not. (3) Uræmia: convulsions and coma supervening upon scarlatinal dropsy are more often symptomatic of the onset of some local inflammation, than indicative of uræmia. Indeed true uræmic convulsions are not very common; when they do occur they induce a fatal issue in a minority of cases: on the other hand the prognosis is most unfavorable when the convulsions merely assume the place of a rigor.

The cause of renal dropsy is unknown: that exposure to changes of temperature will insure dropsy when the urine is albuminous (or rather, perhaps, when that condition which tends to produce albuminuria is present) has been admitted by most observers; but that a chill is a frequent cause of albuminuria is a dogma much more disputable, and apparently disproved by the fact that albuminuria is a sequela almost constantly present in some epidemics and almost constantly absent in others.

IX. *Serous Inflammations.* — They mostly, not always, are accompaniments

[1] Soon after the first edition of this volume was published, Dr. Weber read a paper upon some cases of this kind before the Medical and Chirurgical Society. Transactions vol. xlix. p. 199.

either of albuminuria or of the rheumatic state. 1. In the latter, the pericardium is especially prone to inflame, a complication which, by itself, does not render a prognosis unfavorable. 2. In albuminuria, pleurisy is the most common. One or both pleuræ are inflamed, the lymph effused rapidly breaks up into pus, and in most cases, death ensues in a few days; yet occasionally a chronic empyema is the result. States intermediate between pure hydrothorax and pleurisy are sometimes met with. Pneumonia is frequently combined with the pleurisy. In the pericarditis and peritonitis likewise the lymph has a great tendency to become puriform. It is not rare to find all the serous membranes inflamed at once. 3. Occasionally pleuro-pneumonia, pericarditis, or peritonitis supervenes, and there are neither dropsical nor rheumatic symptoms present. Yet such sequelæ are especially apt to occur in what may be termed the rheumatic period; that is, the second week. 4. Systolic cardiac murmurs sometimes spring up during the illness: they are heard with greatest intensity at the apex or the second left interspace. Neither albuminuria nor rheumatic symptoms necessarily accompany the development of the apex murmur: nay more, it can hardly be said to be proved that the occurrence of a systolic murmur at the apex of the heart is always due, in these cases, to endocarditis. These apex murmurs in my own experience persist for a month or two at least,—persist as long as the patient is under observation. Sometimes possibly dilatation of the heart originates in an attack of Scarlet Fever.

X. *Other Sequelæ and Complications.*—(1) Sloughing of the cornea is an accident of which the occurrence may probably be always prevented. (2) Hemorrhage in consequence of a sloughing bubo, of destruction of the pars petrosa, or in the form of hæmaturia, has already been described. Epistaxis is an occasional phenomenon. Here may be mentioned that variety of Scarlet Fever which many writers, probably somewhat biased by the analogy of smallpox and measles, have been very ready to admit into their nosology, I mean Sc. hæmorrhagica; a form of the disease which must be very rare (except, perhaps, in certain epidemics),—so rare that the occurrence of a passive hemorrhage from several mucous membranes at once might make one with justice suspend a diagnosis of Scarlet Fever until the notion of possibly having to do with variolous roseola was discarded. I have known Variola hæmorrhagica to be mistaken for Sc. hæmorrhagica, and the truth not appear until, in course of time, those who had dealings with the case were themselves attacked by smallpox. (3) Gangrene. Cancrum oris is a very uncommon sequela of Scarlet Fever. Gangrene of the pharynx is said to have occasionally supervened. Sloughing of the skin over a bubo is much more common; so also is gangrene of blistered or ulcerated parts of the skin. Here may be classed such sequelæ as necrosis of parts of the jaw-bones, and hip-disease. (4) Tubercle. Scarlet Fever by no means tends to develop tubercle even in a subject predisposed. (5) Other acute specific diseases. The eruptions of Scarlet Fever and measles may appear at the same time upon the same patient (Rilliet et Barthez, iii. 281). Smallpox has been known by the same writers to complicate Scarlet Fever. I myself have made this observation; a girl of three had hooping-cough; about a week after she began to whoop she became very feverish,—this was on April 5th; on the 6th and 7th she vomited beyond what the cough would account for; on the 8th an indubitable varioloid eruption appeared upon her, and on the 9th, a rash which possessed in every respect the scarlatinal character. In another girl of two and a half I saw the eruptions of Scarlet Fever and varicella appear upon the same day. I have known the long course of typhoid fever to be broken asunder, as it were, by an attack of Scarlet Fever. Diphtheria is a comparatively frequent sequela: the patient seems to be in a fair way of recovery, when an acrid discharge from the nostrils is noticed, the neck swells again, the pyrexia returns: death is inevitable (Graves, Clin. Med. i. 318; Trousseau, i. 15); this complication may ensue as early as the fifth day (Rilliet et Barthez, iii. 168). The writer has seen diphtheria supervene upon the chronic dropsical cachexia, the new disease here also inducing rapid death. Lastly, Scarlet Fever may be intercurrent during the acute period of rheumatic fever, and neither disease be perceptibly modified.

[Although much more rarely than is the case with diphtheria, Scarlet Fever may be followed by partial *paralysis;* especially of the lower limbs. This is seldom of a very severe grade, but it may require weeks, or sometimes months, to pass away.—H.]

Diagnosis.—During the invasion period the diagnosis depends upon the presence of vomiting or angina, and the absence of sneezing, lachrymation, or pain in the back, in a person who has been taken ill suddenly, and has not previously suffered from Scarlet Fever. The frequency of the pulse, and severity of the nervous symptoms from the first, are sometimes valuable aids to diagnosis.

The rash may be confounded with the eruption of measles, smallpox, typhus, roseola, miliaria, or urticaria. Measles

may be wrongly suspected when a Scarlet Fever rash is of a darker color than usual, and especially when it is discrete also. And, contrariwise, in several cases of measles I have been perplexed by the existence of a scarlatiniform rash upon the body several hours before the proper eruption of measles appeared. The roseola which precedes smallpox occasionally causes an error in diagnosis. The resemblance which the rash of Scarlet Fever occasionally bears to that of typhus has been alluded to previously. To know that the minute vesicles of miliaria and the confluent wheals of urticaria are not unfrequently mistaken at first sight for Scarlet Fever rash, will suffice in most cases for the diagnosis. Reviewing some of these statements, and bearing in mind how closely the rash of Scarlet Fever and of measles may simulate each other, that the two diseases may concur, and that symptomatic roseola is not always easily distinguished from Scarlet Fever on the one hand or measles on the other, it is fair to conclude that at least some supposed cases of a disease which has been called rubeola (rötheln, roseola febrilis) are attributed to an impossibility in the diagnosis.[1]

Scarlatinal angina is indistinguishable *per se* from ordinary simple erythematous sore throat, or from pellicular angina (pharyngeal herpes) which forms so large a proportion of the sore throats popularly styled diphtheritic.

The swelling which occurs about the joints during the first two or three days of the disease is sometimes very considerable, while the rash is pale or absent; such Scarlet Fever has been mistaken for rheumatic fever.

Surgical Scarlet Fever.—It has been doubted by some whether the scarlatiniform rash which sometimes follows operations is really scarlatinal. The eruption appears from the second to the sixth day after the operation, and, in the cases which have caused the doubt, is very fugitive, the first and only symptom. Yet, that the disease really is Scarlet Fever, would seem to be proved by the following observations: first, that the disease occurs in epidemics; secondly, that in a given epidemic a severe case occasionally relieves the monotonous recurrence of the very mild form; thirdly, that a precisely similar scarlatinilla attacks, in the same epidemic, patients who have not been subjected to operation, and who have no open sore; and lastly, by way of a veritable *experimentum crucis*, that, however freely these patients are exposed to Ordinary Scarlet Fever contagion afterwards, they do not contract that disease.

[1] [See article on "Rötheln."—H.]

Morbid Anatomy.—Scarlet Fever does not possess any distinctive anatomical character which persists after death: the alterations of texture found *post mortem* are common to other pyrexiæ.

The furred tongue indicates a condition, similar in kind, if not in degree, of the mucous membrane of the whole alimentary canal; there is an excessive formation of epithelium and concomitant hyperæmia of the sub-epithelial layers; casts of the gastric tubuli are to be detected by the microscope in the vomit. (Fenwick.) From the frequency of coryza, pulmonary catarrh, and pneumonia, we infer that the respiratory mucous membrane does not escape.

A peculiar change in the muscular tissue has been described by Zenker; a change more constant, as it would seem, in typhoid than in Scarlet Fever.

The puffy swelling which sometimes precedes the rash or coincides with it, may indicate a direct implication of the connective tissue.

The spleen, the lymphatic glands, the tonsils, and the lymphoid (solitary or agminated) glands of the stomach and intestines are swollen and injected; the tonsils and the gastro-intestinal glands sometimes go on to ulcerate. In this place may be just mentioned the leuchæmic deposits which have been detected here and there.

The liver and the kidneys are involved to a slight degree; a little cloudiness of the cortex of the kidneys by no means indicates the existence of albuminuria. The kidneys of renal dropsy will be described under the head of Kidney Diseases.

The cerebral symptoms are, so far as is known at present, unaccompanied by anatomical change.

Prognosis.—I. *Prognostics derived from pre-existing conditions.*—1. The social position of the person attacked has no influence upon the course of the disease, which is quite as fatal among the rich as among the poor. 2. Family constitution seems to influence Scarlet Fever to a degree which can be hardly overrated; that one or more members of a given family have succumbed, renders the prognosis in the case of any other persons in that family who may chance to be attacked, very grave. 3. Pregnant women are said to be peculiarly exempt from the liability to contract Scarlet Fever; but the puerperal state predisposes to the occurrence of not only Scarlet Fever, but Scarlet Fever of a very fatal form. The rate of mortality seems to differ in different epidemics; but whether of thirty-six patients attacked all die (Trousseau), or the mortality be twenty-five per cent. (MacClintock), or all of nine

patients recover (Blakely Brown), the prognosis cannot be too guarded. 4. A scrofulous vulnerability of the mucous membranes or the lymphatic glands greatly adds to the gravity of the prognosis. 5. A previously feeble state of health by no means predisposes to a bad type of Scarlet Fever—almost the reverse; certainly the majority of cases of Sc. maligna are in persons of previously robust health. 6. Age and sex have no effect. The epidemic constitution does not always afford trustworthy aid in a particular case.

II. *Prognostics derived from the actual disease.*—The number and the gravity of the possible complications and sequelæ of Scarlet Fever should render the prognosis very wary even in the mildest cases. Excluding the malignant forms, we judge of the severity of the disease more from the number and severity of the local lesions, than from the height of the pyrexia or the color of the rash; each additional local lesion, complication, or sequela, being an addition to the gravity of the prognosis. Bubo and coryza are the most formidable symptoms of the first ten days, rheumatism and albuminuria of the next ten days. There are no guides to prognosticating the probability of the occurrence of bad sore-throat, rheumatism, or renal dropsy. If on the first day of deflorescence of the rash the fever still keeps up, we must attribute it to a new or an increased local lesion, and this will mostly be found to be anginal. The gravity of a brawny bubo is always great. A tendency to relaxed sore-throat does not predispose to bad angina. In most cases in which coryza has appeared on the fourth or fifth day, the secondary sore-throat will ensue to a certain extent. Of the patients seen by MM. Rilliet and Barthez, all who during the first fifteen days of the disease exhibited convulsions, convulsive movements, rigidities, in other words, symptoms referable to the locomotive apparatus, have died; according to my own experience, the prognosis which might be derived from this statement would be too unfavorable. The nocturnal talkative delirium (noctibus aliena loqui) is, no doubt, as Heberden says, an unimportant symptom. But when delirium becomes more active than this, more constant, more independent of surrounding things; when attended by restlessness, sleeplessness, drowsiness, or by vomiting and diarrhœa, it is impossible to dissemble the grave significance of the symptom. Suppurative arthritis is a most serious accident; multiple abscesses are much less grave. In the renal dropsy, moderate œdema pulmonum and pleural effusion may be recovered from. The reader will find many other prognostics under the heads of the sundry symptoms.

Prophylaxis. — Removal from all sources of contagion is the most obvious, the surest, and probably the only means of preventing Scarlet Fever. The prophylactic virtue of an infinite number of fumigations and drugs has been vaunted from time to time, but fruitlessly, with one exception, namely, belladonna; yet even of that remedy the reputation, not wanting the support of hundreds of observations tabulated, has in the present day sunk very low. Inoculation by means of the blood, the fluid of the miliaria, or the secretions of the fauces, has been practised in a few cases, and so far, apparently, not without favorably modifying the disease thereby communicated.

With reference to the purification of materials which have been exposed to Scarlet Fever, it may be here remarked that the morbific principle is destroyed by a heat considerably below the boiling-point of water.

Treatment.—In the following pages I shall endeavor to trace an outline of the plan of treatment ordinarily adopted at the present day. It will be impossible not to omit mention of many curative means which may be of real value; in a common disease, not very powerfully controlled by any therapeutic method, the remedies which have been and which are employed must be innumerable.

To know the natural course of Scarlet Fever is a great guide to the appropriate treatment; for, although we cannot abridge that course, we can be prepared for the occurrence of different accidents at different stages, and so be able to meet those accidents from the very first. Provided that no complications occur, Scarlet Fever will terminate favorably within a week from the onset of the disease. Among the complications we must place the malignant form of the disease, and also any unfavorable pre-existing condition, such as the puerperal state.

The regimen of acute diseases must be put in force. The patient should be kept in bed, as a rule to which there is no exception; the bed-clothes should be those to which he has been accustomed in health, and no more; carpets, curtains, and porous materials must be removed; the bed-room should be carefully ventilated (in part by an open fire), bearing in mind that there is no special reason to fear cold during the first week; the whole surface of the body should be sponged with tepid water once or twice a day, and subsequently to grease the skin with mutton suet often brings comfort to the patient. The diet is to be unstimulating, consisting of milk, broths, the farinacea, an egg, light puddings; drink should be freely supplied. Purgation is to be avoided.

In many cases a consideration of the previous and the present condition of the patient will indicate the administration of wine: the child seems low, the pulse is not only frequent but soft and feeble, there is possibly coryza present. At the same time full doses of carbonate of ammonia should be given, in milk, every four hours. And it must be conceded that no great harm comes from the moderate employment of stimuli, even when they are not absolutely necessary.

When the throat is much inflamed, great relief may be afforded by either of the following methods of treatment. The first is to cause the patient to use ice freely; he is to allow lumps of it to dissolve in his mouth. Under this treatment tenderness of the submaxillary glands sometimes passes away in a few hours, an improvement which is an index of that which has taken place within. Ice can be easily given to patients above five or six years of age; but the second remedy is one which is inapplicable in the case of children; I mean the inhalation of the steam of hot water. Puffy swelling of the neck may often be removed by external applications of spongio-piline wrung out from hot water, or of hot linseed-meal poultices, frequently renewed.

In like manner coryza is a symptom which, when treated early, may thereby cease to be of evil omen. To check the coryza is to check, in the majority of cases, that worst form of otorrhœa which proceeds from the destruction of the tympanum. In patients above eight years of age, the valuable method of washing out the nostrils introduced by Dr. Thudichum may be employed. An ounce of salt is dissolved in a pint of warm water, and this solution, contained in a vessel a little raised above the head of the patient, is conveyed by means of a flexible caoutchouc tube into one nostril; respiration being carried on through the mouth, and all attempts at swallowing forbidden, the fluid passes out freely by the other nostril.[1] In young children we are reduced to the expedient of syringing the nasal fossæ with a weak solution of nitrate of silver (gr. v. to ℥j) once a day when the coryza is troublesome.

Of the remedies employed in the treatment of the malignant form of Scarlet Fever, there is one which stands out from among the rest, the cold affusion. Yet it is not of equal value in all cases; from the days of Currie downwards the ataxic form of the disease, characterized by delirium, diarrhœa, vomiting, full pulse, and great heat of skin, has been recognized as the special indication for this active treatment. The patient is to be seated naked in a bath, two or three bucketfuls of water at 70° F. are poured over him quickly, so that the affusion does not last longer than half a minute; he is then returned undried into bed, and laid between blankets. The first affusion having had a markedly beneficial effect, should the indication symptoms return in the course of the same day or the next, the water treatment may be repeated, and this even two or three times if necessary. When this treatment has been objected to, or has seemed too bold, I have seen very good results follow from packing the patient in a wet sheet for an hour. A still milder method remains to be mentioned, that of occasional cold sponging. Ammonia and brandy are nearly always needed, sooner or later, by these patients.

[Cold sponging is often very useful in non-malignant, *open* cases, with high temperature and inflammatory eruption. It may be practised more than once a day; but is especially serviceable for the promotion of sleep at night. Some practitioners prefer inunction with lard, or the application of glycerin, when the irritation of the skin is great.—H.]

In the primary adynamic form all treatment will be baffled. The cold affusion is the only means which has seemed to me to be of even momentary benefit. Hot mustard baths, which would at first sight appear to fulfil the indication better, may be tried; strong tea or coffee, brandy, ether, camphor, are to be given internally. The treatment is much the same, and also, unhappily, the prognosis, in adynamia, sequential to an excited state. A full description of the treatment formerly adopted (and not, perhaps, without success) in cases such as these, will be found in the pages of Armstrong.

[Certain cases of a dangerous kind, attended by stupor, without reduction of temperature or marked feebleness of pulse, are best treated with purgatives and diuretics. A good combination for this purpose is that of jalap, digitalis, and squills. —H.]

In the more prolonged adynamia, quinine is a useful addition to the remedies just mentioned. A most nutritious diet and a rather liberal allowance of wine will often enable such patients to recover.

The Hippocratic remedy of warm affusion to the head is most soothing in the nocturnal delirium and sleeplessness.

However favorable an attack of Scarlet Fever, the patient should be kept in bed for three weeks from the commencement of the disease; he may then get up, but he should not leave his room for another week. Even after four weeks have elapsed he is not free (if we are to believe some observers) from all danger of albuminuria.

If on the morning of the fifth or sixth day any ulcerous appearance that the

[1] For fuller particulars refer to Dr. Thudichum's paper. Lancet, Nov. 26, 1864.

fauces may have previously presented does not show signs of yielding, it is well to cauterize the morbid surface. For the tonsils undiluted hydrochloric acid is to be used; for any other part of the soft palate, solid nitrate of silver. The latter caustic is to be applied to those excoriations which are apt to appear about this time or later. It need hardly be added that these potent escharotics are, as a rule, to be used once for all; if repeated four or five days at least should have elapsed since the previous application. The external swelling should be assiduously fomented while in the puffy stage, for as soon as brawniness sets in, anything that may be done will be of very small service; poultices are to be continued, but the occurrence of suppuration will hardly be thereby hastened. As soon as suppuration has occurred, however small the spot to which the process is limited, an incision should be made to let out the pus; the poultices being afterwards resumed. The abscess should never be allowed to open itself. In the worst cases of Scarlatina anginosa, openings and counter-openings will be required; a free discharge of sloughs and ichor affords the patient his sole chance. Should hemorrhage occur, the wound is to be stuffed with lint soaked in the solution of the perchloride of iron; this moderate pressure will stop the bleeding, which is more often venous than arterial.

All cases of otorrhœa are to be treated by syringing the meatus gently with warm water three or four times a day. Should a discharge either from the ear or from the nose become chronic, quinine and sulphuric acid are the drugs indicated.

The suppurative tendency likewise is an indication for quinine, and an abundance of fresh air and substantial food. All abscesses are to be opened early. A post-pharyngeal abscess should be evacuated if possible (and it mostly is possible) through the neck; should this be impracticable, a cut must be made with a guarded bistoury through the posterior wall of the pharynx upon the vertebral column—an operation devoid of risk, if performed with ordinary care. The matting together of tendons is remediable to a great extent by fomentations, friction, and passive motion.

The treatment of the rheumatism is that of its symptoms: Dover's powder for the pains, diluents for concentrated urine, aperients if necessary, cotton-wool or poultices round the affected joints. Alkalies are not of any special value. The treatment of intercurrent pericarditis is often difficult; whether local blood-letting is admissible, and when counter-irritation more or less severe should be used, depend entirely upon circumstances beyond the local inflammation.

The remedies to be employed at the onset of renal dropsy may be thus arranged in order of importance. First come active purgatives; elaterium is good, but the uncertainty of the ordinary drug often causes the loss of much valuable time; compound jalap powder in doses of not less than a scruple to a child of six or eight years of age, repeated at intervals of eight hours, until the bowels act freely, is altogether more trustworthy. The hot-air bath, preceded by a hot-water bath and a dose of antimonial wine, may be used every night so long as the quantity of the urine is much diminished. After four or five such baths the strength of the patient should be carefully estimated, and the baths continued or not accordingly. Dry-cupping, not practicable in the case of children, would no doubt deserve trial in older patients. Vomiting is an intercurrent symptom which will perplex the practitioner; not the least valuable remedy will be found to consist of ice swallowed in the lump. Diluents should be given as much as possible. It may as well be observed that there is a concentration of the urine which is due to insufficient ingestion of liquids or to excessive sweating; to drink water freely is all that is needed in such cases to increase the quantity of urine.

[*Lemonade* is not only an agreeable, but a useful diluent in such cases. Among diuretics, digitalis is most valued by some practitioners. The late Dr. L. Gebhard, of Philadelphia, after a very extended experience, considered it one of the best of remedies in Scarlet Fever. Several practitioners have reported well of the use of quinine in considerable doses, in scarlatinal dropsy.—H.]

The less acute stage of albuminuria (called the second in the previous pages) requires an altogether different plan of treatment. The bowels must not be confined, but purgation is no longer necessary. Hot-air baths have little or no beneficial effect upon the condition of the urine. When there is much hæmaturia, gallic acid in sufficient doses may be tried; it should be discontinued if it does not bring about a decided improvement in four or five days; to diminish the quantity of albumen as such, gallic acid has little if any power. Quinine has at times a remarkably good effect in these chronic cases. But upon the whole no remedy is equal in value to the perchloride of iron. Counter-irritation to the loins will be useful, and generous diet necessary. In the great majority of cases the disease tends to wear itself out in time. When extensive anasarca is present, the whole condition is more grave; should the dropsy tend to the lungs and pleural sacs, the danger can hardly be exaggerated. We must try laxatives and diuretics so long

as the anasarca remains simple. The hot-air bath may be used cautiously, but there comes a time when it will increase the anasarca rather than diminish it. When acute hydrothorax or pleurisy with effusion occurs, the question of paracentesis presents itself, but is rejected not only by experience, but also on *à priori* grounds; both sides are involved at once, and the lungs mostly suffer as much as the pleuræ.

Sloughing of the cornea would probably be preventable in many cases by the simple expedient of keeping the eyes shut, as recommended first by Trousseau.

DENGUE, OR DANDY FEVER.

By William Aitken, M.D.

Definition.—A febrile affection, *sui generis*, commencing suddenly, and associated from the commencement with severe pains in the large and small joints. About the third day a peculiar cutaneous eruption or efflorescence appears upon the palms of the hands, rapidly spreads over the whole body, and rarely continues visible beyond twenty-four hours. A distinct remission succeeds, but relapses are numerous, and the disease may thus persist about two months marked by prostration and cachexia, its course being characterized by intervals, or remissions, and the exacerbations marked by rheumatic or neuralgic-like phenomena.

Synonyms.—Scarlatina Rheumatica, Cock; Exanthesis Rosalia Arthrodynia, Cock; Dandy Fever, Natives of West Indies; Dunga Bouquet; Bucket; Epidemic Inflammatory Fever of Calcutta, Mellis; Eruptive Epidemic Fever of India; Toohutia, Natives of East Indies; Three-day Fever, Natives of East Indies; Rheumatic Fever with Gastric Irritation and Eruption, Furlonge; Eruptive Articular Fever; Eruptive Rheumatic Fever; Plantaria; Febris Exanthematica Articularis; Giraffe, on account of the stiff holding of the neck; Epidemic Anomalous Disease, Stedman; Peculiar Epidemic Fever; Colorado, on account of the red spots; Exanthesis Arthrosia; Stiff-necked Fever; Broken-wing Fever; Break-bone Fever.

History.—During many months previous to October, 1824, there prevailed in Calcutta an epidemic fever so impartial in its attack that few remained untouched by the distemper in a population of nearly half a million of beings. Passing from the East Indies the disease was next heard of in 1827-8, amongst the islands of the Archipelago and in the Southern States of North America.

Between the months of September and January of these years it prevailed in the islands of St. Thomas and Santa Cruz. Almost every individual in a population of 12,000 persons is reported to have suffered. (Stedman, Ed. Med. and Surg. Journ. Oct. 1828.) With few exceptions the disease spared no one of either sex, of any age, or complexion, or caste. The new-born infant, the young child, the aged, the weak, the robust, the rich, the poor, all were alike the objects of attack. Physicians too, invariably became patients, and hence perhaps the details of symptoms in the epidemics of this disease are so extremely minute; and although the disease was a very painful one, it was not one dangerous to life. The attacks were invariably sudden. In families of ten or twelve persons (including servants), a half or even eight members would lie down at once (Furlonge, loc. cit. vol. xxxvii. 1832). Attention was therefore immediately arrested by epidemics of such a disease, and the general public, not less than medical men, were curious to learn the natural history of a disease with febrile characters so peculiar, and in results so unlike the epidemic or endemic fevers of tropical regions with which the physicians of the East and West Indies had been familiar. When the disease first attracted attention in Calcutta, it was generally believed that nothing of precisely the same nature had ever existed there before, unless the disease known to the native "conductors" of India by the name of "the Three-day Fever" were of the same nature. (Cavell, Trans. Phys. Soc. of Calcutta, vols. i. and ii.)

For the earliest accounts of this disease we are indebted to the physicians of the East Indies, and especially to Drs. James Mellis, Kennedy, Twining, Cavell, and J. Mouat; and the natural history of the fever given by them was subsequently corroborated, in all essential particulars, by the physicians of the Southern States of

North America and those of the West Indian islands, more especially by Drs. Stedman, Cock, and Furlonge, in these islands, and by Dr. Dickson, of Charleston, in America. [Dr. Benjamin Rush first described it as occurring in Philadelphia in 1780.—H.] When the disease was first described by Dr. Mellis, he was disposed to regard it as "inflammatory fever;" but he found that such a name did not fully indicate the peculiar characters of the fever; nor could it be identified as exactly similar to the fever described by Cullen under the name of Synocha; nor to the fever described by Good under the name of Cauma; nor to the Febris acuta sanguinea of Hoffman. Many different opinions have accordingly been entertained regarding the nature of Dengue. By some it was considered as a rheumatic fever; by others as a remittent. Some regarded the complaint as measles; others as scarlatina. Some considered the fever synocha, with gastric irritation and an eruption associated with it, similar to that which in some constitutions follows fish-poisoning. Treatment therefore varied, alike in principle and in detail. But rather than subscribe to a definite name in any then existing nosology, the physicians who first described Dengue preferred comparing the phenomena of the peculiar cases they saw, as regarded their symptoms and sequelæ, with diseases of a similar kind; and so the first records of the history of this disease are peculiarly valuable when the speculations with which they abound are eliminated.

Symptoms. — *Commencement, Development, Duration, Termination.*—The invasion of this disease as a rule was very sudden, and the progress of successive phenomena was rapid. Lassitude, drowsiness, heavy sensations in the eyes, frequent yawning, slight vertigo, a sense of coldness creeping down the back, or of numbness in the extremities which became cold, occasional rigors, pains in the head in most cases confined to the forepart, or most severe there, acute pain in different parts of the body, sometimes in the larger muscles and joints, and occasionally in the smaller ones, such as the fingers and toes, are among the phenomena, one or other of which, or several combined together, suddenly expressed the commencement of the disease; and in the several epidemics whose histories have been recorded, some of these phenomena were more prominently marked than others. As a rule, however, the attacks were so sudden that no sensation of any deviation from the usual health indicated the approach of the malady. It often happened that people had a most violent attack, with severe headache and burning pains in the temples, within three hours after having boasted of their escape from the disorder. (Twining.) In general the patients woke out of their sleep with great pain in the head, loins, shoulders, arms, wrists, hips, thighs, and ankles, fingers or toes. (Mouat.) Acute pains in one or both knees, in the ankles, the wrists, or in all of these joints at once (though in general only one was at first affected), marked so suddenly the invasion of the disease, that the symptoms would first express themselves as the person walked along the street. (Stedman.) The motion of the joint was at first arrested, partly by stiffness, and partly from the pain caused by movement; and in cases where the symptoms commenced in the lower limbs the patient fell to the ground. The most usual mode of attack in the West Indian epidemics was expressed by a sudden stiffness, amounting to pain, in one of the fingers—generally the little finger. The stiffness increased, spreading rapidly over the whole hand and up the arm to the shoulder, so that in a few hours the fingers of both hands became so swollen, so stiff, and so painful, that all attempts to bend the joints were useless. At later periods in the various epidemics the approach of the fever was indicated at least a day before by anorexia, languor, listlessness, and a white tongue. A dry heat at the scrobiculus cordis was among the earliest symptoms noticed by Twining. The face was soon flushed—a phenomenon often observed by others before it attracted the attention of the patient. Intense headache followed upon the burning sensation in the forehead. The eyes became watery and the conjunctiva suffused. The whole countenance appeared bloated and swollen, the face assumed a scarlet hue, and the surface of the skin was everywhere flushed. The signs which then predominated were a chilliness extending over the whole frame, quickly followed by pain and weariness in the limbs, a general sensation of stiffness or soreness, with a heaviness over the eyes so excessive as to render the effort to open them painfully oppressive, and a headache so severe as to be beyond description. (Cavell.) Pressure over the eyes increased the pain, but light did not affect them. The eye-balls appear to the patient as too large for their sockets, as if ready to start from the head, and the pain in them so intense as to cause extreme distress. The expression of the eyes was ferrety. The lobes of the ears were likewise greatly pained. (Cock.)

The rapidity of the pulse, the aspect of the tongue, and the condition of the skin, each of which at first might not seem to be much influenced, yet rapidly passed from their normal condition.

The pulse, soon after the accession of the fever, was in most instances above one hundred per minute, becoming more

frequent, full, hard, and strong, till it averaged about thirty or forty beats per minute above its normal rate, within six hours after the fever became expressed. Twining once observed it 140 in an adult whose usual pulse in health was eighty beats per minute. In some instances its force was so greatly increased that the temporal arteries were felt and seen to beat with violence. In children it was often so extremely rapid as to be indistinct and weak. Respiration was relatively extremely hurried; while determination of blood to the head was considerably increased, as indicated by bleeding at the nose, increasing redness of the face and eyes, confusion of thought, and sometimes delirium.

The tongue soon became of a scarlet color, at the sides, furred with a white or brown coat in the centre, so that in a few hours it appeared as if covered with a dense white paste, or with a thick dirty-white coating, always moist, and associated with a disagreeably bitter taste in the mouth. The bowels were generally confined at first, while oppression at the præcordia, nausea, vomiting of viscid mucus and of bile were present from the commencement, and continued for some time. Irritability of the stomach was often indeed so very great that it retained anything with difficulty. The desire for food was inconstant. In some cases appetite was entirely gone; but not infrequently in children, the desire for food was increased. Thirst was not commensurate with the distress from other symptoms. Extreme prostration of strength became apparent at a very early period of the disease, and with rapid increase of pain in the loins, with not less severe pains in the muscles of the limbs, especially the legs, attended with a remarkable degree of anxiety and jactitation. The febrile anguish was extreme, with aching in the back of the neck. In short, suffering from pain was a leading feature in the accession and course of the attack of Dengue. The debility, the restlessness, and the general soreness rendered every position alike uneasy and intolerable, not less distressing to the patient than alarming to the spectator. (Twining.) Such was the excruciating nature of the pains, that few had fortitude sufficient to support them without complaint. (Cock.) These pains have been known to shift from one part of the extremities to another; sometimes attacking the patient in his knees, or in his toes, sometimes in one knee or toe, and sometimes in another; each new invasion of a part being accompanied with twitchings of the muscles of the part affected. So sudden were the attacks of pain in a fresh place, that a person might be calmly conversing with you when he would suddenly scream out from the severity of returning pain. (Cock.) In some cases the headache and the pain in the back and loins were the most distressing symptoms, although not at all times equally severe during the first twenty-four hours. These pains would frequently subside a little to return in paroxysms with redoubled violence. It was, indeed, a disease of extreme severity as far as relates to the sufferings of the patient; but having regard to the result of the disease, as affecting life, and compared with its universal prevalence, it was a fever of unexampled benignity.

Such were the phenomena which, more or less severely expressed, marked the accession of an attack of Dengue; but towards the end of the first twenty-four hours the symptoms, which had gradually increased in severity, began to abate. The headache and flushing of the face somewhat subsided. The heat became more general and burning all over the extremities, to the relief of the head in some degree. With this remission of the pyrexia, the headache after the second day became still less, and the pains in the loins and other parts were attended with less jactitation; the character of the pain was changed to that of a dull aching kind, gradually leaving the fingers and ankles, and the toes last of all. During the first two nights there was little or no sleep, in consequence of the pain and febrile anguish; and although for several succeeding nights sleep was in most cases still interrupted by thirst and pains in the loins and legs, extending down to the toes and fingers, yet there was little or no disturbance of the intellect. Great prostration of strength; general debility of the whole system; weakness of the stomach, of the loins, of the limbs, of the knees, and of the joints generally; continued pain in several joints, large and small, sometimes limited to a finger only; œdematous swelling of the extremities; and general cachexia—denoted the exhausting and debilitating nature of the malady. The debility was so great that sudden death during the period of remission or of spurious convalescence was known to occur in some instances during the epidemic in Calcutta. The debility was not diminished by the sudden occurrence of perspiration, which during the early hours of the febrile accession had been suppressed, and, although this return of perspiration was accompanied by warmth of the feet and a remission of all the more distressing symptoms, yet the prostration of strength was all the more apparent. The urine was now copious and pale-colored; and the evacuations from the bowels, even if freely moved by remedies, were of a dark green color, or even black, glutinous, scanty, and always offensive. During the three days following this re-

mission, the patient, in the more severe cases, lay in a state of extreme languor, but irritable and restless; but as there are no records of temperature in this disease, it is not known if fever was entirely absent during the remission, although it is sometimes stated to have been so. It is rather to be presumed that the temperature did not fall to the standard of health; for although there was no acute suffering yet the feelings were said to be very different from those of health. Thirst continued to prevail in some, and the desire for food did not return. The sense of taste seemed entirely lost; while in some cases little aphthous sores, on the inside of the lips and on the edge of the tongue, rendered attempts at eating painful in the extreme. (Stedman.) About the end of the third day the febrile phenomena again expressed themselves with even increased severity, compelling the patient to return to bed if he had ventured to leave it, deceived by the apparent but treacherous and spurious convalescence. With this febrile exacerbation, the cutaneous system exhibited remarkable and peculiar phenomena. With great turgidity of the skin an eruption or rash appeared on different parts of the body. The appearance of this rash has been variously described:

(*a*) As an efflorescence, beginning at the palms of the hand, and gradually spreading over the whole body. In general appearance this efflorescence differed considerably in different cases. In some it consisted of blotches of red-colored skin, resembling in appearance, something between measles and scarlatina. In some it was raised perceptibly above the surface of the skin, imparting a distinct feeling of roughness and elevation to the fingers passed over the eruption. In severer cases more obvious local swellings accompanied the efflorescence, and a distressing tingling of the skin resolved itself into an itching so intense, while the eruption disappeared, that the sufferers were almost driven to distraction. As a rule this efflorescent eruption remained only for about a single day, beginning to fade on the second day, and before the third morning it was generally entirely gone. Some degree of desquamation followed, generally in proportion to the intensity of the eruption. One instance is on record, in which a man eighty years of age had the eruption in so severe a form that the cuticle came off in flakes like pieces of parchment, leaving the surface of the body quite red. (Stedman.) The itching was extremely distressing, and the patient was in the utmost misery. In old men, the desquamation from the scrotum was attended with most intolerable itching, and in some of them the testicle became swollen to a great degree. In others, extensive abscesses formed beneath the skin, probably of the nature of the pyogenic fever of Tessier and Jenner, and popularly regarded as "the dregs of the fever." In the epidemic at St. Thomas's, two children, each about five or six months old, died from the irritation attending the desquamation of the cuticle. The true skin, so exposed, was red, raw, and exceedingly painful from the inflammation of its texture.

(*b*) About the third or fourth day an erythematous eruption appeared on the hands and feet (Cock), accompanied with swelling of those parts. This eruption gradually extended over the rest of the body, continuing for about thirty-six hours, when it faded, and the cuticle peeled off as in scarlet fever, leaving a considerable degree of soreness. The soles of the feet were sometimes rendered so sore, that walking, for many days after restoration to health, was attended with pain. The pains were apt to remain after the eruption had completely disappeared, and to become fixed in one or more of the joints, where they would remain for several months with morning and evening exacerbations; the pains then were most severe. Sometimes the glands in the groins were swollen and painful. (Cock.)

(*c*) Dr. Furlonge regarded the eruption as altogether symptomatic of the gastric disturbance. He observed that its intensity and extent were proportional to that disturbance; that those who were "known dyspeptics" had the eruption more extensively than others; that literary or sedentary people and those whose occupations were such as to derange the chylopoetic organs, suffered most from nausea and other symptoms of gastric disorder, and in them the eruption was always more extensive and marked. This eruption, he thought, resembled measles elevated with papulæ and wheals; a sort of hybrid between urticaria and rubeola, and of its nature similar to that which is known to attend the gastric disturbance from fish-poisoning. (Ed. Med. and Surg. Journ., p. 52, 1830.)

(*d*) Dr. Mellis regarded the eruption as similar to roseola or to the lichen simplex of Willan.

(*e*) Dr. Twining regarded it as resembling rubeola; while in some instances it resembled patches of an inflammatory appearance, in others a papillary, and in one or two instances that of a vesicular eruption. In many instances little more than a flush attended the febrile excitement.

(*f*) Mouat says of the eruption, that it is like erythema papulatum, or purpura simplex not disappearing on pressure, and resembling roseola miliaris or lichen tropicus.

(*g*) In some cases there was said to have been no eruption (Cavell); but when

the short persistence of the eruption is taken into account, it is not improbable that it may have been overlooked in such cases. In most cases indeed discoloration of the skin was evident from the first; and it seems to have been characteristic of Dengue that the eruption which attended it assumed many characters. In one case the eruption might be referred to the class papula, in a second to exanthemata, in a third to vesicula, in a fourth to bullæ, and in a fifth to that of wheal. But whatever the form of the eruption, all the cases were attended during its continuance with very high excitement of the vascular system, which at last suddenly subsided, leaving the patient convalescent. The eruption came on suddenly and vanished suddenly, hence it may have been very often overlooked, or its characters might not have been observed, for it does not seem to have gone through any definite changes or marked stages. In some cases it appeared simultaneously with the febrile symptoms, in others not till twenty-four or thirty hours afterwards, and in two or three instances after all fever had apparently subsided. Whenever it appeared the color of the eruption was similar, and the amount of the surface covered by the rash of various extent. In proportion to its early appearance it generally portended a further mitigation of the more distressing symptoms, and more especially if the efflorescence uniformly covered the whole body and the extremities. When the eruption was only partial on the body and less on the extremities, but increased about the chest, neck, and face, there was, for the most part, some increase of feverishness, more uneasiness and anxiety, with aggravation of headache. It remained persistent for a comparatively short time, and had for the most part considerably faded at the end of twenty-four hours after its first appearance, although it might continue obvious for two days. It was always attended with some degree of heat or itchiness, particularly when the minute exfoliations of the cuticle began to separate during the period of desquamation. It seemed to be chiefly in cases where the eruption remained persistent beyond two days that it assumed the aspect of urticaria, affecting more particularly the extremities; the fingers and toes, hands and feet being swollen, red, and affected with distressing itching and burning, combined with febrile exacerbations and extreme debility for twenty-four or thirty-six hours. Associated with the eruption, some patients had boils; others had small acuminated vesicles with hardened bases. Children suffered most from urticaria, and in them large watery vesicles sometimes led to the formation of ulcers. In one case the sloughing was so deep, the ulcers so foul, and the fever so high, that, convulsions supervening, the case terminated by death. In most instances, if not in all, as in the epidemic at Suzuratte, the throat and fauces were so affected as to make deglutition painful. The secretions from the lungs and salivary glands were very copious, unhealthy in appearance, and distressing to the patients. The salivary glands were in some much swollen (the parotid as early as the fourth day), and the discharge of saliva in some instances amounted to ptyalism, although mercury had not been taken. It was also observed (Mouat) that very small doses of calomel frequently induced disagreeable ptyalism.

The disease was not considered to leave the patient till he had suffered from a second and even a third relapse or paroxysm of fever. The relapses were each at one time considered as separate attacks of fever; but the history of subsequent epidemics showed that these several paroxysms, each nearly equal in severity (although it was not noted where the eruption repeated itself), combined to constitute one and the same attack of a fever marked by such peculiar remissions and exacerbations. There were also daily remissions observed by Dr. Mouat, there being two or even three paroxysms in twenty-four hours. Few recovered, under three months, from the debilitating effects of the attendant fever and the aching pains in the wrists, fingers, toes, and ankles. Tardy recovery, and the tendency to repeated relapses, were characteristic phenomena. In these periods of exacerbation the third days were decidedly "critical" (Kennedy); the pains in the limbs would abate, and the capability of taking food would return; but the furred tongue, the foul taste, and the disordered stomach generally continued for ten or twelve days after the final remission. The secondary prostration of strength evinced itself in proportion to the constitutional powers of the patient. Females in various periods of pregnancy went through the severer forms of the fever without any tendency to abortion.

Although the actual temperature of the body in cases of Dengue has not yet been recorded, there is reason to believe that the fever reaches its fastigium at a very early period—probably within three days; and during this period the surface of the body is subject to an irregular distribution of the blood, so that while the hands and feet are cold, the rest of the body, and particularly the head, may be intensely hot. During the height of the fever the head and eyes seem to indicate the greatest suffering, but the whole body is racked with pain, especially in the joints. Not an inch of the body from head to foot seems to have been exempt from suffering; hence the American name of "break-

bone fever." In some cases the features, especially the eyelids, were swollen and distorted, associated in one or two instances with profuse ptyalism. But although the skin in cases of Dengue felt intensely hot to the touch of the observer, yet the patient experienced feelings of intense cold during all the different stages of the disease. When it prevailed as an epidemic at St. Thomas's, although the weather was extremely sultry, yet the patient felt the warmest coverings (of two or three blankets) scarcely enough.

ETIOLOGY AND PROPAGATION.—The origin of Dengue is unknown. The disease is said to have been first noticed in Rangoon about the end of May or beginning of June, 1824; and on the 10th of June a large portion of the troops employed on the expedition under Sir Archibald Campbell, then at Rangoon, had been ordered out to attack the Burmese, and were thus exposed to incessant heavy rains for four-and-twenty hours.

The disease extended in various directions, not only to Calcutta, but to Chittagong in the southeastern extremity of the Province of Bengal, and to Guzerat, in the Presidency of Madras. It was particularly severe in the large and populous towns of Benares, Patna, and Chunarghur, and prevailed generally from Buxar to Benares, Churnar, and Mirzapore.

In all of these places it seems to have become epidemic during or subsequent to the existence of heavy rains, associated with the close cloudy heat of sultry weather. Heat, moisture, and stagnation of air seem to have been always associated with the origin and transmission of the disease.

Stedman implies that the disease was imported into the free port of St. Thomas's, and the accounts of the disease generally show that it was propagated from place to place in the course or route of human intercourse. It travelled from Barbadoes to Jamaica. It raged in America and the East Indies. No disease, indeed, with the exception of influenza, ever had so wide a diffusion. From St. Thomas's it proceeded upwards to Barbadoes. It seemed by the mode of attack as if something were applied which in a moment had the power of destroying the balance of health, and of producing a disease, the symptoms of which were so appalling at first sight.

Protracted debility, with long-continued pains in the limbs, were the invariable sequelæ of the fever. In several instances, tedious visceral disease, mainly of a subacute form, especially of the liver, with jaundice, were associated with the protracted duration of fever. Hemorrhoidal affections sometimes preceded an attack of subacute hepatitis with slight jaundice. Returns of pains in the extreme joints of the fingers, distension of the abdomen, anorexia, slight tenderness of the belly, and thirst, betokened the advent of incipient visceral disease.

In a few cases the eyes were affected with ophthalmia. (Mouat.)

Partial anchylosis is known to have occurred in the fingers of the hand. (Cock.)

DIAGNOSIS.—While the severe pains, on the one hand, caused the disease in some instances to be regarded as rheumatic, the paroxysms of the febrile attack and the intercurrent remissions, on the other hand, caused the disease to be considered as one of a malarious nature. But there was to be noticed the peculiar eruption already described, which, combined with the other characteristic phenomena, at once stamped the disease as one *sui generis*. The suddenness of the attack, the redness and the watering of the eyes, the acute pain in all the joints, rendered excruciating on the slightest touch, the scarlet or crimson efflorescence on the surface, its ephemeral duration, the disease sparing neither age, sex, nor habit of body, its seizing the acclimatized as well as those recently arrived, stamp Dengue at once as a disease different from the endemic remittent fevers of the countries where it has prevailed. The diagnostic differences from measles and scarlatina must be studied in connection with the natural history of those diseases. Dengue attacked indiscriminately those who had suffered from scarlatina and those who had not. (Cavell.)

TREATMENT.—Amongst the natives of India, Dr. Mouat had convincing proof that the disease was protracted and severe when no remedies were used. Prostration and severity were evinced in the great emaciation, the more extreme debility, the severer pains and the swollen extremities and the longer duration of the disease, in those cases left to run their own course.

Experience has proved that emetics and free eliminative remedies (especially purgation) insure an early freedom from fever. Time is no doubt an essential element in relation to the cure of the disease, which seems to run a specific course, and may be aided or retarded by remedies.

These remedies have been on the one hand (1) bleeding, on the other hand (2) purgation and eliminative medicines.

Twining did not resort to general bloodletting, but in several of the earlier cases in which the head was much effected, he sometimes applied a considerable number of leeches to the temples. A further observation of the progress of the epidemic and nature of the fever convinced him, however, that even leeches were by no

means necessary, "as other cases with symptoms parallel in nature and severity were as speedily remedied without leeches." He considered that general bleeding was not adapted to the treatment of this fever. Cold affusion he also considered a hazardous remedy. The experience of Cavell also proved that depletion did not afford the relief expected of it. So also, Mouat records that bleeding did not cut short the disease, nor mitigate the symptoms. It added to the exhaustion of the patient; it impaired the powers of digestion; it induced vertigo during convalescence, with a tendency to fainting, distressing dreams, and bad nights. It was only of use in cases of local affection of the lungs, liver, or intestines.

With reference to purgation, Twinning's experience showed that a moderate dose of calomel, combined with an active dose of colocynth and scammony, and repeated every twenty-four hours, *till the evacuations were free, and of a more natural and healthy color*, were the remedial agents he observed to be followed by the best effects. The use of purgatives in this way was indicated by the stools being of a dark-green color; and with the disappearance of greenness from the stools, the symptoms were ameliorated. Calomel is never to be given alone. It is probable that calumba, rhubarb, and soda, combined in equal proportions, will equally answer as an alterative remedy. The eliminative action of purgation is to be obtained without the result of watery purgation; and so far as the use of purgatives is concerned, the only objection that is urged against them is the aggravation of the pain which they cause by the motion which their action entails. But the skill of the nurse and the physician must be combined to obviate this very secondary consideration. Eliminative remedies in an opposite direction were also shown to be of use. Emetics of tartar emetic and of ipecacuanha discharged large quantities of bile, relieving the pains of the head and of the limbs almost immediately. An emetic given at first always relieved the head and eased the pains; and this evacuation, followed by purgation, or even an open state of the bowels, tended greatly to facilitate recovery. A free, open state of the bowels is then best preserved by a dose (every two hours) of tartar emetic and sulphate of magnesia; or a compound of jalap powder or of sulphate of magnesia with infusion of senna. (Mouat.)

In thirty-six hours after the action of such remedies was obtained the fever was subdued with less prostration of strength than by any other treatment; but if no bile followed the emetic, the symptoms were not relieved, and it was necessary to repeat it till the bile was discharged.

In the cases where ophthalmia was a consequence, leeches applied to the inner membrane of the eyelids sufficed for the cure, with free and repeated purgation.

After the bowels were freely opened, a light febrifuge mixture of sweet spirits of nitre, nitrate of potass, tartarized antimony, and colchicum, was attended with benefit, given every two hours, with an occasional effervescing draught, a pediluvium at bedtime, and ten to fifteen grains of Dover's powder. After all the acute symptoms had subsided, forty to sixty drops of the wine of colchicum, with twenty-five drops of laudanum, always insured a good night's rest, and thirty drops of antimonial wine were now and then added to the draught. (Furlonge.) Tonics (such as infusion of calumba, quinine, iron, or strychnia) ought to follow the free action of the bowels. Cordials, stimulants, and good diet, consistent with the habits of the individual patient, must also be thought of, and remedies of a local kind to allay the itchiness of the skin may also be of service; such as emulsions of almonds, with hydrochlorate of ammonia and corrosive sublimate judiciously combined and carefully applied.

ROSEOLA.

By Hermann Beigel, M.D., L.R.C.P. Lond.

Definition.—Roseola is a disease characterized by the appearance on the skin of spots, separate from each other, of a roseate, scarlet, or dusky red hue; of minute size, like marks made by the point of a pin; but which may exist in such large numbers and so close together as to form large patches, of most varied shapes. These are not raised above the surface of the skin; are not communicated by con-

tagion; and are generally unaccompanied by fever.

Willan is known to have described seven forms of Roseola, which number has been raised to twelve by Bateman, Rayer, and Wilson. Whoever wishes to take the form and hue of an irritation of the skin or any other part of the organism as a basis for classification, can, with great facility, introduce hundreds of new species into medical science. But whether science would gain anything by our calling a pneumonia, in which the inflamed part of the lung is round, pneumonia circularis, or if triangular, pneumonia triangularis, is another question. Quite the same may be said in reference to roseola annulata and punctata; and if the words æstiva and autumnalis express anything, which may be taken as a basis for division, we might just as well find in the 365 days of the year material for a division into the same number of forms, not only of Roseola, but of any other disease. Classifications of this kind, which neither define correctly nor are of any utility, ought to be banished from medical science. Those who know the former unpractical, incorrect, and circumstantial classification of ulcers, and compare it with that of the present time, simplified and based upon correct observations—admitting what influence nomenclature has on treatment—will acknowledge the necessity of a judicious division in other spheres of medical science, and particularly in dermatology.

We may observe Roseola in the course of different, mostly feverish, diseases (Roseola symptomatica), or independent of diseases (Roseola idiopathica). The Symptomatic Roseola is a part of the disease in the course of which it occurs, and is therefore excluded from our consideration. We have now only to speak of Idiopathic Roseola, which rarely requires medical interference, but which gives us an important hint in reference to the pathology of skin-diseases in general.

Cause.—Anything which weakens or lowers the action of the nervous system, and this for a long time, may cause Roseola. Severe illnesses—such as typhus or syphilis—which change the vitality of the blood and nerves; bad nourishment, and general debility, are the common causes of R. symptomatica.

Idiopathic Roseola is brought about in a similar manner, but the action of the nervous system is weakened for a short time only. Not only errors of diet, but some particular kinds of food may produce Roseola. I know a lady who has a regular attack of Roseola, of seven to eight hours' duration, after eating strawberries, and this without any other disturbance; another lady who can voluntarily produce Roseola by getting hot through dancing or other exertion and by drinking water afterwards. The eruption constantly appears in about half an hour, causes no other disturbance, and disappears after several hours. I attended a child who was troubled by hooping-cough. The milder attacks passed as usual, but the violent ones, during which the whole body of the little patient took a dusky hue, always brought on Roseola, which after a few minutes disappeared.

That sudden changes of temperature may produce Roseola is well known; and its appearance during dentition is remarkable. Sometimes it comes and goes so quickly, that a child, while teething, may exhibit five or six attacks during the course of one day. These attacks are usually unaccompanied by any pain. At the first appearance of the eruption, an anxious mother may send for the physician; but, so transient is its duration, that often, when the doctor comes, there is nothing to be seen. I am attending a family of two little boys and two daughters, and both of the boys have exhibited the phenomena of Roseola to a very high degree. The boys, who are strong and healthy, exhibit it at the commencement of every trifling indisposition, and this quite independently of dentition. The girls, who are twin-sisters, have not suffered. It is not rare, however, for Roseola, in some women, to precede every menstrual period.

It would appear, therefore, that Roseola depends immediately upon changes in the vitality of the blood and nerves; and we may be tolerably sure that if another Willan—endowed with Jussieu's capacity for observing nature—should appear, we should find Roseola, and many other skin diseases, ranging among the diseases of the nervous system.

Course.—Idiopathic Roseola appears almost feverless; yet feverish symptoms may exist, although they never reach a high degree. Headache, excited pulse, constipation, want of appetite, disturbed sleep, itching may occur, but their occurrence is rather the exception than the rule. In the majority of cases Roseola idiopathica occurs suddenly, over a large surface, and without marked forerunners; it remains a few hours or days, and either disappears as suddenly as it came on, or turns into a more dusky hue, becomes at length pale, and a fortnight may expire before the last spots vanish. But it may happen that the rash disappears on one part of the body while new spots make their appearance on another.

In other cases a few little red dots may occasionally be discovered and increase in number until the body is partially or totally covered with them. I never saw slowly developed Roseola disappear sud-

denly; but slowly as it came. The mucous membranes may participate in the eruption, but this is mostly observed in dentition, when these membranes are already affected.

That the same individual may repeatedly be subject to the eruption, is evident from the above statement.

Diagnosis.—Roseola bears the greatest resemblance to flea-bites, and to those partial inflammations of the skin which are caused by the stings of other insects. But bites and stings are distinguished from Roseola by the black central spot which, in an early stage, is clearly seen in almost all of them. Erythema Roseola differs in the fact that its confluent spots are raised above the level of the healthy skin. With measles and scarlet fever Roseola cannot be easily confounded; for beside the symptoms proper to those exanthemata, the skin, in the latter, is in a state of turgescence, and forms a red basis for the eruption, whereas in Roseola the skin between the spots is healthy. Its extremely mild course, independence of epidemic influences and character, and the absence of catarrhal phenomena, are sufficient to distinguish Roseola from measles.

Prognosis.—Idiopathic Roseola is a slight affection which never reaches a degree causing anxiety. Even in those cases where Roseola occurring during dentition is accompanied with fever, we scarcely have a right to refer the latter to the eruption, as it forms a symptom, which often occurs in dentition without being followed by Roseola or any other eruption.

Treatment.—Cases in which Roseola idiopathica requires medical interference seldom occur. The eruption comes and disappears without any disturbance of the general health. But if there exists a local irritation which keeps up the eruption or causes its recurrence, then, of course, it must be removed. Ascarides, which often cause Roseola, must be expelled; gentle laxatives will be applied against constipation, and impaired appetite improved by carbonized water, soda, seltzer water, and by dilute mineral acids, particularly phosphoric acid. If during dentition the necessity arises to lance the gums, it will not be Roseola which necessitates this proceeding.

MEASLES.[1]

By Sydney Ringer, M.D.

Definition.—An acute febrile contagious disease, mostly occurring in epidemics. It generally attacks the patient but once, but sometimes again occurs after the interval of a few months, or many years. Relapses are very rarely met with. They may commence immediately or three or four days after the fever of the first attack has declined.

Generally coexisting with epidemics of other diseases, it is especially related to hooping-cough. The one disease apparently predisposes to the other. Thus often an epidemic of Measles precedes one of hooping-cough, or *vice versâ*. In the latter case, the paroxysmal cough may continue, or temporarily disappear during the existence of the Measles. It is stated that persons with pulmonary disease, such as bronchitis, are especially apt to catch the disease.

The period of incubation, in those cases in which the disease was produced by inoculation, was seven days.

Synonyms.—Measles; Flecken; Masern; Morbilli; Rubeola; Rougeole.

Symptoms.—Measles is generally abrupt in its commencement, and is then ushered in by chilliness which may amount to rigors, or not uncommonly, in children, to convulsions; or, on the contrary, the invasion is so insidious that it is impossible to determine with accuracy the first day of attack. The disease, when established, is accompanied with a variable amount of prostration, but which is usu-

[1] For much of the information contained in this article, the author is indebted to Willan's Miscellaneous Works, Dr. Armstrong's Practical Illustrations of Scarlet Fever, Measles, &c.; Trousseau's Clinical Lectures, Graves's Clinical Lectures, Hebra, Rilliet et Barthez's Maladies des Enfants, Journal für Kinderkrankheiten, Schmidt's Jahrbücher, Canstatt's Jahresbericht, Aitken's Practice of Medicine, Parkes on the Urine.

ally not extreme, and sometimes amounts only to a feeling of lassitude. The patients take voluntarily to bed, and are indisposed to either physical or mental exertion. They are fretful and irritable, in some cases only when disturbed; in others they are constantly restless, whining, and peevish—differences dependent on peculiarities inherited, or developed by bad education. The expression is vacant, and the powers of perception and reflection are much impaired. Delirium is in some cases present, always slight and usually limited to the night. The skin is hot and dry; the lips are parched, and in severe cases covered with sordes; the tongue, thickly coated, is mostly moist, and a few red papillæ may be often observed to project through the thick coating of fur. The appetite is much impaired, and the thirst often extreme. Vomiting not infrequent at the commencement, may be repeated and persistent, and then indicates a severe attack of the disease.

The bowels, in some cases confined throughout, are often relaxed, and are generally easily influenced by medicine. Diarrhœa not unfrequently first occurs at the period of the eruption. Usually from the very commencement the mucous membrane of eyes, nose, mouth, and respiratory tract suffers changes. The conjunctivæ are injected, and the eyes suffused with tears. There is generally some intolerance of light, and occasionally the patient complains of a sensation as of sand beneath the lids; the eyelids are swollen and red at the edges; there is often repeated sneezing, with at first a thin watery discharge from the nose; epistaxis not infrequently accompanies the coryza at its commencement. The mucous membrane of the mouth and throat is mottled with redness, and a feeling of weight and tension over the frontal sinuses may be complained of. There is generally some soreness of the throat, but without much swelling of that part. The cough is dry, hacking, and frequent, and there is a feeling of weight and oppression at the chest, at which part wheezing and rhonchal fremitus may often be felt. The voice is often hoarse; the respirations are hurried and shallow: the pulse is increased in frequency, but loses in force. The urine is scanty, and deposits an abundance of lithates on cooling. In rare instances severe pain in the abdomen has been noticed, with or without diarrhœa; pain and tenderness have been sufficient to lead to the idea of peritonitis, but they entirely disappear on the resolution of the rash.

Usually on the fourth day from the commencement of the disease the characteristic rash appears. First noticed on the forehead close to the scalp and on the chin, it from thence spreads over the face, trunk, and extremities, in the accomplishment of which it occupies a period varying from a few hours to two days. On the appearance of the rash the fever is stated to increase; it certainly does not diminish. The lachrymation of the eyes and injection of the conjunctivæ become more marked. There is slight swelling of the whole surface of the body (if the rash be abundant and general); this is especially noticeable in the face, and causes a variable amount of alteration of the features, so that in some cases the patient can scarcely be recognized. The feet and hands at the same time feel full and tense; deafness may be present, being due to the swelling of the mucous membrane of the Eustachian tube.

The cough at this time generally increases in severity, and is occasionally rather paroxysmal.

Examination of the lungs reveals sonorous, sibilant, and even a small amount of submucous rhonchus, the latter being most abundant at the bases of the lungs.

After the second or third day of the eruption the fever disappears, the temperature becomes normal, the pulse much less frequent, and the patient at once enters on the period of convalescence.

The period elapsing before perfect health is restored varies greatly; in some cases the recovery being rapid, in others prolonged to an indefinite period, this diversity being dependent on the previous state of health of the patient. Thus, if the health has been impaired by excesses of any kind, or by bad hygienic conditions of life; or if the patient be the subject of tuberculosis, scrofulosis, or syphilis, the recovery is often very greatly retarded, and more or less serious sequelæ often result. Further, other conditions being the same, recovery is more speedy in the young than in the old.

Varieties.—The severity of the disease varies greatly, and has thus led to its division into Morbilli mitiores and Morbilli graviores. Occasionally the disease is unaccompanied by any catarrhal symptoms, and is then termed Morbilli sine catarrho. This latter form is said not to protect the patient against another attack. Morbilli mitiores has already been described.

Morbilli graviores, Malignant Measles, or Black Measles, appears to have been far more common formerly than at present; still, cases do now and again present themselves.

Due either to individual peculiarity, or more commonly to a previously depraved state of health, or to epidemic causes, it may begin mildly, or the symptoms may be at once violent and severe. Usually the rash is but imperfectly developed, and partial in its distribution, of a livid, pur-

plish, or even black color, apt to disappear and again appear. There are marked prostration, great tremulousness, twitching of the muscles, delirium low and muttering in character, stupor or partial coma. The lips and teeth are covered with sordes, the tongue becomes dry and brown, the pulse is rapid and feeble, the respirations are hurried, and often the symptoms of congestion of the lungs are present. The extremities are cold, and petechial spots appear on various parts of the body.[1] The urine may contain blood, and bloody effusions are found in the various cavities and organs of the body. Death usually occurs early; should the patient rally, convalescence is much prolonged, and death not infrequently results from diarrhœa, bronchitis, or other of the sequelæ.

Rash.—Usually appearing on the fourth day, in some instances it is noticed as early as the first, or may be postponed to the seventh or eighth day of the disease.[2] Earliest seen on the forehead, close to the scalp, it from thence rapidly spreads to the rest of the face, then to the trunk and lastly to the extremities, in the accomplishment of which it occupies, in some cases but a few hours, but more commonly three days. On the first day chiefly limited to the face, it increases greatly in quantity over the trunk on the second and is most abundant on the extremities on the third day of the eruption.

The development of the rash is best observed on the trunk. There are earliest seen minute red, not elevated points, probably the papillæ of the skin reddened, and but little, if at all, enlarged. These rapidly develop, increase in breadth, and become elevated and acuminate in form, the diameter measuring at this stage about a line. In their further growth they increase chiefly laterally, and thus become flattened. The ultimate size they attain varies, although sometimes not advancing beyond the size mentioned when they are acuminate; they may become as large as a pea: in the former case they can be felt, and seem to be but slightly elevated; in the latter they feel hard and even "shotty," though this is always much less marked than in the rash of smallpox.

The amount of the rash varies greatly. At first much scattered, fresh spots quickly appear on the previously unaffected skin. The spots are generally grouped, and often assume the form of crescentic or irregularly circular patches. Always at first discrete, they may become so numerous, that on enlarging laterally they coalesce and form patches of various size, sometimes sufficiently large to cover the chief part of the chest, or one or other of the extremities. These patches for the most part have their long axis across the trunk, they are always abruptly elevated, and the redness is sharply defined, though generally irregular in outline. Should the rash be very intense, the surface and redness of the patches are quite uniform; but mostly in the redness numerous small papillæ[1] can be seen, and even in the former case, on the decline of the rash, the papillæ become visible.

Mostly abundant on the face, the quantity of rash developed becomes less the lower we descend, and the eruption is often only thinly scattered on the furthest extremities of the body.

The individual parts of the rash have a very short duration. Each spot, usually at its height in twelve hours, has often in

---

[1] Dr. Veit (in Virchow's Archiv, Bd. xiv. Heft. 142; reported in Canstatt, iv. p. 225, 1858) states that petechial eruption is in some cases dangerous, whilst in others the disease runs a favorable course. When serious, he says that the condition is brought about by previous excesses or irregularities of life, and that such cases are met with in all epidemics. He states that if the rash be at its commencement petechial, it is not an unfavorable sign; but that if it subsequently become so, if the rash change from wine-red to black, in form of round spots, extensive patches, or in streaks, then it is of almost fatal significance.

It is certain that not unfrequently small petechial spots may be met with on the upper part of the chest, and occasionally elsewhere, in cases which run a favorable course; but extensive and numerous petechiæ, especially if seated on the legs, are to be looked upon as of very serious import; though less so than if they occur in scarlet fever or smallpox, in which diseases their appearance warrants us in expecting a fatal termination; nevertheless, though this is the rule, cases of extensive petechial rash do occur, which run throughout a favorable course. [During the civil war in the United States of America, quite a considerable number of cases of measles occurred amongst the soldiers, often assuming a typhous or malignant character; the rash being ill developed, or petechial and dark purple in color, with a tendency to prostration throughout the attack. These cases corresponded very nearly with what was formerly called Black Measles.—H.]

[2] Of twenty-eight cases—

In 1,	the rash appeared	in twelve hours.	
2,	on the	1st	day of the disease.
6,	"	2d	"
6,	"	3d	"
10,	"	4th	"
2,	"	5th	"
1,	"	6th	"

The commencement of the disease in these cases was dated from the very earliest symptoms noticed or complained of.

For some of these cases the author is indebted to Mr. Butt, and also to Mr. J. Bartlett, of Notting Hill.

[1] These are much larger than the punctiform elevations seen in scarlet fever.

twenty-four hours again subsided; so that no elevation remains judging by both sight and touch—a mere red mottling being left. The rash declines in the order of its invasion. In rare instances, if the eruption be intense, small, clear, acuminate vesicles may be developed at the summit of the papillæ, or minute petechiæ may occur; these latter, usually not numerous, are perhaps most frequently met with on the neck, upper part of the chest and the bend of the elbow.

Though generally described as of a rose color, the rash appears to the author to be a mixture of red and yellow, the red greatly predominating at the beginning of the eruption; but as the spots enlarge and become flat, the redness declines, and the yellow tint becomes much more apparent.

It is of a brighter and more intensely red tint at those parts of the body which by exposure have become florid. It is thus well marked on the face and the hands. On the palms of the hands and the soles of the feet the papules generally look deeply situated, as if indeed they were placed beneath the cuticle.

By pressure the redness disappears, and a slight yellow discoloration of the skin remains.

After the rash has lost its elevation, a reddish coppery discoloration is left. The redness (which is most marked and remains longest on the face, where the vascularity of the skin is greatest) is always removable by pressure, quickly declines, and leaves a coppery discoloration. This coppery discoloration on the other hand is unaffected by pressure; occurring in spots a few lines in diameter, it is ill-defined in outline; sometimes of a uniform color, but more generally punctiform; is proportionate to the previous intensity of the rash, and is consequently most marked on those parts of the body where the rash is most developed: hence it is well seen on the face, upper part of the trunk, but especially on the shoulders and over the scapular regions. It lasts a variable time, but has mostly disappeared by the tenth or twentieth day from the commencement of the disease.

The rash occasionally departs from the above description, in respect both of its nature and its course. Thus it may appear first on the trunk, and then spread, sometimes involving, at others leaving free, the face; or the face may be the only part affected. In some cases it is very limited, the rest of the surface remaining free from rash. It is apt to appear earliest and to be the most marked in the neighborhood of sores or inflamed parts of the skin.

Sometimes, owing to a serious internal inflammation, mostly of the lungs, the rash suddenly recedes and may entirely disappear.

The desquamation is always slight, and is most marked on the face, or where the rash has been confluent: it is limited to the discolored patches of the skin, and sometimes is only seen when the skin is tightened and viewed sideways; it begins usually on the sixth or eighth day of the disease, and continues eight or ten days.

The scales that are separated are usually very fine, often scarcely visible, unless dropped on a black ground. It is very rare for continuous pieces of the cuticle to be thrown off.

The elevation of the temperature, or fever, varies greatly in degree. The highest temperature reached in ordinary cases is usually 103° Fahr. If the temperature rises above this, it indicates a severe, if it continues below this, a mild attack.

The temperature corresponds, in respect to its daily or other variations, to that of most fevers; thus it cannot assist us to make a correct diagnosis in doubtful cases.

The fever is stated to increase till the eruption and catarrhal symptoms reach their height.

As measured by the temperature, the disease has a very variable duration; in some cases declining on the fourth, or continuing to the eighth or tenth day of the disease.[1]

At the end of the disease, the temperature (*i. e.* fever) suddenly and greatly declines, but still usually rises slightly (to 100° or 101° Fahr.) on the one, and sometimes two days following. After this, it becomes normal, and continues so throughout convalescence; or it sinks for some few days below the point usual in health.[2]

The glands behind the jaw, down the neck, and in the groin become enlarged. The degree of enlargement behind the jaw is regulated by the amount of throat affection; that of the glands in the neck and groins, by the amount of rash present: hence the enlargement is greater in the neck than in the groins. The glands usually resume their proper size from the sixth to the tenth day of the disease.

The enlargement is rarely so great in any of these regions as it is in scarlet

[1] Of 14 cases—

2	ended on the	4th day.
4	"	5th "
4	"	6th "
2	"	7th "
1	"	8th "
1	"	9th "

It is right to state that in many of these cases the rash appeared before the fourth day, in several even on the first or second day, calculating from the day on which the very earliest symptoms appeared.

[2] The temperature in convalescence from all diseases is often below par, is easily depressed by exposure, and moreover is very slow to rise again after it.

fever. In both diseases the enlargement is greater, the amount of irritation being the same in persons whose previous health has been bad, and thus the degree of enlargement serves as an indication for the treatment of the patient.

There may be no affection of the mucous membrane of the eyes, nose, throat, and lungs. Mostly, however, the whole of the tract suffers.

On the conjunctiva, especially of the lids, is seen much fine capilliform and ramiform injection; the former declines as the rash fades, but the latter remains a few days longer.

The suffusion of the eyes usually continues some days after the injection has left, and in some cases may be noticed on the twentieth day of the disease.

Discharge from the nose, at first clear and watery, is one of the earliest symptoms observed. The mucous membrane of the alæ of the nose is red, and not unfrequently excoriated. On the decline of the rash, the inflammation also subsides; the discharge may cease immediately, but more commonly it becomes muco-purulent, and continues for a few days longer. If, however, the child has been badly nourished previous to the fever, or is the subject of the scrofulous diathesis, these symptoms are apt to be much more marked and of longer duration. The alæ of the nose are rather swollen. The mucous membrane, much excoriated, easily bleeds. The discharge is either thin and sanious, or thick and muco-purulent. This accumulates, dries, and blocks up the orifice. The upper lip may become much enlarged, hardened, and at first painful. The prolabium often cracks, bleeds, and scabs. These conditions may last from three weeks to as many months. Occasionally the discharge from the nose brings out an attack of eczema on the lip, which may spread over the rest of the face.

These appearances, being due to the previously impaired health of the child, become important indications in respect of the treatment.

The mucous membrane of the lips, gums, cheeks, soft and hard palate, usually reddened, in rare cases remains normal. The intensity of the redness varies, and it is not equally distributed, being usually most marked on the gums[1] and lips. On the cheeks and palate the redness, sometimes uniform, is mostly mottled. The gums are much reddened, rather swollen, and easily bleed. On the lips the redness, whilst uniform, is more intensely marked at places forming spots, mostly round, sharply defined, and especially noticeable in the neighborhood of the frænum. Almost always on the gums, often on the lips, and occasionally on the cheeks and soft palate, are seen thin opaque white patches or films, looking as if the mucous membrane had been swept over with a solution of nitrate of silver; these vary in size, are often sharply defined, can be easily removed, and leave the mucous membrane entire, but red and sometimes bleeding. On the decline of the rash and fever, the inflammation of these parts subsides. The redness for the most part disappears, leaving only a coarse ramiform injection, best seen on the lips. The swelling of the gums subsides, but the whole mucous membrane retains for some time a swollen, spongy, and not unfrequently a glazed appearance. The white patches cease to enlarge, thin away, especially at their edges, and finally disappear; often, however, remaining for ten or fifteen days.

Occasionally ulceration is observed, usually slight—but sometimes severe—commencing close to the teeth (especially the incisors), appearing generally after the fever has subsided, but sometimes as late as the tenth or twelfth day of the disease. If slight, this usually heals in three or four days. It is noticed mostly in sickly children.

Aphthæ, and superficial, sharply cut ulcerations—usually small, round, or oval, but occasionally as large as a shilling, and irregular in outline, covered in some cases with an ash-gray, easily removable exudation, at others by a tough, smooth, leathery adherent membrane—are sometimes seen on the gums, lips, tongue, &c. They especially occur in certain epidemics. Of no danger, they heal in a week or ten days; the membranous form being more obstinate, lasting sometimes three weeks.

The tongue is rarely clean throughout; mostly thickly coated, moist, and with a few red prominent papillæ, it is sometimes dry and brown, the latter state indicating much depression, and being suggestive in respect to treatment. It begins to clear from the fourth to the eighth day of the disease. The fur is at first thrown off in the form of patches—leaving clear spots, sharply defined, oval or round, and about two to three lines in diameter. The rest of the tongue becomes clean by the eighth to the twelfth day, but still the patchy appearance remains, and can often be seen till the twelfth or fifteenth day of the disease. If any intercurrent disease supervenes, the tongue remains coated for a longer time.

The tonsils and mucous membrane of the pharynx, generally red and rather swollen, may be severely affected, and the tonsils may be so enlarged that they nearly meet. The mucous membrane, red, and at first dry, on the subsidence of

[1] Especially that part corresponding to the incisor and canine teeth.

the inflammation, becomes moist and covered with much muco-purulent matter. This ceases in a few days, leaving the mucous membrane spongy and sodden in appearance. Whilst the inflammation lasts, there is usually some pain, especially on deglutition. This is usually not severe. The Eustachian tube may be involved, and there is then great pain, and difficulty of hearing. This is uncommon.

Vomiting, not common except at the very onset of the disease, may be persistent and continuous, the blandest food being immediately expelled. Sometimes the vomited matters are of a grass-green color, usually at the same time there is diarrhœa with motions of a similar color. Such vomiting only occurs in serious cases, and calls for a grave prognosis.

Diarrhœa, beginning usually at the time of the eruption, is not infrequent. Often severe, and sometimes bloody, it may cause death in young children. The color of the motions varies; they are light-colored, clayey, and sometimes green. The diarrhœa continues for a variable period, often after the fever has subsided, even to the tenth or twelfth day. The motions may continue clayey in color after they have become less frequent and of greater consistence.

During the course of the disease, and for some time after, the bowels are very easily influenced by purgatives, a fact that should be borne in mind in the treatment of the disease.

The diarrhœa lasts longest in the weak and sickly.

The cough, at first hacking, frequent, and troublesome, in a few days becomes more annoying, violent, ringing, clanging, or often paroxysmal in character; sometimes so violent as to cause retching, and even vomiting: it usually greatly improves when the rash fades, and often disappears in a few days more. It may, however, continue for some time, retaining its paroxysmal character. The cough may become paroxysmal as late as the eleventh or fourteenth day.

Expectoration, at first scanty, clear, and viscid, on the decline of the rash and the subsidence of the inflammation of the bronchial tubes, becomes muco-purulent, abundant, sometimes of a bright-green color, and nummulated, the masses floating in a clear, thin watery fluid. It usually quickly lessens in quantity, and mostly disappears by the tenth or twelfth day of the disease.

Urine, scanty, and on cooling depositing an abundance of lithates, has generally, during the fever, a peculiar yellow color. Albumen in small quantities is frequently, blood less commonly, found during the fever days. Both the urine and breath have a peculiar odor. This, in the author's experience, is only met with in children, in whom it can be detected in all acute febrile diseases, especially when the mucous membrane of the mouth is affected.

COMPLICATIONS AND SEQUELÆ are either the usual anatomical lesions, so increased in severity, or continued after the fever has declined, as to become of serious importance; or intercurrent diseases not usually attendant on Measles. Very various in nature, and often of great severity, they not unfrequently terminate in death.

They vary in different epidemics, and are, to some extent, influenced by the season of the year. Pulmonary affections are more common in winter than summer. Some are moreover dependent on the previous depraved health of the patient, such as the gangrenous inflammation.

Convulsions, not uncommon in children at the very commencement of the attack, are usually without danger. They may however be repeated, and terminate fatally. Occurring by no means commonly, at a later period of the disease, they are then usually repeated, and accompanied with some severe internal inflammation, mostly of the lungs. At this period they are generally fatal.

Laryngitis, commencing with slight cough, soon followed by much oppression at the chest, hoarse cough, noisy and stridulous breathing, occurs before the eruption, or with a recession of the same. It may be croupous or diphtheritic.

It mostly disappears on the evolution of the rash, and, if not then, on the decline of the fever.

Chronic inflammation of the larynx is commonly met with, and generally proves obstinate. It disappears only to return again on slight exposure to cold. The voice and cough are hoarse, and sometimes at the same time there is deafness in one or both ears. When the latter occurs, the tonsils are usually enlarged, and at the same time the patient snores much during sleep. This affection may continue to recur for several years.

Chronic inflammation of the bronchial tubes, with or without the last-mentioned sequelæ, may follow Measles. The cough is hollow, ringing, hacking, or sometimes paroxysmal, and is easily aggravated by exposure to cold. With or without expectoration, but accompanied usually by some oppression of breathing, it may continue with remissions, or for a time disappear; but may, for years, return on exposure to slight exciting causes.

Capillary bronchitis may occur either during or subsequent to the decline of the fever. It is almost always fatal in young children under two years of age.

Pneumonia, simple or lobular, occurs both as a complication or as one of the sequelæ.

The symptoms of lobar pneumonia are mostly well marked and characteristic; they may, however, be masked or take on the character of those of typhoid fever.

Lobular pneumonia, by no means uncommon, is often difficult to diagnose. The fever is high, the respirations are much hurried, the nares act strongly with respiration. Dulness on percussion of the chest is absent always at the commencement, and sometimes throughout the disease. There is heard, irregularly scattered over the whole of both lungs, fine crepitation—occurring with both inspiration and expiration—but in children this crepitation is by no means easy to distinguish from the subcrepitant rhonchus of bronchitis.

In bronchitis the rhonchus is at first limited to, and always most marked at, the bases of the lung. It is usually larger and more evenly distributed than the crepitation of lobular pneumonia, which is generally limited to different parts of the lungs. In bronchitis the fever is mostly less severe, and there is greater lividity of the lips, with less disturbance of the respiration. But, though the above differences often hold good, yet cases occur in which it is often impossible to make the diagnosis. Its influence on the rash is greater than that of bronchitis, the fever being higher.

Broncho-pneumonia is by no means uncommon either as a complication or one of the sequelæ of this disease. The disease begins as ordinary bronchitis; this increases in severity; abundance of loose, subcrepitant rhonchus is heard over the chief part of the lungs, at first limited to, and always most abundant at the bases. This is at first moist and distant; but as the consolidation advances, it sounds very superficial, ringing, dry, and high-pitched. This change in the character of the rhonchus is usually the only sign by which it can be told that the bronchitis has passed into broncho-pneumonia. For mostly all dulness is absent; nay, there may be, and at first generally is, increased resonance, whilst the respiratory sound is merely harsh, or completely covered by the rhonchus. If the consolidated portions coalesce, dulness may be developed, and tubular breathing caught, but this only happens in rare and very severe cases.

In this affection the fever is very high, the nares dilate greatly during respiration, the face is livid, the respiration very greatly labored, the jugular vein often full, and the expectoration, where it occurs, is bronchitic in character.[1] The child very frequently, perhaps generally, sinks; the surface becomes cool; the pulse weak, feeble, and fluttering; the respiration superficial: the expired air cool; sordes collect on the teeth and gums; delirium of a low muttering character is present. There is a muscular trembling, picking of the bed-clothes, and after a period varying from six to twelve days the child dies.

It mostly occurs in children from two to six years of age, and is very frequently fatal. Its duration is said to be two to four weeks, but the child either dies before, or the fever subsides on the eighth or tenth day. Rhonchus may continue to be heard some time longer.

Beginning as simple bronchitis, the inflammation spreads laterally from the fine bronchial tubes, producing minute red spots or streaks. Red and then gray hepatization soon follows, and little bodies varying in size from a millet seed to a pea are formed.

Ulceration may begin in the mucous membrane of the bronchial tubes of the hepatized tissue and spread laterally, till small cavities, filled with a grayish tenacious fluid, are formed. A bright red line in the hepatized portion, and bounding the cavity, may be seen. These hepatized portions of lung may be so numerous that as they enlarge laterally they coalesce, and thus much of the lung may become completely consolidated, and such portion is sometimes riddled with small cavities.

When in the state of red hepatization, the little bodies mentioned look not unlike tubercle, but they are most abundant at the base, less sharply defined, stand out less prominently, are less dense, and are at the same time more granular-looking.

Gangrenous inflammation of the mouth usually begins insidiously: it occurs sometimes during, but mostly after, the fever. There is at first slight excoriation, the inflammaiion then quickly spreads, the parts become much swollen, and the eye may be closed. The tissues become hard, red, and painful. The surface is sometimes at first covered with a dirty, fetid, gray exudation. The tissues slough, the cheek may be perforated, the gums destroyed, and the teeth drop out. Both cheeks may be affected. The affection is only met with in children, who almost always die.[1] Similar inflammation occurs sometimes in the vulva.

Acute tuberculosis or chronic phthisis may occur during the course of the disease, but it usually first gives evidence of its existence after the fever has declined. Acute tuberculosis follows Measles more frequently than any other of the acute specific diseases, hooping-cough being perhaps excepted.

Bleeding at the nose in rare cases may be so great as to endanger life.

Inflammation of the ear may at first

[1] Children under twelve rarely expectorate.

[1] See Cancrum Oris. Vol. II.

escape notice. The child cries greatly, and apparently without cause, and puts its hands to its ears. There may be some redness and swelling of the meatus. On the third or fourth day a muco-purulent discharge occurs.

[Inflammation of the eyes (conjunctivitis) is far from uncommon; sometimes leaving the eyes in an irritable state, incapable of much use, for a considerable time. Examples of blindness from this cause, if they occur, must be rare; but such are said to have occurred; probably from neglect of the ophthalmia present during the attack and convalescence.—H.]

Gangrene of the lung, diphtheritis of the labia, acute Bright's disease with anasarca, and some other complications, may occur.

It may be added that the complications occurring during the eruptive period are usually without danger.

DIAGNOSIS.—This is made chiefly from the rash, and the co-existing coryza.

As the rash in the different stages somewhat simulates that of scarlet fever, roseola, smallpox, typhus fever, and syphilitic roseola, the distinction between these rashes must be mentioned.[1]

Scarlet Fever.—The rash appears on the second day of the disease. It is very generally diffused. In Measles the papillæ are often widely scattered. The elevations always present in scarlet fever are very small (punctiform); those in Measles much larger (papilliform). Where the rash of the Measles forms patches, the elevations seen in the general redness are larger than those of scarlet fever; the edges of these patches in Measles are sharply defined and abruptly elevated; in scarlet fever, ill-defined and not at all elevated.

Occasionally in scarlet fever there are a few prominent elevations on the arms and legs; these may be mistaken for the papillæ of Measles, but the rash on the rest of the body is sufficient to establish its true nature.[2] The tint of redness in most cases is different in the two eruptions. Coryza is not noticed in scarlet fever, though the conjunctivæ are almost always injected.

Roseola Æstiva may have a rash not unlike that of Measles. This rash in appearance is intermediate between Measles and scarlet fever, but generally more closely resembles the latter. Thus, the rash is generally much diffused and ill-defined, the elevation is slight, coryza is absent, and the fever is trifling. It frequently occurs many times in the same patient, and often at the same period of the year.

Smallpox.—This disease in some respects corresponds to Measles. In both there are catarrhal symptoms. In both small red points beginning on the face and then spreading over the rest of the body, are met with. In smallpox the disease begins abruptly; in Measles not infrequently insidiously. In smallpox there is often severe pain in the back and head, with much vomiting. These symptoms are most generally absent in Measles. The rash of smallpox is from its commencement hard and "shotty," quickly becomes crowned with an umbilicated vesicle. Moreover, the fever greatly declines or even disappears temporarily, when the rash is fully out, in both respects differing from Measles. In smallpox, if unmodified, a characteristic smell is present. The rash appears on the third day in smallpox, usually on the fourth day in Measles, but the departure from this rule in Measles is so frequent, that no great stress can be laid on this distinction.

Typhus Fever.—The mottling left after a severe attack of Measles is often extremely like the petechiæ of typhus fever, but the early appearance of the rash of Measles will in all cases be sufficient to ensure a correct diagnosis. The rash of typhus fever, moreover, appears first on the wrists.

The mottling left by the rash of Measles is in all respects like the mottling seen in syphilitic roseola, but the rashes are different at their commencement, and the mottling left by Measles is most marked on the shoulders and over the scapulæ, the mottling of syphilitic roseola is generally absent from these regions, and is most marked on the trunk. Moreover, the general symptoms are sufficiently different to render a mistake impossible.[1]

Occasionally copaiba, morphia, and certain fish, produce a rash which in some instances it is impossible to distinguish from that of Measles. The rash, however, produced by these causes, is often very irregular in its distribution, and lasts a much longer time than that of Measles,

[1] The rash of Measles is often, at its commencement, compared to that of flea-bites, but the differences between them are always well marked, and by the most superficial examinations they can be distinguished. Flea-bites take on two different forms. In the simpler there is no elevation, but a central point of redness, not removable by pressure, surrounded by a small ring of a less intense color; this latter redness disappears on pressure. In another form there is a round firm swelling, varying in size, often as large as a pea; this itches greatly. It has all the characters of the wheals found in urticaria.

[2] It is stated that the two fevers may co-exist, and that these rashes may be mixed. No such case has come under the author's notice. [See article on Rötheln.—H.]

[1] The author believes that Dr. Hare was the first to point out this resemblance between the rash of Measles and that of syphilitic roseola.

if the medicine be continued. Occasionally there are, mixed with this rash, wheals of urticaria; when such is the case, there is much itching.

Prognosis.—Usually a mild disease, the prognosis is very favorable.

The degree of danger is dependent on the previous health of the patient, on the time of year at which the disease occurs, and on the nature of the epidemic.

If the health has been bad, the prognosis is more serious, but even in such cases the result is mostly favorable.

If the patient be tubercular, the prognosis becomes grave. Cold and damp weather, favoring the development of pulmonary affections, increases the danger of the disease.

The severity and the nature of the complications occurring during different epidemics, vary greatly. Yet, in the worst epidemics, the majority of cases recover.

Treatment. — Not possessing any medicine capable of shortening the duration of the disease, it must be our object, in treatment, to conduct the fever to a favorable termination, and to ward off any intercurrent disease.[1]

The patient must be confined to bed. Too much light should be excluded, for strong light is annoying to all febrile patients, and in Measles causes some pain in the inflamed conjunctivæ.

The room should be well ventilated, and all excreta and dirty linen immediately removed. The patient must be most carefully protected from draughts. The sense of heat and dryness of the body, sometimes most distressing to the patient, can be much alleviated by washing the surface with soap and tepid water; too great exposure being avoided by one part of the body being cleansed, dried, and covered, before the rest is exposed. The feeling of tension of the hands and feet can be relieved by rubbing these parts with some firm greasy matter, such as suet.

All sources of annoyance or irritation, all noises should be avoided, and thus sleep is promoted, a condition which most materially affects the welfare of the patient,—sleep lessening the fever and increasing the appetite. Food, light and nutritious, such as arrow-root, gruel, good beef-tea, milk, chicken or veal broth, jellies, &c., should be given at the usual meal hours. The quantity should be moderate, great care being taken that the digestion be not impaired by too large ingestion of food. Should the patient be very weak, the food must be administered in small quantities and at short intervals. There is no stimulant so important as food. The prospect of recovery in all acute febrile diseases is very greatly, if not mainly, dependent on the power possessed by the patient of digesting and assimilating food.

If there be often-repeated sickness,[1] food of the very blandest nature, pounded raw meat,[2] Liebig's beef-tea,[3] uncooked white of egg diluted with water or barley water, should be given in small quantities, and be very frequently repeated.

Thirst must be assuaged, and thus the restlessness of the patient allayed by drinks regulated with respect both to quantity and quality. Large draughts should be prohibited, as they tend to impair the digestion, and cause diarrhœa: small quantities, swallowed slowly, or ice to suck, are sufficient to allay thirst, and they also prove grateful to the patient.

The patient, however, must be allowed to take larger quantities of fluids than in health, as an increased quantity is required by the system during the existence of fever.

Acid or acid and bitter drinks are generally found to lessen thirst to a greater degree than mere water, and are moreover grateful to the patient. "Imperial drink," lemonade, with very little sugar, raspberry vinegar, or weak infusion of cascarilla, with a few drops of hydrochloric or nitric acid, are all—especially the latter—useful.

Stimulants are administered to support the strength of the patient. This they do in a great measure by promoting digestion, and by also directly increasing the force of the heart's action; for in fever-patients the pulse, under the influence of alcohol, most certainly gains in force.

[1] The remarks made respecting the treatment of Measles, of course apply for the most part to all febrile diseases, and especially to those of long duration.

[1] A very unfavorable symptom, for sickness itself very greatly prostrates the patient, and by the vomiting the nutritious matters are returned: nay, more, the vomiting is due to a condition of the mucous membrane of the alimentary tract most unfavorable to digestion.

[2] Before pounding the meat all fat and tendon should be removed.

[3] Liebig's beef-tea is made by cutting one pound of beef very small, pouring on this one pint of water, to which have been added thirty minims of hydrochloric acid and two scruples of common salt, and allowing this to stand three hours. It should then be strained and strongly expressed: it is then fit for use. [Better than Liebig's preparation, in the estimation of many, is beef-tea made by pouring a pint of cold water upon two pounds of lean beef, chopped small, letting it stand near the fire for two hours, then boiling for twenty minutes, skimming while hot, and pouring off at once, *without filtration*. This contains all the nourishing qualities of the meat, in an easily assimilable state. It admirably exemplifies the Hippocratic aphorism (ii. 11), Ῥᾷον πλησθῆναι ποτοῦ ἢ σιτίου.—H.]

The administration and quantity of stimulants given must thus be regulated by the condition of the patient. Guidance in this respect can be derived from the previous history of the patient, the existing state of the patient, and the nature of the epidemic.

If the patient previous to the fever, has been in an unhealthy state from any cause, such as overwork, excesses of any kind, or is the subject of scrofulosis or tuberculosis, in whom the fever always tends to become typhoid in character, and to leave one or more of its sequelæ[1] behind, stimulants may with benefit be given early in moderate quantities: and at the same time the patient should be carefully watched, so that their quantity may be increased in anticipation of any great amount of prostration.

Again, in some epidemics the disease is violent, and apt to become typhoid. Patients met with in such epidemics may have stimulants given them early with benefit. But the chief information respecting the administration of stimulants, and the quantity of these to be given, is derived from the then existing state of the patient. If the disease be progressing favorably (the previous health having been good), stimulants had better be altogether abstained from. But if the patient become at all prostrate, the tongue dry, the pulse small and frequent, stimulants must be given freely and often. It should be the anxious endeavor of the physician to anticipate such a state, and prevent it by the early use of alcohol.[2]

In reference to the amount to be given, no exact rules can be laid down. Experience at the bedside alone can teach this. It may, however, be stated that young children bear stimulants well in disease.

It is a matter of but little importance in what form the stimulant is given, provided it is sound in quality. The patient's taste should be consulted. Thus, brandy, gin, whiskey, port or sherry wine, bitter or draught beer, may be given.[3]

Only one kind of stimulant should be administered at the same time, though it may be frequently changed at the request of the patient.

[There are certainly very many cases of Measles, indeed the large majority under ordinary circumstances, which require no alcoholic stimulation at any period of the attack. While young children in states of positive exhaustion bear stimulants very well, the doses in which they are given must, for beneficial action, be reduced in greater proportion than is needful with any other remedies except opium and other narcotics.—H.]

At the commencement of the disease, if vomiting has not occurred, an emetic can be often given with benefit, and the bowels, if confined, should be opened by a mild purgative, it always being borne in mind that in Measles the intestines are usually very easily influenced by purgatives. Acetate of potash, acetate of ammonia, and nitric ether may be given in suitable quantities every three or four hours; and if the cough be troublesome, some ipecacuanha wine may be added. If the skin be hot, the pulse hard, and incompressible, aconite, given hourly, will be of much service.

Opiates are but rarely required; sleep should be obtained by removing, as far as possible, those conditions that are liable to render the patient restless.

Bleeding at the nose, if severe, should at once be stopped. This can be accomplished by the injection of cold water, or the breathing up of astringents, in fine powder, into the nose; should these fail, plugging becomes necessary.

For the convulsions that occur, often but little is required, and but little can be done—those occurring early quickly disappearing; those occurring late almost always ending in death.

In the treatment of laryngitis, Trousseau warns against the application of leeches, and adopts Graves's recommendation of a sponge wrung out of boiling water, and applied over the larynx.

[Some practitioners of large experience believe, nevertheless, that leeches may do a great deal of good, at least when the application of the hot wet sponge for a time does not afford entire relief.—H.]

If suffocative catarrh supervene, or a serious internal inflammation cause the recession of the rash with much prostration and possibly with coma, a warm bath, to which two tablespoonfuls of mustard have been added, may be used with benefit. The child should be kept in the bath till the arms of the nurse holding the child tingle and smart.

Gangrenous inflammation is best treated locally, by the application of strong hydrochloric or nitric acid, and generally by the free use of stimulants, with sesqui-carbonate of ammonia and tonics.

If there be much inflammation of the fauces (but this is not usual), linseed

---

[1] The probability that the various anatomical lesions met with in Measles will become chronic, is determined by two conditions—first, by idiosyncrasies, inherited or acquired—thus patients who have had many attacks of bronchitis, will be very liable to have some chronic bronchitis left behind; and secondly, and chiefly, by the previously impaired health of the patient.

[2] Whilst in such cases stimulants should be freely given, it must not be forgotten to give the patient food in small quantities, frequently repeated; for food is the best and most natural stimulant.

[3] The patients will sometimes take stimulants in this form when they refuse them in all others.

poultices to the throat, and the inhalation of steam or ice to suck will be found of benefit. On the subsidence of the inflammation, if the formation of muco-purulent matter continue, adults can use astringent gargles. It must be borne in mind that children under eight years of age cannot gargle, and thus the fluid should be injected into the throat with a syringe. There is no fear of choking, provided the stream of the injected fluid be not too great.

Inflammation of the lungs, when occurring in children, requires active stimulant treatment. Bleeding in these cases can never be borne.[1]

The chronic sequelæ being dependent on the impaired health of the patient, are best treated by those means and medicines that promote the general health of the patient, and also when possible, by local applications. The child should be warmly clothed, with flannel next the skin. Much of the day should be spent in the open air and in direct sunlight, unless the patient be too young, or the sun's heat too powerful. Hours of rest should be long, and sleep may be indulged in with advantage for a short time during the afternoon.[2] Simple healthy food, at regular hours, with a very moderate allowance of stimulants, or even without any, should be given. Sea-bathing or cold-sponging will be found to be of the very greatest advantage for the cure of these sequelæ. The sea-bath should be taken about three hours after breakfast. If the patient be very weak, a very short stay in the water of one to two minutes at most should be allowed. The sea should be smooth, and if the weather be wet or stormy or cold, a tepid sea-bath is preferable to open-air bathing. Children should be coaxed, not dragged into the water, as baths fail to act as a tonic, and produce much depression in persons laboring under strong mental emotions.

The cold sponge-bath may be used before breakfast. If the patient be very weak, and the weather very cold, a little warm water should be added. The time the sponging should be continued depends on the condition of the patient; the weaker the subject the shorter the time; thus one to two minutes is in many cases as much as can be borne, for if continued too long, in such cases no tonic effect follows, but depression, which generally lasts during the rest of the day. Very young children can be much benefited by cold-sponging, even during the winter months, if the bath be properly used. The sponging should not be continued longer than a minute, and if the weather and water be very cold, the child's feet should be placed in warm water, and the bath administered before a good fire; by these means reaction is promoted. Salt may be added to the bath with benefit.

Under such treatment all the sequelæ greatly diminish, nor need there be any apprehension that the chronic bronchitis should be aggravated; for if the baths be administered in the manner described above, this disease almost invariably greatly improves.

Of medicines, iron, quinine, and especially cod-liver oil, are always very beneficial. The latter should be administered after food, for at this time the bile is poured out in largest quantity into the intestinal canal, and it is found that animal membranes moistened with bile allow fats to pass through.

The chronic discharge from the ears, nose, and vagina is usually easily arrested by local applications. The affected part should be washed frequently (in proportion to the amount of discharge) during the day with tepid water, and injections of lead lotion, or of solutions of alum (℥j. to ℈j.), or sulphate of zinc (gr. iv. to ℥j.), will generally promote the cure.[1]

The chronic inflammation of the larynx, or of the bronchial tubes, is most quickly mitigated and generally cured by inhalation of steam with ♏x. to ♏xx. of tr. iod. poured on the water, from which the steam is given off. Creasote may be sometimes used, but does not give such good results as the iodine. The inhalation should be used night and morning for about ten minutes.[2] The chronic diar-

[1] [From this *dictum* it is necessary for the American editor to express absolute dissent. While it is true that pneumonia complicating measles does not often *require* bleeding, it may, at least, in a certain number of cases, be well borne, even by children; and the universal employment of active stimulation (by which the author means, no doubt, the free use of alcohol) to this complication must, it is believed, while saving some cases, aggravate others to a fatal end.—H.]

[2] Every care should be taken, during convalescence, to insure sound sleep. The supper should be light, and be taken one or two hours before going to bed. No stimulants should be taken during the evening, unless the patient be very weak. If baths cause restlessness, they must be discontinued for a few days, or the patient should be directed to remain in the water for a shorter time. During convalescence the patient is in much the same condition as a child. It is a period, if not of growth, of great repair—a condition analogous to growth. The appetite, digestion, and assimilation are greatly influenced by sleep. This influence is well seen in ulcers on the surface of the body. After a restless night these are painful, throbbing, inflamed, and swollen, and apt to spread; whilst after a refreshing sleep they have a much healthier appearance.

[1] In the author's experience lead lotions are preferable to the others.

[2] The cough is greatly influenced by change of climate. If the cough be easy and the expectoration scanty, a warm moist climate, but

rhœa, which occasionally follows Measles, should be treated by a carefully regulated diet, and the occasional use of hyd. c. creta. If the diarrhœa be exhausting, it can be generally temporarily stopped by an injection of starch, of the consistence of cream, and about two ounces in quantity; to this some laudanum or one of the metallic astringents must be added.

Raw pounded meat sometimes has a beneficial influence on the diarrhœa.

[RÖTHELN; GERMAN MEASLES.

By Henry Hartshorne, M.D.

Definition.—An exanthematous disorder, intermediate in character between, or composite of, Scarlet fever and Measles.

Synonym.—Rubeola. This term is, by some writers, restricted to it: although by many others it is applied indiscriminately to Measles.

History.—Apparently more common on the continent of Europe, this affection has not been much noticed in Great Britain, and has been scarcely described until within a few years in the United States. Yet there is reason to believe that it has long, if not always, been tolerably frequent; its resemblance to both Measles and Scarlatina causing it to be usually assigned in description to one or other of those diseases.

Dr. Murchison and others have expressed the opinion that it is a distinct disease, having nothing to do with either Measles or Scarlet fever, and not protecting its subjects from either of those affections. Dr. J. Lewis Smith, of New York,[1] observed and described an epidemic of it prevailing in that city in 1874. In 1875 more than a dozen cases of it occurred at Haverford College, near Philadelphia. More than one such an epidemic has been witnessed in rural localities, in Maryland and elsewhere; the popular designation for the affection being French Measles.

Symptoms.—In the cases observed at Haverford, Pa., the early general symptoms were mild; malaise, headache, slight sore throat, loss of appetite, and debility. In two or three days, with some increase of fever, a rash appeared, having more the color of that of Scarlet fever than of Measles, but dotted or miliary (sub-papular) in form. In some cases it became almost continuous, with a moderate amount of tumefaction of the limbs and general surface. In one instance the fauces exhibited a scarlet hue to the eye, although no feeling of soreness of the throat existed in that case. Catarrhal symptoms were not noticed in any of these patients. In some of them, the glands of the neck were swollen. None had a very high degree of fever. The duration of the attack was about a week; and convalescence was rapid. No sequelæ were observed in any instance. Dr. J. Lewis Smith mentions that albuminuria and dropsy occasionally follow this disorder, as well as chronic bronchitis. Altogether, in several characters, Rötheln most nearly approaches Scarlet fever; while, in the absence of severity in any of its symptoms, it is more like Measles.

Pathology.—Opinions may readily differ as to the nature of this affection, whether it is a specific disorder, or a *hybrid* of Measles and Scarlet fever. The latter would seem to be at least possible, according to the analogy of some other diseases; although hybrid maladies are certainly rare. Examples of such are met with in malarial regions bordering upon yellow fever districts, where malignant remittent combines the characters of two usually distinct fevers; in typho-malarial fever, seen in many cases in the U. S. Army during the civil war of 1861–65; and in scorbutic dysentery, occurring among soldiers during that war as well as in 1854–56 in the Crimea. In the absence of any record of fatal cases, the morbid anatomy of the affection can only be conjectured; and its true pathology may be considered as yet undetermined.

Diagnosis.—While a place in description and classification seems to be practically needed for a combination of symptoms not included in the typical account of either of the commonly recognized exanthemata, reasons have already been given for obscurity in the diagnosis of its examples. In a case occurring some years ago in Philadelphia, two physicians of similar experience saw the patient within

---

if the expectoration be abundant, a warm dry climate is beneficial.

[1] [Sanitarian, July, 1874.]

an hour of each other; one pronounced it Measles and the other Scarlet fever. When, however, after about three days of indisposition, a rash breaks out which is miliary in form, but of a nearly scarlet hue, attended with slight sore throat, with or without a disposition to cough, and moderate fever, all of which symptoms subside within a week, leaving no sequelæ, and especially when a number of such cases occur in the same neighborhood, exhibiting the same absence of severe character, and all followed by rapid convalescence, we may conclude it to be an epidemic of Rötheln or Rubeola. This is confirmed if neither Scarlet fever nor Measles is prevalent at the same time and place.

Treatment.—Nothing peculiar exists in the indications of this affection, apart from those of other exanthematous fevers. Almost no treatment is usually required. Confining the patient to his room as a measure of precaution, even if not ill enough to seem to need it, a single dose of citrate of magnesium or Rochelle salts may be given. If the fever should be considerable at night, solution of citrate of potassium or acetate of ammonium may be prescribed. Flaxseed lemonade will be suitable as a demulcent and diluent, if either sore throat or cough should call for any treatment. These mild measures, with care to avoid exposure during convalescence, lest a bronchial attack, or possibly suppression of perspiration, renal congestion, and anasarca might occur, are all that are likely to be appropriate. A second attack is not to be expected; but it is not proven that Rötheln affords protection against either Measles or Scarlet fever.]

PAROTITIS.

By Sydney Ringer, M.D.

Definition.—An acute febrile disease, characterized by an anatomical lesion situated in one or both parotid glands, which runs a short course, and almost invariably terminates favorably.

Synonyms.—Parotitis; Cynanche parotidea; Ziegenpeter (Germ.); Parotide, Parotidite (Fr.); Mumps (Engl.).

Symptoms.—Beginning abruptly—rarely with rigors, more commonly with a feeling of chilliness, with or without vomiting, pain in the head, back and limbs—the disease in its course is accompanied by the symptoms common to all febrile diseases. The face is sometimes flushed, the lips may be dry; impairment of strength, variable in degree, is generally slight, and sometimes absent; the tongue is furred, but usually moist; the appetite, in some cases natural throughout, is in most impaired, and may be *nil*. The patient usually complains of thirst, and the bowels are often confined.

The pulse and respiration are increased in frequency, the former often greatly so, especially in children; it also sometimes gains in force; the urine is scanty and high-colored; the temperature of the body is raised, but this elevation varies much in degree.[1] Usually at the very commencement of the disease, but occasionally postponed for twelve, twenty-four, or even thirty-six hours, the affection in one or other parotid gland, sometimes in both, manifests itself by pain, followed in a few hours by swelling of the glands and stiffness of the jaws.

The pain and swelling first appear immediately beneath the ear, and posterior to the ramus of the jaw, and from this part spread in all directions, upwards to the face, downwards and backwards in the neck. On the face the swelling appears earliest on that part immediately in front of the lobe of the ear, and then quickly extends upwards to the zygomatic arch, and forwards involving a variable extent of the face. The swelling disappears in the inverse order of its invasion. It is at first flat, but soon becomes more prominent, and is usually most marked anterior to the lobe of the ear. Firm and elastic to the feel, it is generally tolerably well defined, but does not pit on pressure; and, whilst the skin over the enlargement is mostly natural in color, it is in some cases mottled with a slight red blush, and is in rare cases of a bright scarlet hue. The redness disappears on pressure, but quickly returns on the pressure being removed. The degree of enlargement va-

[1] In one case the highest temperature reached was $101\frac{3}{5}$; in another, however, it rose to $103\frac{1}{5}$.

ries: whilst in some cases apparent only behind the ramus of the jaw, or even so slight that it easily escapes notice, it is more usually co-extensive with the parotid gland, and occasionally extends far beyond this, involving a large part of the face and neck, and, in some rare cases, reaching to the upper part of the chest, giving to the head and neck a pyramidal shape. The swelling increases from three to six days, then usually remains stationary for twenty-four or forty-eight hours, after this rapidly declines, and often has entirely disappeared by the eighth or twelfth day from the commencement of the attack; the redness subsides much earlier, and is occasionally followed by a superficial desquamation of the cuticle. Not unfrequently one or both submaxillary glands are also involved, in which case the swelling extends along the body of the jaw, reaching nearly to the symphysis.

The pain and tenderness, dull and aching in character, vary greatly in degree; sometimes they are only complained of on movement of the jaw, and are then seated beneath the ear, and behind the ramus of the jaw—a fact easily accounted for when it is remembered that this part of the enlargement is most affected by the movement—in other cases, however, the pain is constant and severe, and occasionally extends beyond the limits of the apparently affected tissues, reaching even to the chest and shoulder.

The pain more severe in adults than in children, usually lasts only during the time the swelling is increasing: on the other hand, tenderness on pressure, which is always present, continues for some days longer, and is longest observed in the part first affected. The jaw is generally fixed, and the mouth slightly open; moreover, its movements are limited, or entirely prevented, the degree of impediment being proportionate to the amount of pain and swelling present. Consequent on the impediment in the movement of the jaw just mentioned, the speech is affected, and mastication can be but most imperfectly and painfully performed; and when, as is sometimes the case, the tonsils are enlarged and inflamed, and the swelling extends even to the cellular tissue of the pharynx, deglutition also becomes difficult, and danger of suffocation, in very rare instances, is imminent. The mucous membrane of the mouth is unaffected, and the salivary secretion, in some cases diminished, in a very small number increased, is usually natural in both quality and quantity.

Occasionally only one parotid gland is affected; in most cases, however, both suffer. The left side is most frequently the first involved, and the pain and swelling in it precede that in the right for a period varying from twelve to thirty-six or forty-eight hours. The side first attacked suffers the most severely. It is stated that, in rare instances, the gland has suppurated.

Other organs besides the parotid and submaxillary may be affected. In many the testicles, one or both, may suffer; whilst in the female the mammæ, the labia majora, and uterus are the parts occasionally attacked. The tonsils and pharynx may also be involved. When the testicle is diseased, the inflammation involves both the tunica vaginalis and the epididymis; and if the disease be limited to one of these organs, this is situated on the same side with the parotid, solely or most severely, affected. These complications, or metastases as they are termed, usually make their appearance whilst the parotid and the submaxillary glands are enlarged; but, on the other hand, the swellings may decline and disappear from the glands, and not make their reappearance elsewhere until a period, varying from a few hours to one or two days, has elapsed. In this last case, whilst the swellings are in abeyance, active general symptoms, sometimes of an alarming character, may occur; there may be a feeling of great anxiety, pallor of the face, coldness of the extremities, smallness and great frequency of the pulse, and to these symptoms delirium, vomiting, and purging are sometimes added. However, on the reappearance of the local mischief at any part of the body, these symptoms disappear. These metastases, rarely occurring in children, and by no means common in adults, are prone to occur in individuals of the same family; in other words, family idiosyncrasies tend to their development.

The duration of the disease varies; thus, in mild cases it may run its course in four days, whilst in severe cases it may continue for ten days. Its duration is usually longer, and the fever higher, when metastases occur.

Pathology.—As the disease rarely kills, the opportunities for investigation on this point are necessarily but few. It is generally, however, held that the organ affected, and the cellular tissue within and around it, are inflamed, and that there is an excess of serosity in these parts.

The glands may remain somewhat enlarged and hardened for a considerable time after all the acute symptoms have disappeared, and it is even stated that in rare cases the affected testicle has atrophied.

In Mumps, have we primarily a general disease of which the local effects are the sequence; or, on the other hand, is the disease in the first instance local, and are the general symptoms dependent on such local mischief? In the present state of medical knowledge, this question cannot be answered. Suffice it to say that, whilst

some diseases, such as typhoid fever and dysentery, were formerly thought to be primarily "general," further observation on these affections has at least rendered it possible that both of them are in the first instance "local," and that the general symptoms are secondarily dependent on these local lesions. Should this be fully established, it will go far to render probable that most, if not all, diseases are at their commencement local, and, amongst others, the disease under consideration. In favor, however, of the older view, the author may mention the following case in which, after the temperature of the body had become normal (*i. e.*, after all fever had disappeared), the right parotid, which had previously remained healthy, began to enlarge, became painful, and corresponded in all respects in its behavior to the left, this latter gland having been previously affected during the time that the temperature of the body was raised. As no subsequent elevation of the temperature occurred in this case at least, the local mischief in the right parotid was insufficient to elevate the temperature, and was certainly in point of time sequential to the general condition.

Most common between the ages of five to fifteen, the liability to the disease rapidly diminishes in those under or above these ages; and whilst old age does not afford an entire protection from the disease, it is unknown in children under one year.

It occurs with equal frequency in both sexes. Some authors, however, assert that it is more commonly met with in boys than girls.

It is a contagious disease, not usually recurring a second time, subject to epidemic influence, said to be most common in spring and autumn. Its period of incubation varies from eight to twenty-two days.

Being a disease of short duration and of slight intensity, the patient usually recovers quickly both strength and weight. As in convalescence from all acute affections, so with Mumps, the rapidity of recovery from the anæmia, loss of flesh, &c., is proportionate to the age, being quicker and more perfect in young than old people, and is, moreover, determined by the previous health of the patient: if this has been good, the recovery is accomplished perfectly and with rapidity; whilst, on the other hand, if the health has been impaired by excesses of any kind, or by bad hygienic conditions of life, or if the patient be the subject of chronic disease, or of the tubercular or scrofulous diathesis, the restoration to perfect health is much retarded.

Diagnosis.—This is rarely difficult. An acute febrile disease, accompanied with a swelling in, and assuming the shape of, the parotid gland, is diagnostic of Mumps.

Parotid bubo[1] may in some respects simulate this disease. It is, however, a rare affection; almost invariably follows in the course of one of the acute specific fevers. The swelling does not take on the shape of the parotid gland, and quickly gives evidence of the existence of pus in various parts of the swelling. The mamma, testicle, &c., are never affected.

Enlarged lymphatic glands situated in the neighborhood of the parotid perhaps ought to be mentioned, though a careful examination would at once establish the nature of the swelling. Thus there is often more than one enlargement, usually the size of a Barcelona nut, commonly situated immediately in front of the ear. There is no swelling behind the ramus of the jaw; the tumors can be felt to be superficial to the parotid, over which they are mostly movable, and moreover these enlargements of the lymphatic glands are always due to some irritation in the neighborhood, which can generally be discovered.

Treatment.—In common with the other acute specific fevers, Mumps has hitherto failed to be arrested in its course by any mode of treatment at present known. The utmost therefore that can be done is to mitigate the severity of the symptoms, and thus conduct the disease to a favorable termination. Being mostly a disease of trifling importance, but little danger is to be apprehended and but little treatment is required.

All active treatment, whether general or local, is in most cases to be avoided. At the very commencement, an emetic followed by a purgative will be generally useful. The bowels should be kept regularly open, but active purgation should be avoided. By this means the severity of the fever is lessened, for it is well known

[1] My friend Dr. H. Jeaffreson, late Resident Physician to the London Fever Hospital, has supplied me with the following notes concerning parotid bubo. It begins below the ear and behind the jaw, is hard and brawny to the feel, and ill defined. The skin over the swelling is almost invariably of a dusky red color; is immovable over the swelling; in three or four days becomes boggy, and in five or six bursts, and discharges pus. It is by far most common in persons of middle or of old age. It is very rare in children, but may occur at the age of five or six.

Of the acute specific fevers, it almost invariably follows typhus. No case has occurred for some years past at the Fever Hospital after measles, scarlet fever, or typhoid fever. Not more than 3 per cent. resolve. Both parotids may be attacked, and the submaxillary glands may also be attacked in conjunction with, or independently of, enlargement of the parotid. The former condition is by far more frequent than the latter.

that constipation has the effect of elevating the temperature in febrile patients.

In Mumps, as in all fevers, we have an undue elevation of the temperature of the body, due in part at least to increased combustion of some of the tissues. The treatment must be directed so as to control this increase of waste, and supply the loss by appropriate food. This is especially needful with patients whose health previous to the disease was impaired: for in such, a great amount of bodily and mental weakness, lasting for a considerable time, may result.

With this object in view, two chief points should be attended to, namely, rest and the appetite.

Rest and even confinement in bed should be enjoined; for it is found that in all febrile diseases, exercise, both of mind and body, is capable of increasing the abnormal elevation of the temperature. The pain also, which may accompany febrile disease, is much allayed by perfect rest.

The appetite must have strict regard paid to it; for the increased waste of the tissues is compensated for in proportion to the amount of food digested. Should no food be taken or assimilated, the patient is placed in all respects in the position of a starving person, and to this must be added an active, increased consumption of the tissues.

To secure or promote the appetite and proper digestion of the food, attention must specially be paid to pain, sleep, and the nature of the diet.

Pain, if severe, destroys entirely the appetite and arrests the digestion of food; therefore, should the pain in the affected organ be great, appropriate treatment must be employed. For the most part, hot fomentations or poultices are sufficient for the purpose. Should, however, these fail, one or two leeches applied in the neighborhood of the affected organs will generally quickly afford great or even entire relief. This is especially the case when the testicle is affected. The pain is further mitigated by perfect rest of the part; thus the jaws should be moved as little as possible, and the testicle, when it is affected, should be carefully supported.

In regard to the second point—sleep—this can in most cases be attained by easing the pain of the affected part in the manner just described, and by allaying any distressing symptoms that may be present, such as thirst, heat of skin, &c. The thirst can be removed by sucking ice, or the patient may be directed to rinse out the mouth with cold water, or to swallow small draughts of cold water slowly, and at short intervals. It may be much relieved by drinking acid drinks, especially if weak bitter infusions be added. Thus, "whey, or common water acidulated with currant jelly or raspberry vinegar," or a very light infusion of cascarilla acidulated with hydrochloric acid (Graves), will generally succeed. Large draughts of water should be avoided, as they distend the stomach and give rise to annoying sensations to the patient, and because they retard digestion. Effervescing drinks, moreover, by distending the stomach, are apt to distress the patient. Acid fruits will allay thirst, but they must be used with moderation, as they may produce diarrhœa, flatulence, colic, and even nausea.

The disagreeable sensation due to the hot, dry skin, may also increase the restlessness of the patient and thus prevent sleep; this can be allayed by sponging the body with cold or tepid water, one part of the body only being exposed at one time, to prevent the bad effects of cold. Soap may be added with advantage to the water, as it cleanses the skin more thoroughly and removes any smell that may be connected with the cutaneous excretion; this is advantageous, as it is well known that smells of any kind, and especially when disagreeable, lessen the appetite, and may cause headache, nausea, and even vomiting and much depression. And lastly, strict attention should be paid to the diet, both in regard to its nature and the time and method of its administration. The food should be liquid, so that mastication is unnecessary; thus the inflammation in the parotid is not increased by the movement of the parts, but at the same time it should not be too dilute, otherwise digestion is impaired. Thus, good beef-tea, strong mutton or veal broth, gruel, or arrowroot, milk and eggs may be given, the latter with caution, as eggs often disagree with febrile patients. Pounded meat, either cooked or raw, and without spices, are also serviceable. Liebig's beef-tea will in some instances be found especially beneficial; this, indeed, can often be tolerated by the stomach when all other foods are rejected.

In respect of time and manner of administration, it must be borne in mind that the digestion of febrile patients is mostly impaired, and therefore only small quantities of food should be given at one time, and these should be taken at the ordinary meal-times, and at no others. When prostration is great, and the quantity that can be given at one time is very small, the food must be administered at shorter intervals, and sometimes even every half hour.

It is inadvisable that the patient should take to allay the thirst any nutritious food, such as milk, as the appetite for food at the proper time is thus much lessened.

During the grave general symptoms that sometimes occur after the disappearance of the swelling in one organ, and be-

fore another is involved, more active treatment may be adopted, though mostly the disease passes on to a favorable termination.

If the pulse be weak, the surface cool, and the features nipped, chloric ether, musk, wine, and brandy should be given, and warm baths with mustard to the extremities may prove of service

SUDAMINA AND MILIARIA.

By Sydney Ringer, M.D.

Although Sudamina and Miliaria generally occur at the same time, they differ so much in respect of their appearance and the method of their development, that they require separate description.

It may be first stated that, while Sudamina often occur without Miliaria, the latter, on the other hand, are generally accompanied by Sudamina. The probable reason of this will afterwards appear.

Sudamina.—These vesicles are minute and highly transparent, spherical or oval in shape, and often appear deeply seated in the cuticle. They may sometimes be so small as easily to escape notice, or on the other hand, they may measure two lines in diameter at their base.

Partly on account of their minuteness, but chiefly owing to their great transparency, they are apt to escape notice. They are best seen when looked at obliquely, and may often be more easily detected by the touch than the sight.

These vesicles are sometimes widely, if not equally scattered, but at other times grouped and limited to particular portions of the surface of the body. The skin at their base and in their neighborhood is usually unaltered, whereas, in rare cases, a narrow rim of redness is seen around them. They vary greatly in number; being sometimes so few that they can be easily counted, and at other times so numerous that the chief part of the trunk is covered with them. The base of the neck, the neighborhood of the navel, and the sides of the thorax, are the parts mostly affected. They reach their full development in a few hours, remain so for about one day, and then either burst or dry up. When of large size and at the height of their development, they look tense and full, and feel hard and "shotty." As they decline they lose their tenseness, and the cuticle covering them becomes wrinkled and loose, at the same time they may extend laterally, and lose their regular form. If they be numerous, they even coalesce, and hence in rare cases rather considerable patches of cuticle may be detached from the corium beneath, the cuticle itself retaining its continuity. On their disappearance, the cuticle forming them is detached from the surface of the body, leaving the skin entire beneath. If the conditions producing them continue, fresh crops appear, and run their entire course in three or four days.

These vesicles are most commonly limited to the trunk; they may, however, occur on the extremities, but are rarely seen on the face, hands, or feet. Occasionally they become slightly turbid, but they mostly remain transparent throughout. Their contents, watery, colorless, and transparent, are generally acid, occasionally neutral, and very rarely alkaline; they contain chlorides. No organic elements are seen on microscopic examination, with the exception of a few epithelium cells probably derived from their cuticular covering.

They are characteristic of no particular disease, but are produced by sweating;[1]

[1] Sweating is most commonly due either to a fall in the temperature of febrile patients or to general weakness and exhaustion. It especially occurs towards the close of typhoid fever, during the early convalescence of scarlet fever, and in the course of phthisis, this last-named disease being often accompanied by great daily variations in the temperature. In scarlet fever, according to the author's experience, profuse sweating is more common, and lasts longer during the early convalescence of the patient than in other febrile diseases; and hence Sudamina, in very large quantities, are often met with at that period of the malady. Profuse sweating at the commencement of febrile diseases (with the exception of rheumatic fever), when the temperature remains permanently high, indicates great weakness, and thus adds to the seriousness of the prognosis.

In non-febrile persons, if sweating be easily produced by excitement, exertion, or sleep,

and hence they often occur on the decline of fevers, and especially on those "critical days" when the sweating is most profuse.

Much difference of opinion exists regarding the anatomy and the method of production of these vesicles, or Sudamina. According to some authorities they are due to accumulation of sweat in obstructed and distended sweat ducts. Others, Bærensprung for instance, hold that they are produced by the exudation of the perspiratory fluid between two layers of the cuticle, the exudation being caused by obstruction of the ducts from accumulation of effete epithelium cells.

As Sudamina produce no annoyance or symptoms, no treatment is required. They are best prevented by checking as far as possible the sweating that produces them. This sweating as has been stated, is often produced or increased by weakness and impairment of the health, and may be reduced by relaxation from work, out-door exercise, sea-bathing, and tonics.

Dr. Druitt states that the sweating of hectic fever can be controlled for some hours by the sponging of the body with water as hot as can be borne. In many cases of phthisis all treatment fails to lessen the amount of perspiration, for in this disease the perspiration is caused both by the daily fall of the temperature and the exhaustion produced by the disease.

MILIARIA.—The vesicles of Miliaria, in both their appearance and method of development, differ from those of Sudamina. They are like the latter, however, in size, and are produced by perspiration.

They are at first acuminate in form, and round or oval at their base; but in the course of twenty-four hours they sometimes extend and become irregular in outline. They never present the tense rotund appearance of Sudamina. They are almost invariably surrounded at their base by a narrow rim of redness, and the surface on which they are seated is not infrequently somewhat elevated. Their contents are from the first turbid, opaque, and white; are acid in reaction, and by means of the microscope are seen to contain a large number of granular cells, often shrunken-looking, as if badly developed. On the addition of acetic acid, the granules disappear, and there are seen one, two, or three nuclei. The vesicles of Miliaria, when freely exposed, quickly dry up, and a little redness remains for a short time longer. The skin between these vesicles is often mottled with redness, and here and there small red papillæ are seen, on the summit of which, by means of a lens, a small vesicle can often be detected.

Vesicles, in all respects similar to those last described, are not infrequently seen during the eruptive stage of scarlet fever, and they are situated on the papillæ of the skin, which are elevated in this disease. They are formed only when the rash is intense, and on those parts most affected; and are therefore seen most frequently under the clavicles and around the navel. They may, however, occur on the extremities, and are then best developed on the thighs. They are often arranged in elongated groups, corresponding to the furrows of the skin. Miliary vesicles are often abundant on the surface of patients who suffer from rheumatic fever, when sweating is profuse, and especially when this has an offensive smell. Hence, in this disease, they are most developed and abundant in young adults, and are rarer in children and old people. They are probably produced by inflammation. This is shown by the large quantity of cells they contain.

To the author it appears probable that the vesicles of Miliaria are not formed during the act of sweating, but that they result from the irritation which the sweat causes. This view is supported by the following considerations:—The vesicles are especially apt to occur on those parts of the body from which the free evaporation of the sweat is prevented. Thus they are found under the band of the drawers when seen nowhere else; and in cases of profuse sweating, if a piece of flannel be worn for some time, firmly tied round the neck (at which part of the body these vesicles are easily produced), they not infrequently appear—ceasing, however, to be formed when the flannel is removed. Moreover, in rheumatic patients these vesicles are very abundant over the back, at which part the perspiration is confined, and often allowed to accumulate, because of the pain caused in such movement of the patient as would be required for washing this part of the body. They are also most numerous in those rheumatic patients whose sweat is usually offensive and disagreeable. And further, according to the author's experience, they are most apt to occur in other patients when, from impairment of the health, either by overwork, want of sleep, excess of smoking, or other causes, the sweat smells offensively, and when probably it causes greater irritation of the skin.

For the further settlement of this question the following experiments were made: Dry spongio-piline was placed on various

the health is generally impaired. It must, however, be recollected that great differences in respect of the amount of sweating are met with in different individuals. Persons who have returned from tropical climates often continue to sweat greatly on the slightest provocation.

parts of the body, especially round the neck, and kept on the surface several days. By the action of this dry spongio-piline, Sudamina, but in no case Miliaria, were produced. On the same patient, and to the same parts, linseed-meal poultices (which contain much acrid resin) were applied, and vesicles of Miliaria were often produced, and especially on the neck. Bread poultices, which are less irritating, produced these vesicles, but in a much smaller number. In favor of this view may be further advanced the fact that the vesicles of Miliaria are often accompanied by troublesome itching. This is most marked over the back, the part most dependent in rheumatic patients, and it may be very annoying, and prevent sleep.

In the treatment of Miliaria but little is required. Frequent sponging of the surface of the body with soap and tepid water lessens their production, and removes the itching.

VARICELLA.

By Samuel Jones Gee, M.D.

Definition. — A contagious, febrile disease which is attended with an eruption of vesicles, does not last longer than a week, and does not recur in the same individual.

Just as smallpox and measles were not at first distinguished from each other, and mediæval measles included scarlet fever, so were smallpox and chicken-pox confused together until the last century. In the year 1730, appeared what seems to be the earliest assertion of the doctrine of non-identity; we read that "the pestilence can never breed the smallpox, nor the smallpox the measles, nor they the crystals or chicken-pox, any more than a hen can breed a duck, a wolf a sheep, or a thistle figs, and consequently one sort cannot be preservative against any other sort." So far, Fuller. In the well-known paper read before the College of Physicians in 1767, Heberden enumerates what were to him sufficient reasons for disallying the two diseases. Yet since that day there have never been wanting those who have disputed the validity of the distinction drawn; indeed the doctrine of Fuller and Heberden has hardly taken fair root in any country except their own.[1]

Let us review the arguments in favor of the non-identity of chicken-pox and smallpox.

1. Chicken-pox and smallpox are not interchangeable:—

(i.) By infection. (A) There is not a single authentic instance on record wherein either of the diseases was the result of exposure to the infection of the other. (Trousseau.) (B) Chicken-pox may prevail as an epidemic isolated completely from cases of smallpox. (Möhl.) Now, an epidemic of varioloid, free from concurrent examples of non-modified smallpox, has never yet been seen.

(ii.) By inoculation. (A) Chicken-pox is not inoculable. (Bryce, Trousseau.) (B) Smallpox, whether modified or not, inoculated, has never yet been proved to beget chicken-pox.

2. Chicken-pox and smallpox are not mutually prophylactic:—

(i.) Smallpox did not prevent the occurrence of chicken-pox. (Heberden: Halford, quoted by Gregory.) The reader need not be reminded that undoubted smallpox very rarely recurs.

(ii.) Chicken-pox does not prevent the occurrence of smallpox. In the Children's Hospital a girl sickened with chicken-pox on January 17, and communicated

[1] In the first edition of this volume there stood in this place a detailed criticism of the arguments, in favor of the identity of chicken-pox and smallpox, adduced by Hebra, the living Coryphæus of that doctrine. Since Hebra's book has been translated by Dr. Fagge for the New Sydenham Society, the reader who wishes to know what Hebra has to say may easily procure that information. I confess that I was annoyed at one or two of his assumptions, and amused at his logic; I marvelled that a man with so great a reputation should write so loosely. Was Heberden a physician without experience, Gregory a mere apprentice to his art, and Trousseau's opinion unworthy of consideration? If Hebra will confuse varicella and varioloid, he must even do so; however, let it not be said that he does not deny the existence of varicella as a distinct disease. But "quandoque bonus dormitat Homerus" shall cover all faults.

it to the child in the next bed; in April, the girl first spoken of was attacked with modified smallpox (there were good vaccination marks on her arm), attended by severe invasion symptoms (vomiting, headache, backache), which preceded the eruption several days; she recovered; soon afterwards her mother and sister were laid up with smallpox.

3. Chicken-pox and cow-pox are not mutually prophylactic.

(i.) Cow-pox does not prevent the occurrence of chicken-pox; this we see every day.

(ii.) Chicken-pox does not prevent the occurrence of cow-pox. (Abercrombie, Bryce.)

If the very existence of chicken-pox admits of dispute, we cannot be surprised when we find that the published descriptions of the disease present differences and discrepancies without end. No doubt all physicians who have written upon smallpox, from the days of Rhazes downward, have left some notices of chicken-pox; but these are, with a few exceptions, either vague or confused: because up to the beginning of the last century the two diseases in question were regarded as essentially identical; confused, because hardly had a clear separation been made, before the introduction of vaccination, or rather the exaggerated expectations to which vaccination gave rise, led men to thrust into the realm of Varicella every example of varioloid. And even at the present day, the evanescence of the disease and its lack of all gravity militate against better knowledge; the inducements and the opportunities for study are small indeed.

The epithet *chicken*-pox is derived from *cicer* (chick-pea) through the French *chiche*. Varicella (*varicula*) is a legitimate diminutive of *varus*, a pimple.

CAUSES.—The efficient cause of Varicella is "contagion." The disease is readily communicated through the air to a distance of several yards at least; in degree of contagiousness chicken-pox "seems as infectious as smallpox." (Heberden.)

Chicken-pox has never been transmitted by inoculation. Heberden (naturally enough) presumed that the disease was inoculable; the only inference he drew from his failure to inoculate a person who had previously suffered from the disease was that it did not recur. About the end of the last century, the prevailing opinion in France was that chicken-pox could not be so transmitted. The instances of supposed inoculation narrated by Willan are most unsatisfactory; the notion that such transmission might be impossible seems hardly to have crossed his mind. Berard and De Lavit effectively inoculated Varicella, but Varicella which presented the same symptoms, progress, and form of pock, as variola. Heim (quoted by Cross) was not less successful; he took his lymph from umbilicated vesicles which equalled in duration the pustules of smallpox. Next came Bryce, who in 1818 published the result of his attempts to inoculate thirteen persons with the fluid of what, from his description, we may freely admit to have been undoubted Varicella; the operation-wound healed up, and that was all. Lastly, Trousseau has failed in all his inoculation trials.

Chicken-pox is not known to recur. It is a disease of childhood. The following table has been drawn up for me from the records of the Children's Hospital:—

	Under 1 month.	Under 2 months.	Under 3 months.	Under 6 months.	Under 12 months.	Under 18 months.	Under 2 years.	Under 3 years.	Under 4 years.	Under 5 years.	Under 6 years.	Under 7 years.	Under 8 years.	Under 9 years.	Under 10 years.	Under 12 years.	Total.
Boys	2	2	4	29	45	34	36	36	47	44	33	19	10	4	3	1	349
Girls	0	6	9	28	52	28	39	42	53	52	25	11	19	6	2	6	378
	2	8	13	57	97	62	75	78	100	96	58	30	29	10	5	7	727

Judging from the same reports, there seems to have been an epidemic of chicken-pox in 1856.

Adult females are occasionally attacked. (Gregory.)

DESCRIPTION OF THE DISEASE.—1. *Pre-eruptive Period.*—(i.) *Duration.*—It does not exceed four days, and is certainly less than a week (Gregory): it lasts eight or nine days (?) (Heberden): from fifteen to seventeen days (Trousseau): my own observations would lead me to place the duration at about a fortnight. (ii.) *Symptoms.*—There are no symptoms to be noticed before the eruption (Gregory): they are absent or slight (Heberden, Mohl): poorliness, headache, and feverishness precede the eruption by a few hours (Trousseau): cough is sometimes observed.

2. *Eruptive Period.*—The eruption appears within the first twenty-four hours of poorliness in the form of small rose spots, slightly acuminated; from ten to fifteen come out on the first day; they appear on

any part of the body. (Trousseau.) Heberden and J. P. Frank also describe a red spot as the first appearance of the eruption of chicken-pox. According to Bryce and Gregory, the first thing seen is an eruption of vesicles. For my own part, I have always noticed the vesicle to be preceded by a red spot, and such a spot as I should not hesitate to call a papule, but a papule due to mere hyperæmia of the cutis vera, and not to an exudation into it, for tension of the skin causes the varicellous papule to disappear.

On the second day there may be a hundred or a hundred and fifty fresh spots; those of the previous day have the epidermis raised in the form of a bleb, sometimes perfectly round, containing serosity as clear as water; there is no inflammatory areola. (Trousseau.) The vesicles from the first have the size of split pease (Bryce and Gregory); at any rate that size is soon attained or exceeded. The patient has the appearance of having been subjected to a shower of scalding water. (Bryce.) The vesicle is unicellular, not umbilicated, has a very delicate cuticle, and when pricked collapses perfectly; "after the vesicle is emptied, the finger passed over it does not detect any swelling of the cutis vera or the parts beneath." (Bryce, Möhl, Cross, Gregory.) The latter statement seems to me to be much too exclusive; a distinct elevation may often be detected, but is, like the swelling of the rose spot of typhoid fever, dispersible by pressure. The eruption occupies all parts of the body, the hairy scalp not excepted. The shape of the vesicles on the trunk is often oval, the long axis being athwart that of the body. Itchiness is common, and impels the children to rupture the vesicles.

The next morning a hundred or a hundred and fifty new spots will have appeared during the night, the eruption of the preceding day having become vesicular. (Trousseau.) The contents of vesicles which have lasted twenty-four hours become slightly milky; the turbidity, however, is uniform. A slight inflammatory areola appears.

This nocturnal outburst of spots (which become vesicular within ten hours) is repeated for four or five succeeding nights from the beginning of the disease. (Trousseau.)

Many vesicles, as soon as they have attained their full size, get broken, and so encrust at once. Those that remain unbroken present, on the third, fourth, or fifth day of their existence, a small central scab, which quickly attains to the size of the vesicle, and falls in a day or two.

This scab is thin and granular; it falls in fragments, and leaves no enduring redness and no cicatrix. If the vesicle have been subjected to unwonted irritation, the scab may be thick, coherent, and may leave, when it falls, a permanent pit. Gregory never saw a pit left.

3. *Concurrent Symptoms.*—These are of no importance; the tongue is clean, the pulse unaffected, there is no appearance of feverishness. (Heberden, Gregory.) There are outbursts of fever, sometimes violent, for four or five nights, ceasing by day. (Trousseau.) Catarrh is common; it may occasionally be serious, as in the case of a child who was under the care of Dr. West, on account of a chronic swelling of the glottidean mucous membrane; her respiration was therefore somewhat labored at best; by an attack of the chicken-pox she was brought to such straits that for forty-eight hours it seemed as if she could hardly escape tracheotomy; with the cessation of the eruption the urgent dyspnœa ceased.

It is with reluctance that no reference has been made to the name of Willan; yet I think that the reader who consults his book will find it difficult to believe that at least some of the cases which afforded materials for his descriptions were not cases of smallpox, especially those which were admissible into the variety of Varicella coniformis.

4. *Sequelæ.*—An attack of chicken-pox sometimes leaves children in a poor state of health, such as may not be overlooked.

Diagnosis.—Modified smallpox constantly resembles chicken-pox in (i.) the mildness of the symptoms, premonitory and concomitant; (ii.) the scarcity of the eruption, and its character of coming out in successive crops; (iii.) the shortness of the duration of the disease. (Cross.)

Two or three days' high fever, with vomiting, headache, and light headedness, before the eruption, would exclude chicken-pox. The absence of those symptoms would not exclude smallpox.

Although the papule of chicken-pox has a certain elevation, it is something unmistakably different from the peculiar shotty hardness of the papule of smallpox, modified or not. "All cases in which any of the pocks are observed to be indented on the surface, whilst their contents are clear, and before incrustation has commenced, are at once to be distinguished from the water-pox." (Cross.) The base of the varioloid vesicle is hard and raised to a degree never observed in chicken-pox. The pocks of varioloid are not necessarily indented; when not, we must trust to the "greater firmness and less rapid growth, although of equally short duration." "An elevation left after the scab separates determines the question." (Cross.) The commencing scab of Varicella may be mistaken for umbilication. A perfect vaccination scar (it may not be unnecessary to add) often coincides with varioloid.

The characters assigned by Gregory to his "variola varicelloides" are: that it has at least forty-eight hours of premonitory fever; that there are tubercular elevations of the skin; that an umbilication is always present; and that the scabs differ. The last character is comparatively unimportant, the first and third are put too absolutely.

Prognosis.—"No physician has ever seen a child who has died of chicken-pox; fatal complications are quite independent of the exanthematous fever." (Trousseau.)

Treatment.—"Curatur hic morbus quiete animi et corporis, et abstinentia a carne, vinoque." As much as possible, children should be prevented from picking the vesicles and scabs present on the face. Small doses of quinine will be useful during convalescence.

Varieties and Synonyms.—(1) Varicella lentiformis. (Willan.) On the first day of the eruption appear flat red elevations, in the centre of each of which a vesicle is soon formed; the vesicle never exceeds the tenth of an inch in diameter; the scab falls without leaving a scar.

(2) Varicella coniformis (Willan); swine-pox. The vesicles rise suddenly, and have a hard inflamed border; on the second day the surrounding inflammation is more extensive; on the third the fluid is purulent; a permanent scar results from each pock.

(3) Varicella globularis (Willan); hives. The vesicles are larger than in varic. lentif. and the cutaneous hardness less than in varic. conif.

(4) Varicella sine varicellis. (Wilson.)

(5) Varicella solidescens, verrucosa, papularis, variola cornea (Van Swieten), pemphigus variolodes solidescens (J. P. Frank), stonepox, horn-pox, or wart-pox, is a form of true smallpox.

(6) Varicella cellulosa (Cross), pustular umbilicated varicella (Wilson), variola varicelloides (Gregory), are names for that form of modified smallpox which most resembles Varicella.

Synonyma of true Chicken-pox.—Crystalli (Ingrassias); variolæ crystallinæ, spuriæ, volaticæ, benignæ (Morton); lymphaticæ (Sauvages), pusillæ (Heberden); pemphigus variolodes vesiculosus (Frank); varicella bullosa (Cross); water-pox, water-jags (provincial, to dag, daggle = to sprinkle).

The following are some of the best accounts of the disease:—

Wm. Heberden: 1st. Med. Trans. Coll. Phys. vol. i. 1768. 2d. Commentarii, 1802.

Jno. Cross: Hist. of Variolous Epidemic in Norwich, 1820.

Nicol C. Möhl: De Varioloidibus et Varicellis, 1827.

Geo. Gregory: 1st. Cyclop. Pract. Med. vol. iv. 2d. Lectures on Eruptive Fevers, 1843.

A. Trousseau: Clinique Médicale, vol. i. 1861.

SMALLPOX.

By J. F. Marson.

Definition.—Smallpox is a febrile, eruptive, and infectious disease, the product of a morbid poison; which, after a period of latency, causes the development of an eruption on the surface of the body; this passes through the stages of pimple, vesicle, pustule, and scab; and, as a rule, exhausts or destroys the susceptibility to the disease, in the same person, for the remainder of life.

Synonyms.—Jadarí, Arabic; Εὐλογια, Modern Greek; Variola, Latin; Smallpox, English; the Pocks, Scotch; Galra breac, Irish; Petite Vérole, French; Blattern, German; Vaiuolo, Italian; Viruelas, Spanish.

History.—The origin of Smallpox is involved in much obscurity. A great deal of labor and learning have been bestowed in endeavoring to trace the beginning of the disease, but seemingly without any very decided success. The ancient Greeks and Romans do not appear by their writings to have been acquainted with Smallpox, although De Haen, Dr. Willan, Mr. Moore, and Dr. Baron have endeavored to prove the contrary. Dr. Friend, Dr. Mead, and Dr. Mason Good were of opinion that the disease was not known to the Ancient Greeks; and Dr. Adams, in the Appendix to the Commentary on Book Second of his translation of Paulus Ægineta, agrees entirely with the opinion

of Friend and Mead, that the disease was not known to the ancient Greeks.

One of the earliest notices of a disease exhibiting the striking characteristics of Smallpox is to be found in the historical writings of Procopius,[1] who lived in the middle of the sixth century. The disease[2] "began A. D. 544, at Pelusium in Egypt, from whence it spread to Constantinople." This corresponds closely with the era commonly assigned in medical books to the first appearance of Smallpox, viz., A. D. 569, the year of the birth of Mahomet. In that year an Abyssinian army, under Abrahah the viceroy, appeared before Mecca, and was unexpectedly compelled to raise the siege. Several circumstances concur to render it probable that the sudden retreat of the army was owing to the breaking out of Smallpox, and the dreadful mortality which it occasioned. Bruce,[3] in his travels, met with a manuscript account of the war, which confirms this story, and strengthens the opinion that Smallpox first appeared in Egypt and Arabia about the middle of the sixth century."

Paulus Ægineta lived at the end of the sixth or beginning of the seventh century. He professed to have treated, in his seven books, on all subjects connected with medicine and surgery, yet he says not one word to lead us to believe he was acquainted with Smallpox.

Rhazes, an Arabian physician, who flourished about 910, is generally referred to as one of the earliest and best writers on Smallpox. No doubt the disease had existed for some time before he undertook to describe it; indeed, there is evidence in his work that it had, and he alludes to others who had written about it, especially to Ahron of Alexandria, and Messue of Bagdad.

In the East, whence we receive the earliest accounts of Smallpox, there is a tradition—a mere tradition[4]—that the disease in man had its origin from the camel. It is well known that this patient animal is extensively used in Arabia, Egypt, &c., as a beast of burden. If we may venture to reason on the subject from analogy, the tradition is not likely to be correct, inasmuch as all other diseases that have been conveyed to man from the lower animals are not communicable by infection, only by inoculation. When once produced in man, they are still not infectious, in the usual acceptation of the term, as Smallpox is; only producible again, from one to another, by inoculation, as in the instances of cow-pox, glanders, hydrophobia, &c. When Smallpox appeared in this country in the sheep in 1847,[1] we tried to communicate it, by inoculation, to the human subject, and thought we had succeeded in doing so, and the virus was carried on from one to another for several weeks in succession. The pock produced was very like cow-pox, having only, as we thought, a bluer tinge, and was protective against Smallpox, as we ascertained by inoculating the patient afterwards with the lymph of human variola; but we had unfortunately used for the original *ovination* the same lancet, instead of having a new one, as we ought to have had, that we had previously used for vaccinating; and although it was, as we believe, perfectly clean, and free from vaccine lymph, nevertheless, as the disease could not be produced again in the human subject, either by Mr. Ceely, of Aylesbury, who made repeated trials with the lymph of sheep-pox, or by ourselves, the experiment was never brought before the medical profession. Sacco writes of having frequently succeeded, in Lombardy, in transferring the virus of sheep-pox to man, and that it was as successful in protecting against Smallpox as cow-pox is. There has never been any reason to suppose that the Smallpox in sheep has produced by infection any disease in man.

Those readers who are desirous of further information on the early history of Smallpox, will find a great deal of interesting reading on the subject in Moore's History of Smallpox; Willan's Inquiry into the Antiquity of the Smallpox, Measles, and Scarlet Fever; Baron's Life of Jenner; Monro's Observations on the different kinds of Smallpox; and in Dr. Greenhill's translation of Rhazes, forming one of the volumes of the Sydenham's Society's publication.

DESCRIPTION OF SMALLPOX.—The disease is divisible into varieties, which, for convenience, may be described separately.

1. *Variola Discreta;* 2. *Variola Semiconfluens;* 3. *Variola Confluens;* 4. *Variola Corymbosa;* 5. *Variola Maligna;* 6. *Variola Benigna;* 7. *Variolæ Anomalæ*, or irregular forms of the disease, embracing those instances in which Smallpox is complicated with other diseases.

It is called *discrete*, when the pustules stand separately; *semiconfluent*, when they partially coalesce; *confluent*, when they join and run into each other; *corymbose*, when the disease appears in patches; *malignant*, when the eruption, besides being, generally, confluent, the initiatory and

[1] De Bello Gothico, lib. ii.

[2] Gregory, Cyclop. of Pract. Med. vol. iii. p. 735.

[3] Travels to Discover the Source of the Nile, vol. i. p. 514.

[4] Moore's History of Smallpox.

[1] For an interesting and able account of Variola Ovina, or Smallpox in Sheep, see a work by Professor Simonds, of the Royal Veterinary College, London, 1848.

succeeding symptoms are very severe, with hemorrhage from the mucous surfaces, patches of purpura, and discolorations of the skin as if from having been bruised; *benign*, when, although perhaps confluent, the eruption is superficial, and the accompanying symptoms are of a mild character; *anomalous*, when the disease is complicated with other diseases, eruptive or otherwise, as measles, scarlatina, urticaria, &c.—or pneumonia, hooping-cough, bronchitis, disease of the brain, mania, &c.

Smallpox is divisible into four stages:—1. The stage of incubation, which lasts twelve days, from the date of receiving the variolous germ. 2. The stage of initiatory or eruptive fever and invasion, lasting forty-eight hours. 3. The stage of maturation, continuing about nine days. 4. The stage of secondary fever, desiccation, and decline, lasting, of course, an uncertain time; varying according to the severity of the disease.

1. *Stage of Incubation.*—Smallpox appears on the skin on the fourteenth day after the infection of the disease has been received into the constitution, the precise time being after thirteen times twenty-four hours have elapsed from the moment of taking the disease; this time will of course occupy twelve whole days, and part of two others. It is believed by the writer that the time from taking the disease to its appearance on the skin is never longer than fourteen days, and his attention has been constantly directed to the subject for upwards of twenty years. It is true but very few cases afford a decided opportunity for judging of the precise time of incubation, not above one perhaps in fifty or more, but still these few cases are the very cases of value in deciding the point; they have to be watched for carefully to be found. Three or four instances have occurred in which it seemed likely the disease had appeared between the tenth and eleventh days after receiving the infection; they were cases occurring after vaccination, under which condition other stages of the disease are often interrupted, or cut short; still these cases, seemingly decided cases, having been so few they can hardly be relied on: but the others, on the contrary, so many in the aggregate, they can hardly have failed to indicate the true time.

2. *Stage of Primary Fever.*—The ordinary course is this,—after twelve days' freedom from illness, there is severe indisposition for forty-eight hours, and then the eruption of Smallpox begins to appear. This is almost the invariable course. Still it is not invariable. In a few cases, but very few, there is more or less illness all through the period of incubation. The patient has not been so well as usual: experienced even at the time of taking the disease some unpleasant sensation, felt some nausea or giddiness, or sense of alarm, without knowing why it had happened.

3. *Stage of Maturation.*—In distinct and semiconfluent Smallpox the early constitutional symptoms are much ameliorated on the third day, or about that time, when the eruption has been developed on the skin; and the same remark applies with some reserve to confluent cases, but not so completely as to the distinct and semiconfluent forms of the disease; the development of the eruption affords only partial relief in confluent cases. The eruption appears first, usually, on the face, forehead, and wrists, and then on the rest of the body; it is generally a couple of days later on the legs and feet than elsewhere. It is not thrown out at random, without order; it may be observed to be in threes and fives, forming crescents, and in some instances, when it happens that two crescents come together, they form a complete circle. The eruption is at first papular, then vesicular, then pustular, and takes about eight days to arrive at its full development, before the pustules begin to discharge their contents. [A marked characteristic at this stage is the *umbilication*, or depression in the middle of the mature pustule; giving it somewhat the shape of a hat whose crown has been pushed down at the middle.—H.] During the stage of maturation, or concoction as the older authors termed it, there is often considerable swelling of the face and eyelids, so that the patient is popularly said to be blind with Smallpox for a certain time: and there is ptyalism in many cases, and in some a very tender state of the skin, so tender that the patient complains of the pain from the act of merely feeling the pulse; all these may be looked upon as favorable signs of the disease; patients who have the face a good deal swelled for four days, who have pretty free salivation, and a very tender skin, nearly always do well.

4. *Stage of Secondary Fever, Desiccation, and Decline.*—When Smallpox is not of such severity as to destroy life by the eighth or ninth day of eruption, there is a great increase of fever again, called the secondary fever, which is of vast importance, and gives rise to a train of severe and complicated symptoms, which will be described under the heading of *Secondary Fever*. Concurrently with it the pustules discharge their contents, and form dry, scaly scabs, and in favorable cases the disease begins to decline; especially in those cases which will be described under the term *Variola Benigna*, and in modified Smallpox, as it is now frequently seen after vaccination.

VARIETIES OF SMALLPOX.—1. *Variola Discreta*, or *distinct Smallpox*, is a term

applied to that form of the disease in which the pustules stand separately, or apart from each other, and might be readily counted. It is the simplest form of the disease, and is hardly ever attended with danger to life, except in children who may be cutting teeth at the time, and may have convulsions or some affection of the brain, produced, it may be, by the combined influence of Smallpox and teething.

2. *Variola Semiconfluens* is that form of the disease in which the pustules partially coalesce, cannot be said to be distinct from each other, nor yet to run generally into each other. It is readily distinguished in practice. Patients with this form of the disease usually do well; when it proves fatal, the cause is from the combination of circumstances above alluded to in children with the distinct form of the disease; or else from some complication, as erysipelas, gangrene, &c., or, as happens now and then, from the petechial or malignant form of the disease being associated with only a semiconfluent form of eruption. The amount of eruption does not alone destroy life in semiconfluent as in confluent Smallpox.

3. *Variola Confluens.*—This is the form of the disease which destroys the greatest number of persons; the danger in fact arising principally from the amount of pustulation. It is found to prove fatal at the Smallpox Hospital, when large numbers are taken into account, at the rate of 50 per cent. From the first the papulæ are very numerous, countless, and as the disease advances the pustules run into each other, and in the worst cases form one mass of disease. Even the confluent form of the disease may be fairly said to have its varieties. When the disease is but just confluent, and the patient has been previously in good health, with an unimpaired constitution, he will probably recover. Patients do so in fact, as previously stated, at the rate of 50 per cent.; but when the disease is severely confluent, when it is almost impossible to put the end of a pencil between the pustules in many parts of the body, especially on the face,—and such cases are often met with, —a quarter of the amount of pustulation would be enough to destroy life. In cases where the eruption is observed at first to be generally confluent from head to foot, there may be said to be but very little chance for the patient's recovery. The danger is always rendered greater, *cæteris paribus*, when the eruption is very full about the head, face, and neck.

The marked difference, from the first onset of the disease, between the distinct and confluent varieties of Smallpox, cannot fail to strike all observers. In the confluent form the initiatory fever is more intense, there is often delirium, sometimes of a very violent and uncontrollable kind, especially in persons accustomed to live freely, and in those more especially in the habit of indulging in taking ardent spirits. Such persons often require to be put under restraint to prevent their injuring themselves or others. They are impressed often with the belief that they are about to be murdered, and endeavor, accordingly, to escape from control,—and have a tendency to commit suicide; therefore it is desirable to put knives, razors, &c., out of their way. The nervous system is implicated; there are tremors of the hands and lips—a state, in fact, often bordering on *delirium tremens*, produced partly by the Smallpox and partly by the previous habits of living. Draymen, barmen, potmen, tailors, and women on the town, are very unfavorable subjects to be attacked with Smallpox, owing to their habits of indulging freely, and almost daily, in strong drinks. A very large proportion of the patients die who suffer in the early stage of confluent Smallpox from delirium; it should be looked upon in every instance in which it occurs as a very unfavorable symptom.

Fortunately, all cases of confluent Smallpox are not of this dangerous kind. There is the confluent superficial eruption, which often goes through its course without an untoward symptom, especially in persons lately from the country, whose health is unimpaired by the injurious habits and bad air of a town life.

Sometimes the pustules in confluent cases are very large and flat, they do not accumulate well, the edge of them is not well defined, and after they have been out some days they have a tendency to spread out, to become larger. These are dangerous cases, and usually end in death.

On the top of each pustule, or on many of them, a dark spot is formed during the stage of maturation in some cases of confluent Smallpox, and it will be observed, when this occurs, the pustules do not acuminate well, they are rather flat; whenever these signs are noticed, the case should be looked upon as one of great danger, and the patient will most likely die.

Persons of a weakly constitution, those especially with fair hair, have sometimes, about the eighth day of eruption, large bullæ, filled with serum, intermixed with the Smallpox eruption. This is an unfavorable sign; such patients require wine, beef-tea, jellies, &c., early, almost as soon as this symptom is observed.

In some cases of confluent Smallpox there is an absence of the damask rose-red areola described, and very correctly, by the old authors, as surrounding, for a short distance, each pustule of the disease; and, instead of this, the skin between the pustules is generally inflamed

from head to foot. These cases always do badly.

The watery-pock is another dangerous variety of confluent Smallpox. These are, usually, very offensive cases: are accompanied with a good deal of secondary fever: and end fatally for the most part, or else there is a very tedious convalescence.

The eruption of Smallpox is formed on some of the mucous surfaces as well as on the skin generally,—in the mouth, on the tongue, in the nares and fauces, on the membrane lining the larynx, trachea, and bronchi. When the vari are numerous on the larynx and trachea, the danger of the patient is thereby very much increased; they produce a viscid secretion, cough, and a peculiar hoarse, metallic sound in coughing, indicative of their presence in these parts. A constant subject of complaint in most cases of confluent Smallpox is the soreness of the throat. To nearly every patient it is necessary to explain that this inconvenience is caused by the eruption being formed on the roof of the mouth, soft palate, fauces, &c., and that it is impossible to interrupt its course there any more than on the surface of the skin generally. Still the inconvenience goes off considerably in a few days; the vari on the mucous surfaces have a shorter duration than on the skin generally; do not maturate and scab as on the outer skin, from being constantly kept moist by the natural secretion of the mucous surfaces; they never reach beyond the stage of vesicles.

4. *Variola Corymbosa.*—This is a very singular and very fatal form of the disease. It is rather rare. It is called corymbose, from *corymbus*, a bunch or cluster of ivy-berries, &c. *Corymbose* is also a botanical term, applied to a class of plants, the flowers of which are formed in clusters, like those of the carrot. We have gone over the register of the Smallpox Hospital for thirty years, for the purpose of investigating minutely the danger to life in this form of the disease, and find that, in this time, 104 cases of corymbose Smallpox have been admitted—29 in unvaccinated persons, 74 in the vaccinated, and one after inoculation. Of the 29 unvaccinated persons, 13 died, or 44 per cent.; of the 74 vaccinated, 32 died; and, deducting 2 who died of superadded disease, there remains a mortality of 41 per cent. The single case of corymbose Smallpox after inoculation died. It will thus be seen that corymbose Smallpox is, in all cases, a very fatal form of the disease, and brings life into danger nearly as much in vaccinated as in unvaccinated persons, varying only about 3 per cent. The danger in this form of Smallpox often seems to be out of all proportion to the amount of pustulation, which rules so powerfully in other forms of the disease: why it is so, it is impossible with our present knowledge to say, and it is probably one of those things which will for ever remain inexplicable. The disease, as stated, appears in clusters, or, it may be, that only a single cluster is formed, and yet the fatal character before alluded to is given to the disease. In other parts of the body the eruption is perhaps but sparsely scattered, and we might expect the disease to rank in danger with a common semiconfluent case; such, however, is not the fact in practice. It generally happens there are two or three patches, about the size of the palm of the hand, in different parts of the body, in which the pimples are as closely set as could be; and in the immediate neighborhood of each patch the skin is for some distance free from eruption, or nearly so, a few spots only of the disease being formed. There is a great tendency to symmetry in this form of the complaint; when a patch is formed on one arm, or leg, it often happens that a similar patch is formed on the same part of the corresponding limb on the opposite side. In some instances there are numerous corymbose patches in different parts of the body, about the size of a half-crown or five-shilling piece. When these corymbose cases seem to be recovering, very frequently some dangerous complications arise to mar our fair hopes of a successful termination of the malady, and generally, under more favorable circumstances, there is a long and tedious convalescence.

5. *Variola Maligna.*—This truly frightful variety of Smallpox was called by the early writers on the disease Black Pock, or *Variolæ Nigræ*. The symptoms are very formidable at the onset. The blood appears to be poisoned from the first by the disease; it is rendered very fluid and watery. If a portion be drawn from a vein, a large part of it will be found to be serum, and what ought to be crassamentum remains almost fluid; it is principally coloring matter—the fibrin seems to have disappeared. The countenance of the patient is sunken, the breathing anxious, and in some instances death takes place before the eruption has been developed, leaving some doubt about the real character of the disease in the minds of those persons not by practice familiarly acquainted with its varied appearances;—a doubt whether it was Smallpox, scarlet fever, or some other form of idiopathic malignant fever.

The eruption in malignant Smallpox is rather slowly developed. There is hemorrhage from some, occasionally from all, or nearly all, of the mucous surfaces; from the nose, from the mouth, from the air-passages, from the bowels; the urine is high-colored from blood mixed with it.

In the female there is invariably hemorrhage from the uterus, and abortion in cases of pregnancy. The fœtus is usually born dead. Early in the attack there is a patch of effused blood under the conjunctiva, which should always be looked upon as a most dangerous symptom. We have seen blood, in some very rare instances, ooze from the ears and eyes. Livid patches from effused blood are formed on the surface of the body, and blood is mixed with the fluid formed in the Smallpox vesicles, which can scarcely be said to become pustules. There is great depression, but not often delirium—indeed, but rarely; the intellect usually remains clear to the last. A confluent eruption nearly always accompanies the malignant form of Smallpox, and death commonly takes place on the fifth day of the eruption. *Petechial* Smallpox partakes very much of the same characters as malignant Smallpox. Numerous little dark spots, resembling flea-bites, especially about the armpits and groins, are observable, and the skin in these parts has a greenish-yellow hue, very like what we see during recovery from a bruise. The condition of the fluids is no doubt very much the same in these two varieties of the disease; malignant and petechial Smallpox are very nearly akin.

6. *Variola Benigna.*—Van Swieten and others have described a form of natural Smallpox under the title of *Variola Verrucosa*, or *cornea*, stone-pock, horn-pock, and wort-pock, which we sometimes see in these days, and in which the disease is of a mild, modified character. It is ushered in with symptoms as severe as in the dangerous confluent form, but on the third or fourth day of eruption all the severe symptoms begin to subside; the eruption assumes a modified form, such as we constantly see in post-vaccinal cases. We have often had an opportunity of observing this form of the disease in children whose mothers were at the hospital with them, and who knew perfectly well no attempt had been made at vaccination. The pustles are of unequal size, some shrivelling and dying off, while others are maturing: there is no secondary fever, and no pitting. These are examples of mild natural Smallpox, such as have occurred no doubt at all periods to a few favored individuals, and in which, fortunately for the objects attacked, the disease leaves no trace behind.

7. *Variolæ Anomalæ.*—Smallpox is rendered irregular by being complicated with other diseases. We have seen it in conjunction with scarlatina,[1] measles, urticaria, syphilis, bronchitis, pneumonia, phthisis, dysentery, &c. Pregnancy may be mentioned as one of the anomalies; and another, the existence of Smallpox on the fœtus at birth; which must have gone through the stage of incubation, the primary fever, and early days of eruption, before it was born. We have several times seen children who were born with the eruption of Smallpox out on the body, but modified as it is on the mucous surfaces. Mead[1] imagined that persons who were insusceptible of Smallpox had possibly gone through the disease before birth.

First Symptoms of Smallpox, or Primary Fever.—This disease begins with rigors, fever, thirst, headache, sickness at the stomach, sometimes accompanied with vomiting, pain in the back, and general indisposition; followed, after forty-eight hours of illness, by an eruption on the skin of pimples, which are generally observed at first on the forehead, face, and wrists. Among the early symptoms of the disease should be enumerated, as now and then occurring in children, one or two convulsive fits. This occasionally happens in adults also, but not so often as in children. On passing the fingers over the points of eruption some hardness is felt in the skin, as if a grain of mustard-seed, or a small shot-corn, were imbedded in it; but the skin is not tender to the touch at these points, nor does pressure seem to produce any pain. In cases of Smallpox after vaccination, which are so frequently met with in these days, the true or distinctive eruption of Smallpox is very often preceded by roseola, which lasts two or three days—the *roseola exanthematica*—which may lead observers, not intimately acquainted with the early symptoms of Smallpox, to suppose the patient has an attack of scarlatina; but this eruption may be known from that of scarlatina by not being so completely diffused over the skin as the rash of scarlatina usually is; it is also of a lighter, brighter, roseolar scarlet tint, than the eruption of scarlatina, which has a rather dingy hue; and, above all, it has a *mottled* appearance.

Secondary Fever.—Besides the initiatory fever, the fever of invasion in Smallpox, there is what is called the secondary fever, which begins, in confluent cases, about the eighth or ninth day. In the milder cases of Smallpox, secondary fever is hardly perceptible; in the malignant and severely confluent cases death takes place before the secondary fever has barely commenced. But, in most instances of confluent Smallpox, patients suffer more or less from secondary fever, which seems to be the cause or forerunner of a very important chain of events. The

[1] See Med.-Chir. Trans., vol. xxx. Marson, on the Co-existence of the Eruptive Fevers.

[1] De Variolis et Morbillis, cap. iv. edit. 1747.

pulse is increased in frequency, there is thirst, dry tongue, and hot skin; in many cases, particularly in the plethoric, some local inflammation arises, often occurring at the elbow, seemingly from leaning on it when taking food. But cellular inflammation takes place, in different patients, in nearly all parts of the body; sometimes it is deeply seated between the large muscles; twice we have known abscesses formed between the gastrocnemius and soleus muscles, causing intense pain during the formation of the matter. These deep-seated abscesses in Smallpox are sometimes the result of injury received months before. In one of the examples just mentioned, a seaman, in jumping from his ship to the wharf, missed his balance, and fell back on the ship, striking the calf of his leg against the side of the ship; no harm would most likely have followed had he not taken Smallpox or some other severe febrile disease.

Numerous small boils take place in many cases of confluent Smallpox; phlegmonous inflammation in others, involving often the greater portion of a limb. Patients who, from their previous good state of health, just escape dying from the severity of the eruption, at the usual time, viz., from the ninth to the thirteenth day, are very apt to suffer severely from secondary fever, and its consequences; such as pleurisy, pneumonia, ulceration of the cornea, &c.: these are amongst the very serious evils that may be expected. It is difficult to account for this peculiar form of fever in Smallpox; some have imagined it is owing to the absorption into the circulation of the pus formed on the surface of the body. If this were true, we should see, more frequently than we do, the results we recognize as belonging to pyæmia. These results we do see in some instances, but they may be said to be exceptional; whereas secondary fever is the usual consequence of confluent Smallpox. The absorption of some fluid forming part of the eruption, and more readily taken into the circulation than pus is, we strongly suspect, to be the cause of secondary fever, but our knowledge of animal chemistry at the present day is not sufficiently precise to enable any one to say what this fluid is; it is one of the problems for the industrious and ingenious to solve. Secondary fever commences after, just after, the pustules have begun to discharge their contents; it may be that the absorbed fluid is not part of the original secretion, but the product of decomposition, or of some chemical change that takes place after the matter of Smallpox has been exposed to the air. In the horn-pock, as it is called from its hardness, in which there is some modification of the eruption in the advanced stage of the disease, such as we observe frequently after vaccination, the matter is dried up suddenly without being discharged at all: these cases are entirely free from secondary fever; therefore it would seem that the discharged matter has something to do with secondary fever.

Pleurisy is one of the most painful and fatal sequelæ of the secondary fever of confluent Smallpox. Patients are attacked with it very suddenly, and hardly ever recover; it runs a very rapid course, and terminates fatally in three or four days, sometimes sooner. The symptoms are generally at the first of a very decided character; violent pain in the side, wiry pulse, shortness of breathing, great difficulty in drawing the breath, and a very anxious expression of countenance. Such cases are all but hopeless; but we have, in some very rare instances, seen patients recover; they should, therefore, not be entirely given up as past hope; we had a very severe case lately in a young Scotchman, who, after a long convalescence, ultimately got well.

Pneumonia occasionally follows severe secondary fever. It comes on much more insidiously than pleurisy, and assumes the congestive character so well described by Mr. Erichsen, under the term of "Congestive Pneumonia," in *Med.-Chir. Trans.* vol. xxvi. p. 29. It is slower in its progress than pleurisy, and is very likely to have existed two or three days before it is discovered. It is more rarely, perhaps, seen than pleurisy, and is not so uniformly fatal; but it should be viewed as one of the very serious complications occurring in the advanced stage of Smallpox, and very likely to prove fatal.

Bronchitis is another serious complication of advanced Smallpox; dangerous at all times, doubly so when the body is weakened by other exhausting disease.

Glossitis sometimes arises during the secondary fever of Smallpox; the tongue becomes very much swollen and dry, so that the patient is unable to articulate or close the mouth; it is a very distressing and perilous symptom; those attacked with it nearly always die.

Otitis, followed by abscess in the ear, not unusually results from Smallpox. The pain produced during the formation of the matter is very great, but it is immediately relieved on the breaking of the abscess. It is probable that, in some instances, permanent injury may remain in the ear from this occurrence.

The abdomen escapes singularly free from complications in Smallpox. We do now and then meet with peritonitis, but very rarely; diarrhœa more frequently; and sometimes with mucous enteritis in children.

Erysipelas, pyæmia, gangrene, &c., are frequently met with, at times, in hospital practice. They form the most serious

drawbacks to all hospitals, and are, as is well known, fatal in their tendency. These diseases are amongst the complications of Smallpox, and are not confined wholly to hospitals; they are met with in private practice, occurring after severe confluent Smallpox when the disease has lasted a fortnight or more. They are very dangerous. Erysipelas, more particularly of the head and face, occurs more frequently than any other form of superadded mischief. Patients with it, for the most part, get well; but erysipelas occasionally gives rise to pyæmia, which is followed by large abscesses, perhaps bed-sores, hectic fever, and death. The scrotum is apt to become gangrenous after Smallpox, especially in those who have the ill luck at the time to be suffering from gonorrhœa. It is a fatal complication; patients generally die who are attacked with it, but not always. We have several times seen the whole scrotum slough away, and the patient entirely recover; and it is interesting and surprising to see what a good covering is formed afterwards to the testicles, almost as good as before the scrotum was injured.

The women of the town are bad subjects for Smallpox. When they have gonorrhœa, they are very likely to have gangrene of the genitals; and, from their previous irregular habits and spirit drinking, their illness commonly ends fatally. We had a patient two years since with gangrene of the genitals, owing to leucorrhœa; it might, however, have been gonorrhœa; she was barmaid at a large hotel.

Variolous Ophthalmia and Corneal Ulceration. — Conjunctival inflammation often begins on the fifth or sixth day in Smallpox, and continues for a few days, and then subsides under the use of simple remedies. But there is another form of mischief—ulceration of the cornea—which often leads to the loss of an eye; both eyes, fortunately, being but rarely affected, although this does sometimes happen. Formerly a large number of the inmates of the asylums for the blind had lost their eyes from Smallpox. The injury to the eye, by which the organ is destroyed, is not from the pustules of Smallpox forming on the eye, as used to be supposed, but from a destructive form of ulceration beginning almost invariably at the edge of the cornea.

After having been in constant attendance at the Smallpox Hospital for upwards of three years, and having witnessed the great epidemic of 1838, and having seen upwards of 1500 cases of Smallpox, the author was induced to write a paper on Variolous Ophthalmia, which was read before the Westminster Medical Society in 1839.[1] Out of 1500 cases, no instance had then, or for some time afterwards, come under his notice in which the pustule of Smallpox was formed on the eye. It does, however, happen now and then. In nearly thirty years the number of cases of Smallpox admitted into the hospital has exceeded 15,000. Out of this number 26 instances have been noticed in which the primary pustule of Smallpox has formed on the eye. It has not, however, in any one of these instances, injured the eye in any way; the cases have all done well.

In these very rare instances in which the pustule does form on the conjunctiva, it has nearly always been observed to have its seat half-way between the cornea and the inner canthus of the eye, where the conjunctiva is thicker than elsewhere. It has never been seen on the cornea. Now and then it has been observed halfway between the cornea and the outer canthus; the conjunctiva is thicker in this part also than over the eye generally. So that in these very exceptional instances, once in perhaps 500 cases, the pustule of Smallpox does form on the conjunctiva, but does not destroy or injure the eye in the least, so far as has been observed.

The ulceration of the cornea that leads to the destruction of the eye in Smallpox begins after the secondary fever has commenced. It has been observed to begin as early as the tenth day after the commencement of the general eruption, and as late as the thirtieth; the fourteenth day is a common time for it to be first seen. It comes on with redness and slight pain in the part affected, and very soon an ulcer is formed having its seat almost invariably at the margin of the cornea: this continues to spread with more or less rapidity, according to the degree of secondary fever present; in the more violent cases an ulcer being formed on each side of the cornea at the same time, showing the disease to be advancing with great severity, and presenting a tolerably certain indication that the eye will be entirely lost. The ulceration passes through the different layers of the cornea until the aqueous humor escapes; and if the part of the cornea destroyed be large, the iris protrudes through the opening. In the worst cases there is usually hypopyon, and when the matter is discharged the crystalline lens and vitreous humor escape; or the humors may escape from deep and extensive sloughing in the first instance, without the formation of matter; this being succeeded, of course, by the total annihilation of the form of the eye as well as the sight. In some instances the ulceration proceeds very rapidly, the entire cornea being swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least

[1] Medical Gazette, No. 32, May 4, 1839.

pain to the patient, or his being aware that anything is amiss with his eye. This destructive ulceration never goes on rapidly, but when there is a high degree of secondary fever present. That is a point which should be particularly remarked. It is likely to occur when there is a hot and dry state of skin, rapid pulse, thirst, loaded tongue; these having been preceded by a very confluent state of the disease, and the patient has just escaped dying at the usual time, namely, the ninth, tenth, or eleventh day of eruption. Then it is that some serious consequence may be apprehended, such as the loss of an eye, formation of large and deep abscesses, sloughing of the cellular membrane, or, may be, formation of matter in one side of the chest; some of these serious results may be expected when the secondary fever runs high in confluent Smallpox, combined with the circumstances above detailed.

It happens occasionally, unfortunately, that persons have had something amiss with their eyes before Smallpox comes on—some scrofulous tendency, or sensitive state of the conjunctival membrane caused by their occupation. For instance, a chimney-sweep was admitted into the hospital, and his eyes were in such a sensitive state from soot getting into them in the course of his work previously, that he had been several days in the hospital, keeping his head constantly under the bed-clothes, before he would allow his eyelids to be opened. When this was at last accomplished, both eyes were found to be entirely lost from ulceration of the cornea.

Conjunctivitis, rather slow in its progress, begins in some cases during a tedious convalescence as late as the third or fourth week of Smallpox: after it has existed a few days, there will generally be found, on close examination, a small ulcer on the cornea; and, in this advanced stage of the complaint, the ulcer is commonly not at the margin of the cornea, but nearer the centre of it.

DIAGNOSIS.—It is often of great consequence to be enabled to decide as soon as possible on the nature of a febrile eruptive disease, as, in the cases of persons employed in large establishments, servants, etc., in order to their removal for the safety of others; and, on the other hand, it is unjust to the patients themselves to send them amongst Smallpox or fever patients, if they are not suffering from these respective diseases, where they may contract, and even die of, a disease of a far more dangerous nature than the one they may happen already to be suffering from: on this account, early and correct diagnosis is of great consequence to all persons concerned, as well for the credit of the medical practitioner as for the safety of the patient. Upwards of twenty diseases have been mistaken, within the last few years, in the early stage of illness, for Smallpox, and the patients have been sent as having Smallpox to the Smallpox Hospital. It has been observed, however, that three or four diseases mislead much more frequently than others; with the symptoms of these diseases, therefore, it will be desirable to contrast Smallpox. The four diseases are—Measles, febrile lichen, varicella, and some forms of continued fever. Some of the early symptoms of Smallpox are common to the other diseases above enumerated, such as fever, thirst, headache, sickness, and vomiting; but there is in Smallpox what there but rarely is in the other diseases—and when it exists it is accidental, not part of the disease itself—acute pain in the back, evidently not muscular pain.

1. *Diagnosis of Smallpox from Measles.*—Measles is far more frequently mistaken for Smallpox than any other disease is mistaken for it. In Smallpox the eruption follows on the *third day*, or after *forty-eight hours'* illness. In measles the eruption generally appears on the *fourth day*, or after *seventy-two hours'* illness; there is, besides, usually some cough, and lachrymal discharge and fiery redness of the eyes. The eruption, too, of measles, although a little elevated above the surface of the skin, is not so distinctly felt as in Smallpox: it appears more superficial. The lapse of forty-eight hours after the commencement of illness before the appearance of eruption, the pain in the back, and the shotty feel of the eruption on the skin in Smallpox, contrasted with the lapse of seventy-two hours of illness before eruption in measles, the cough, redness of the eyes, and less marked feeling of hardness and prominence on the skin, should be enough, compared with the general appearance of the patient, to distinguish the two diseases.

2. *Diagnosis of Smallpox from Febrile Lichen.*—Febrile lichen is more *like* Smallpox especially, than any other form of disease is, not variolous. At first it must be confessed there is great difficulty in distinguishing between febrile lichen and modified Smallpox; still, however, by attending minutely to some leading characteristics, they may be distinguished: and here again *time* comes materially to our aid; lichen appears on the second day of illness, or after *twenty-four* hours' illness, and the eruption is without the order we observe in Smallpox; it appears scattered at random over the surface of the skin, and begins to appear at first generally on the trunk, as well as on the head and face, which is not the case in Smallpox. Two or three days will always, of course, put an end to any doubts there

may be on the subject, as no fluid, or next to none, is ever found in the eruption produced in lichen.

3. *Diagnosis of Smallpox from Varicella Vera.*—Varicella vera leads to doubt in the minds of many practitioners. The distinction, however, between the two diseases is tolerably easy, and hardly ought to admit of mistakes. The initiatory fever of varicella is but very slight, scarcely perceptible; whereas it is generally rather severe in Smallpox, even where the resulting disease is mild. *Twenty-four hours only* elapse in varicella after the commencement of indisposition before the eruption begins to appear; there is no hardness, as in Smallpox, on passing the fingers over the points of eruption, and no areola at the base of each vesicle, or if any, very slight indeed; in most cases none. The eruption in varicella has its seat just under the cuticle, between the external and deeper layers of the epidermis; is vesicular, as if raised by a shower of boiling water; scattered over the skin without the order of threes and fives together, forming crescents and circles as in Smallpox; there is besides always, or nearly so, what is a very good guide, one or two large vesicles on the shoulders, generally between the shoulder-blades, much larger, and more spread out than the rest of the eruption, wanting the defined edge and hardness of the eruption of Smallpox. In the advanced stage the contents of the vesicles become purulent; but still, those who have watched the course of the disease carefully, cannot well be in doubt as to its real nature, and want of identity with Smallpox.

[The vesicle of chicken-pox scarcely pustulates, as a rule, and never fully *umbilicates* like that of Smallpox. In a very few cases one or two vesicles upon a tender part (as the face) may involve the true skin sufficiently to leave a small mark or pit; but this is quite exceptional.—H.]

Even near the present day,[1] the doubt has not been altogether removed that varicella and variola may be of kindred origin. Heberden first pointed out clearly the distinction between the two diseases. We have no doubt whatever that they are quite independent of each other. Patients admitted with varicella into the Smallpox Hospital have often taken Smallpox during their stay there; and the converse happened a few years since; a child who had been in the hospital with variola was discharged cured and a short time afterwards was readmitted with varicella vera.

4. *Diagnosis of Smallpox from continued Fever.*—The slow insidious commencement of continued fever, with none of the suddenness and violence of attack observed in Smallpox, and the languid and general aspect of the patient in fever, ought always to be enough to mark the distinction between the two diseases, Smallpox and continued fever.

[1] See Thomson on the Varioloid Epidemic of Scotland, 1820.

Prognosis.—In foretelling what will probably be the result of any particular case of Smallpox, the judgment should be guided by the most striking points already described. 1. The quantity of eruption; 2. The age of the patient; 3. Whether or not the mucous membrane of the larynx and trachea seems to be much implicated; 4. The state of the fluids giving rise to the malignant or petechial form of the disease; 5. The state of the nervous system, and previous habits of living; 6. Whether the patient has been vaccinated, and, if so, the number and quality of the cicatrices (to be alluded to particularly hereafter); 7. Whether the disease is complicated with pregnancy; 8. The favorable or unfavorable circumstances in which the patient is placed.

TABLE I.

Showing the rate per cent. of mortality from different forms of eruption in 2654 unvaccinated cases of Smallpox, admitted into the Smallpox and Vaccination Hospital, London, from 1836 to 1851, inclusive.

Unvaccinated Smallpox.	Cases.	Deaths.	Rate per cent. of Mortality.
Confluent . .	1838	937	50
Semiconfluent .	614	51	8
Distinct . . .	202	8	4
	2654	996	37

Note.—Eighty-one of the above patients who died were affected with antecedent, or superadded disease, as well as with Smallpox, viz., Confluent, 58; Semiconfluent, 15; Distinct, 8.

1. Confluent Smallpox is always more or less dangerous. Whenever the disease is confluent, the prognosis should always be very guarded in the early stage of the illness. Unvaccinated patients with this form of the disease die, as shown in Table I. at the rate of 50 per cent. Great confluence about the head and face is always to be dreaded, as patients often die with it when the eruption is but thinly scattered on the rest of the body. When the pustules are flat, do not acuminate well, and when the areolæ around them on the extremities are of a clarety hue, and the eruption on the face is white and of a pasty appearance, the patient has but little chance of recovery. Distinct Smallpox is a disease of but little danger *per se* when uncomplicated with other symptoms of a fatal tendency; as shown in Table I.

it produces a mortality of only 4 per cent.; it hardly ever alone endangers life in the adult.

Semiconfluent Smallpox produces, or is implicated in producing twice the mortality of the distinct form of the disease; it is sometimes, in rare instances, accompanied with symptoms of malignancy, viz., hemorrhage from the mucous surfaces, &c., and may become dangerous from the previous bad habits or shattered health of the patient. Under these circumstances a few deaths take place from semiconfluent Smallpox, amounting to 8 per cent.

TABLE II.

Ages of the unvaccinated patients admitted with Smallpox, at the Smallpox and Vaccination Hospital, London, from 1836 to 1851 inclusive, with the rate per cent. of mortality, calculated at different periods of life.

Date 1836 to 1851.	Age in Years.												Total.
	0–5	5–10	10–15	15–20	20–25	25–30	30–40	40–50	50–60	60–70	70–80	80–90	
Patients	356	334	270	571	669	270	154	18	8	2	1	1	2654
Deaths	181	91	62	154	274	124	89	13	5	1	1	1	996
Percentage of deaths .	50	27	23	26	40	45	57	69		75			37

Note.—About 2 per cent. of the unvaccinated patients died from Smallpox complicated with antecedent or superadded diseases.

2. Age should occupy an important place in the prognosis of Smallpox. Its influence is the greatest in early and in advanced life. See Table II. 50 per cent. die under 5 years of age, and upwards of 50 per cent. beyond 30 years. The least mortality takes place from 10 to 15 years of life.

3. The state of the mucous membrane of the air-passages should be duly estimated; this can be pretty well known by the tone of the voice. When the larynx is much implicated there will be a good deal of cough, and the sound from coughing, and the voice in speaking, will have a hoarse metallic resonance. Laryngeal and tracheal complications render the disease very dangerous.

4. A knowledge of the condition of the fluids is very important in estimating the danger in Smallpox. All symptoms indicating malignancy, and a putrescent state of the blood, should be looked upon as very unfavorable signs. Hemorrhage from any of the mucous surfaces, purpura, blood effused under the conjunctiva, or into the Smallpox vesicles, should all be regarded as very dangerous symptoms.

5. The state of the nervous system is amongst the most important points to be taken into consideration. Cases accompanied in the early stage with delirium generally end fatally. Persons of plethoric habit and free livers are very apt to have delirium, with a nervous, tremulous manner, and sleepless nights, and are very difficult to manage. The irritable temperament is unfavorable in Smallpox. Such persons often worry themselves about the merest trifles, and when they would otherwise do well, but for this irritability, the case ends in death. Delirium coming on for the first time about the tenth day is a very bad sign; such patients nearly always die. Children who grind their teeth hardly ever recover. The prognosis in the above cases should be unfavorable. On the other hand, a quiet state of the brain and nervous system, a tranquil cheerful manner, with hope of recovery, are tolerably certain indications of a favorable result.

6. The patient having been vaccinated will make a most important difference in estimating the danger from Smallpox. If the vaccination has been performed in four or more places, and corresponding cicatrices remain of good quality, readily seen, the case will, in all likelihood, end well. The early symptoms of Smallpox may be very severe, often are so, in well-vaccinated cases, but they subside as soon as the eruption is thrown out, which is usually highly modified, and all goes on well. But there are, unfortunately, many persons who have not had vaccination well performed, and they will suffer from Smallpox, probably, accordingly. When one or two cicatrices can but just be seen, doubtfully seen, the case may be as severe as if there had been no vaccination at all, the eruption pass through its several stages quite unmodified, and the disease proceed, and terminate, uninfluenced in any way by the previous vaccination.

7. Pregnancy is a most unfortunate and dangerous complication in Smallpox. Abortion is very apt to take place. In fatal cases the child is usually thrown off

the day before death. It is generally born dead, but not invariably so. Although the danger in Smallpox is very much increased by pregnancy, and should always be taken seriously into account in forming a prognosis, pregnant patients occasionally do well, especially after vaccination. They sometimes abort, and sometimes do not; sometimes both mother and child do well.

8. The circumstances under which a person is placed in Smallpox may influence very much the result; as, for instance, on board ship; in a small, confined ill-ventilated house; hospitals are especially dangerous to the pregnant woman, witness the mortality in the lying-in hospitals;[1] the prejudices of friends in overheating the patient, and giving cordials and strong drinks at unseasonable times; all these things may interfere with the chance of recovery.

Susceptibility to Smallpox.—Each individual of the human species is born, it would seem, with a susceptibility to contract Smallpox, measles, scarlatina, and perhaps some other diseases, belonging to what is called the zymotic class—those diseases produced by a morbid animal poison. There is in the organism, most likely in the blood, some inborn principle or ingredient, clearly not essential to life and well-being, by which we are rendered liable to undergo these diseases. It is no doubt ordained by an overruling Providence that we should pass through these ordeals, from which hardly any are altogether proof, if they live but long enough, and from which large numbers annually die. After recovery from these diseases, the body is generally in no way better or worse for having passed through the change produced by the diseases, except in those instances in which the person is disfigured by the marks of Smallpox, or the seeds, perhaps, of scrofulous disease are brought into action; or, in the case of the measles, some pneumonic mischief may be left behind; or, in scarlatina, injury to the ears. All the functions essential to life usually go on as well as before, after passing through these diseases; therefore it would seem to be some innate principle in no way necessary to the well-being of the individual which is destroyed, or got rid of, during the attack; a principle or ingredient by which we are rendered liable to undergo these respective diseases. Some persons on exposure escape the infection of Smallpox over and over again, but take it at last. In 1844, a woman, 83 years of age, was admitted into the Smallpox Hospital with severe confluent natural Smallpox, of which she died, who had nursed her own children and her grandchildren with the disease, and had otherwise often been exposed to variolous infection, but never took it before. A similar instance is mentioned by Sir Thomas Watson, in his Lectures, of an old woman who had for years acted as a village nurse, and had nursed a great many persons with Smallpox, but at last, at 84 years of age, took the disease, of which she died.

Some persons have been known to pass through a long life, frequently exposed to Smallpox, but have never taken it; others, late in life, have taken it from inoculation, who had resisted taking the disease in the natural way, as it is called, namely, by breathing an infected atmosphere. Some few resisted, in inoculation days, both inoculation and the natural mode of taking the disease; but these were very rare cases. All periods of life seem to be about equally susceptible to the influence of the contagion. In many parts of this country, before the invention of railways, Smallpox was absent for twenty years together. This happened more especially before the introduction of inoculation. Then, on the disease breaking out among the inhabitants of these but little frequented districts, the infection being conveyed to them by tramps, or dealers in small wares, the disease spread with fearful rapidity, and nearly all who came within the sphere of infection, whether young, or those more advanced in life, since the last invasion, took it, and it caused dreadful mortality, as it does in the present day to the unvaccinated. All ages being taken together, it is found that about one-third, or rather more, of those who take Smallpox in the unprotected state—that is, who have never been vaccinated, or had Smallpox before—die of the disease. It is particularly destructive to the dark-skinned races; the blacks who come to the Smallpox Hospital suffer more from the disease than the native inhabitants of Great Britain. The same thing has been found to take place abroad. Dr. Bulkley, in the American edition of Dr. Gregory's "Lectures on the Eruptive Fevers,"[1] mentions an instance in which a tribe of American Indians took Smallpox, and they all died of it. Every individual of the tribe was swept away.

Infectious Nature of Smallpox.—Boerhaave was the first to point out the infectious nature of the disease. Before his time it was thought to depend on some peculiar influence of the atmosphere, and it is a remarkable circumstance that Sydenham, who paid so much attention to this disease, should have overlooked so obvious a property of it. Most likely it

[1] See Lectures by Dr. Barnes, in the Lancet, 1865, vol. i. p. 141.

[1] Page 34.

is communicable from the moment when the initiatory fever begins. It may be given by the breath of the patient before the eruption has appeared on the surface of the body. It continues infectious so long as any of the dry scabs resulting from the original eruption remain adherent to the body; a single breathing of the air where it is, is enough to give the disease. The dead body, for several days after death, has been known to communicate the disease (see Hawkins, in London Med. Gaz., vol. iii.); and in all probability it would produce the disease for some months afterwards. A few years since a lady was walking at Islington, and met a person with Smallpox; twelve days afterwards she was taken ill, and for a few hours was delirious. The illness passed off without eruption. Her married sister, who had not been out of the house for three months, on account of pregnancy, was seized with illness exactly twelve days again after her sister's attack, which illness proved to be severely confluent but modified Smallpox. The case is singular and very interesting, as showing that the disease may be communicated by a person who had the early symptoms of the disease, precisely at the usual time after being exposed to Smallpox infection, but whose illness passed off without the characteristic eruption; a case in fact of *Variola sine eruptione* as first described by Sydenham.[1]

The infecting source bears no relation generally to the resulting disease; a mild case may, and often does, give rise to a severe one; and, on the contrary, a severe case may produce a mild one. The dry scab of Smallpox would most likely set the disease going months, perhaps years, afterwards by inoculation, just as the dry scab of cow-pox has been found to be effectual for the purposes of vaccination after being kept a considerable time.

Clothes that have been worn by a person when suffering from Smallpox may retain the infection for a long time, as may the furniture, especially woollen furniture, of beds, and bedding, unless washed and thoroughly purified by exposure to the air, &c.

Recurrent Smallpox.—Smallpox but seldom occurs the second time. Instances of second attacks have, however, been recorded from the time of Rhazes to the present day. Thirty years ago we began to collect minute statistical information of all cases of Smallpox admitted into the Smallpox Hospital. At that time there were probably as many persons in this country who owed their protection to having been inoculated, or having had Smallpox, as to vaccination. We have communicated to the Royal Med.-Chir. Soc.[1] the particulars of this inquiry for sixteen years—1836 to 1851. Of 5797 cases of Smallpox, 2654, or 45 per cent. were unvaccinated; 47 cases, or less than 1 per cent. were after a previous attack of Smallpox or Smallpox inoculation; 3094 cases, or 53 per cent., were after vaccination. It will, therefore, be seen that the cases of reputed Smallpox after Smallpox have been but comparatively few, and even some of these would perhaps admit of doubt; lichen, varicella, and some forms of pustular syphilis are difficult to distinguish from Variola, and might easily be mistaken for it, except by those intimately acquainted with the minute characteristics of eruptive diseases. The Smallpox Hospital has been founded 119 years, but there is no record of a patient having been admitted there twice, each time suffering from Smallpox. We have, however, no doubt of the disease occurring a second time, as measles and scarlatina do, but we think the instances are far more rare.

An Irishman, the son of a medical officer in the army, who had been vaccinated in infancy by his father, and had a large cicatrix remaining from the vaccination, and who was attended by his father for Smallpox in early life, and bore decided pits of the disease, in 1844, at twenty-three years of age, was admitted into the Smallpox Hospital with severe confluent Smallpox, of which he died.

We have repeatedly seen the disease modified, when it takes place after natural Smallpox, or after inoculation, just as it is modified by vaccination. Among the circumstances that seem to predispose the constitution to receive a second attack of Smallpox is, as after vaccination, exposure for a time to great change of climate, either hot or cold. Women who have had Smallpox, or have been inoculated for it, often have, when suckling children with Smallpox, a few irregular spots formed on the breast about the nipples; these spots are produced on the breasts by contact with the matter of Smallpox from the child's lips and face. There hardly ever is any accompanying indisposition; the effect is purely local, and cannot properly be considered to be a second attack of Smallpox.

Variola sine Eruptione.—Sydenham was the first to notice a form of fever without eruption, which prevailed at times when Smallpox was epidemic, and which he calls "*Variolous Fever.*"[2] "This Fever originated in that particular epidemic constitution of the atmosphere, which, at the time in question, produced the Smallpox. Hence, with the exception only of

[1] Sydenham, vol. i. ch. 3, sec. 2.

[1] Med.-Chir. Trans. vol. xxxvi.

[2] Sydenham, vol. i. ch. 3, sec. 2. Sydenham Soc. Edit.

those symptoms which were the necessary effects and consequences of the eruptions, it was, if not identical, at least closely akin to the Smallpox. Each disease set in similarly. In each there was the same pain upon pressure over the pit of the stomach. The color of the tongue and the color of the urine were alike in the two complaints. The profuseness and spontaneity of the sweats occurred equally at the commencement of both maladies. The common tendency to salivation was also equal. It occurred during the fever, when its heat and violence reached beyond a certain intensity. It occurred during the Smallpox, when the pustules became confluent. Finally, as the fever was most rife at that particular time when the ravages of Smallpox were greater in these parts than at any other time within the limits of my own observation, there can be but little doubt as to the identity of character between the two diseases. Of this I am certain—all those practical phenomena which determine treatment were the same for the two diseases, with the single exception of the eruption of Smallpox, and of its effects."

De Haen has noticed a similar occurrence as having come under his observation. We have seen a few such cases that confirm the supposition. They occurred after vaccination, and are likely to be more numerous in these days than in the days of Sydenham, as we believe vaccination modifies Smallpox, in different persons, at every stage of its progress. Such cases are not likely to be sent in any large numbers to the hospital, as the eruption is the only decided evidence of the disease being Smallpox. Some like cases occurring in a school, were reported to the Epidemiological Society, in 1852, in answer to a series of questions on Smallpox and vaccination extensively circulated among the medical profession.

M. Hedlund, giving an account of the Swedish epidemic of 1824, states (Magendie, Journal de Physiologie, tome vi.; and Gregory, Library of Medicine, vol. i. p. 303) "that three different forms of disease were then observed, all, as he believes, pathologically allied, viz., true Smallpox, the varioloid, and the fever without eruption. This fever, he adds, began and ended at the same time with the epidemic. The early symptoms were identical with those which preceded the variolous eruption. He considered it as a mild undeveloped Smallpox."

TREATMENT.—There is no specific for the cure of Smallpox. "It is a melancholy reflection," says Dr. Gregory, "but too true, that for many hundred years the efforts of physicians were rather exerted to thwart nature, and to add to the malignancy of the disease, than to aid her in her efforts. Blisters, heating alexipharmics, large bleedings, opiates, ointments, masks, and lotions to prevent pitting were the great measures formerly pursued, not one of which can be recommended. What think you of a prince of the blood royal of England (John, the son of Edward the Second) being treated for Smallpox by being put into a bed surrounded with red hangings, covered with red blankets, and a red counterpane, gargling his throat with red mulberry wine, and sucking the red juice of pomegranates? Yet this was the boasted prescription of John of Gaddesden, who took no small credit to himself for bringing his royal patient safely through the disease. We may smile at this; but if either he, or Gordonius, or Gilbertus, were to rise from their graves and inquire whether this is one whit worse than Mesmerism, or at all more absurd than homœopathy, or hydropathy, we should, I fear, look a little foolish. Let us, then, avoid the errors of our ancestors, without reproaching them."[1]

One of the first things to arrange on undertaking to treat a case of Smallpox should be, if possible, to place the patient in a large airy apartment; bed-hangings, carpets, &c., had better be removed. The room should be kept cool in summer, and agreeably warm in winter, and the air of the room should be changed two or three times a day. In hospitals, the space allowed for Smallpox patients should not be less than two thousand cubical feet for each patient.

For a long time the custom was to keep patients with Smallpox as warm as possible; to heap bedclothes on them, to shut out every breath of air, forbid any ablution, or even change of body or bed linen. All this proceeding must have produced a horrible state of things. To Sydenham we are indebted—and a very great debt we may be sure we owe him—for having revolutionized all this. Like many other reformers of abuses, he was not able to accomplish the change he sought from these abuses without a good deal of obloquy. Thanks to his perseverance, he succeeded. We now use light bed-coverings, frequent change of linen, fresh air, ablutions, and cooling drinks, with the greatest benefit to our patients. To Sydenham we are also indebted for having first drawn the distinction between Smallpox and measles; no very great effort to accomplish, we should perhaps think, for any pathologist of the present day, the distinction seems so clear between the two; yet it was a step in pathology of great importance at the time, as Smallpox and measles had been for centuries looked upon as only modifications of the same dis-

[1] Lectures on Eruptive Fevers. Lect. V. p. 93. American Edition.

ease; just as we until quite lately—thanks to the sagacity of Steward and Jenner—looked upon typhus and typhoid fevers as only modifications of the same fever. Like most other things, it seems easy enough to understand when once it has been clearly explained; but honor and praise are none the less due to the original observers. Easy as it seems, we might not have seen it; most likely we should not.

In the majority of instances it cannot be known for the first two or three days of Smallpox what febrile ailment is approaching; and, even if it were known, the mode of treatment would not materially differ. It will be right to give a dose of opening medicine to relieve the bowels, to keep the patient on simple diet, and to give saline medicine; nothing, generally, is better or more agreeable than citrate of potash; or tartrate of soda, in a state of effervescence. In confluent cases of Smallpox it is necessary to cut the hair close; in the unvaccinated, especially in children, the sooner it is done the better. But in the vaccinated exceptions should be made: to females, especially, it is a great mortification to lose a fine head of hair, which will perhaps take two or more years to restore thoroughly; therefore it will be proper to wait until the *fifth* or *sixth* day of eruption to see if the course of the disease is modified, because if it is, it will not be necessary to cut off the hair.

The diet of the patient should consist of tea and toast, without butter, bread and milk, sop, and oatmeal gruel, grapes, the *juice* of oranges, strawberries, and what patients are very fond of, and can have at all seasons of the year, roasted apples. For drink, toast-water, plain water,—which many prefer to anything else,—lemonade, imperial drink, milk and water, apple water, tamarind water, raspberry vinegar and water, and, what makes a very agreeable drink, some boiling water poured on black or red currant jelly. Sydenham says: "The moment that undoubted signs of Smallpox have shown themselves, I forbid the patient wine, meat, and the open air. His ordinary drink is weak small beer with a toast put in to take the chill off. His food is oatmeal porridge, barley broth, roasted apples, and the like; articles which are neither hot nor cold, and which give no trouble to the digestion. I have no objection to a form of diet that is common in the country, and which consists of a roasted apple mashed with milk, only it must be taken at intervals, moderately, and with the chill off the milk. Hot regimen I forbid altogether. I forbid also all such cordials as are used by some under the rash notion of propelling the pustules towards the skin."

Fifty years ago, and later, it was not unusual to take away blood at the commencement of Smallpox; we never think of bleeding patients now at the Smallpox Hospital. Sydenham, Huxham, and others used to recommend bleeding in Smallpox, but, notwithstanding the sanction of their great names, it must always have been a very doubtful proceeding.

Delirium occurs in confluent Smallpox in persons of very different constitutions—most commonly, as previously stated, in persons of full habit, and free livers; but it also occurs in persons of weakly constitutions, and who may have lived temperately: the pulse is small and weak, and the features shrunken. Such persons require stimulants early—indeed it is about the only chance, doubtful as it is, of affording any assistance towards recovery. This form of delirium should, of course, be clearly distinguished, before giving stimulants, from the delirium of plethora.

Most writers on Smallpox allude to suppression and retention of urine as occurring in this disease; occasionally, perhaps not above once a year, we are told, at the Smallpox Hospital, a patient has not passed urine for several hours; but on examining the bladder there is not any distension of it. At the next visit, on inquiry, we always find urine has been passed. We have not had occasion to pass a catheter, in a case of Smallpox, for five-and-twenty years, therefore we conclude retention of urine must happen but very rarely in this disease.

An invariable complaint in Smallpox is soreness of the throat, more or less; this arises from the eruption being formed there as well as on the skin, which has to be explained to each patient, and it is necessary also to explain that we cannot stop the progress of the eruption there any more than on the skin, that it will go through a certain course in defiance of any means we may use to interrupt it. The spots necessarily cause more inconvenience in the throat, from the conformation of these parts, than on the surface of the body. All we can do for the relief of it is to recommend some mild gargle, or a small quantity of fluid to be taken frequently, or a little red or black currant jelly to keep the parts moist.

The bowels should be at once well cleared, at the commencement of the disease, by a dose, in the plethoric, of three or four grains of calomel, and eight grains of compound extract of colocynth, with or without sulphate of magnesia, and infusion of senna, and for the first few days they should be kept open two or three times a day. In the less robust, and in females, a salts and senna draught alone will, perhaps, do. Afterwards, in the course of the disease, if the bowels act daily without aperient medicine, all the better; if not, they should be relieved

every two or three days by a salts and senna draught. So long as the tongue continue loaded with a brownish yellow fur, the salts and senna draught answers better than anything else; when the tongue is clean a dessertspoonful of castor oil, or a rhubarb and magnesia draught, is more suitable; but so long as the tongue is clean, there is but little need for opening medicine at all; still the bowels should be relieved every few days. Not unfrequently it happens in Smallpox that the bowels are too much relaxed; for this we keep a mixture always in the ward of the hospital, and find it very serviceable:—

℞.—Cretæ preparatæ,
Pulveris acaciæ,
Sacchari albi, āā ʒiss.
Aquæ ʒiv.
Tincturæ opii ʒj.
Spiritûs ammoniæ aromaticæ,
Tincturæ catechu, āā ʒss.
Aquæ menthæ piperitæ ʒiij. Misce.

Two or three tablespoonfuls[1] a dose, to be repeated in *three hours*, whether the first dose seems to have answered the purpose or not; as without the second dose the diarrhœa will often return. If after three or four doses of the above chalk and laudanum mixture the diarrhœa still continue unchecked, having waited a suitable time, say three or four hours, it will be right to give three tablespoonfuls every four hours, of the compound infusion of roses; the sulphuric acid often answers the purpose of stopping the diarrhœa when the chalk mixture has failed, but the chalk mixture so generally affords relief that we always try it first. Should the two forms, above given, fail to stop the diarrhœa, ten grains of pulv. kino comp. may be given every six hours, or a scruple to half a drachm of the pulv. cretæ comp. cum opio. Rice and milk should be given as diet. If the above means all prove to be unsuccessful in stopping the diarrhœa, it will perhaps be found there is some tenderness, on pressure, of the abdomen; then a powder, or a pill, may be given every six hours, composed of three grains of hydr. cum cretâ, and two grains of pulv. ipecac. co.

In the early stage of Smallpox many patients are restless and unable to sleep at night; anodynes fail to procure rest. It may be worth while to try them once to see the effect, and repeat the dose or not as may be judged right. But there is in some patients the same wakefulness in the advanced stage of the disease, in patients who are otherwise doing well; then an anodyne given once or twice, just to get them into the habit of sleeping, answers admirably, and nothing does so well as the hydrochlorate of morphia: we have given it constantly for five-and-twenty years; it procures a comfortable sleep without causing thirst, or stupor, or confining the bowels, as tincture of opium does. It is convenient to keep a solution of it ready, four grains to the ounce: from twenty to thirty minims of the solution is a suitable dose; we generally find twenty-five minims to answer well.

One warning we are desirous of giving about the use of anodyne draughts: they should *not* be given when there is copious salivation and mucous expectoration. Patients at such times are very sleepless, because they require to be kept vigilant to discharge the saliva and viscid mucus frequently, almost constantly; if an anodyne be given under these circumstances, the patient goes to sleep, and the saliva and mucus, which ought to be frequently got rid of, go on accumulating during sleep in the air-passages, and thus the patient dies, gradually asphyxiated by the secretion accumulated in these parts.

Although the antiphlogistic treatment should be continued for perhaps the first few days after secondary fever has set in, patients shortly after its commencement require some additional support; beef-tea or calves'-feet jelly is very suitable to add to their diet, and a glass or two of the lighter wines may be allowed. The next step will be, supposing the patient to be going on pretty well, some soup, with a few shreds of thoroughly done meat in it. So long as the tongue continues furred, a meat dinner does not do well. Perhaps great weakness is complained of, and the appetite is bad; under these circumstances, a grain and a half of disulphate of quinine, with two or three minims of dilute sulphuric acid, and half a drachm of tincture of ginger in an ounce and a half of water, twice a day, will be serviceable. Game, poultry, or lightly boiled eggs might be allowed; and in cases of great prostration, some brandy in gruel at night.

When the tongue has become clean, meat may be recommended, and some ale or porter, with or without wine, port or sherry; care being taken not to try to get on too fast.

Things, unfortunately, do not always go on so smoothly as this; some large collections of matter may form, with sloughing of the cellular membrane, requiring to be opened, or numerous boils harass the patient. It often happens that matter is formed under the scalp, small in amount at first, but it goes on collecting and spreading, and there is no disposition to point and break in this part as in other parts of the body. These collections should be *opened early* to prevent their spreading; the operation is rather pain-

[1] [If the stomach be at all irritable, a tablespoonful every three hours may suffice.—H.]

ful, from the thickness of the scalp. A simple incision does not answer well; the matter collects over and over again, and the cavity of the abscess keeps getting larger; it is better done by a crucial incision, and the cavity should be filled with lint; these cavities are generally very tedious and troublesome in healing; nitrate of silver or a solution of it, freely applied to the interior, helps on the process. Instead of opening these abscesses by a crucial incision, a better plan, perhaps, to adopt is, to pass a seton through them, so that the matter may keep constantly draining away; we often resort to this proceeding with good effect, particularly when the abscess has been allowed to become rather large before anything is said about it.

Some form of steel, with or without quinine, is a useful medicine often, especially to females, during convalescence. Quinine and tinctura ferri sesquichloridi, or mist. ferri comp. should be tried. Cod-liver oil may sometimes be advantageously given, under such circumstances, as it is otherwise found useful, in scrofulous subjects, or those inclined to phthisis.

The discharge from the pustules in some confluent cases is considerable and acrid; the itching, and discomfort produced by it on the skin, are relieved by the application of some absorbent powder freely used; flour applied with a common dredging box answers very well, or hair powder, starch, or calamine, dusted on the face, hands, inside of the shirt and sheets, will be found serviceable.

Many patients have numerous boils resulting from Smallpox; they leave ulcers which are tedious in healing; for some time no process of repair seems to be going on, and the discharge from the ulcers further exhausts the patient. Some decoction of bark, or quinine, with a few drops of dilute sulphuric acid, should be taken two or three times a day; and, in some instances, where the tongue is rather furred, two grains of blue pill, and three of compound extract of colocynth, may be usefully recommended every second or third night for a few times. The ulcers improve dressed with ung. elemi, or ceratum calaminæ; bits of lint, dipped in black wash, and applied to the wounds, and left on a couple of days, seem sometimes to do good. The majority of patients only require the wounds to be covered with bits of strapping.

Some patients like cold, others warm, applications in erysipelas; some prefer flour dredged over the inflamed part; collodion may be tried. If one plan does not make the patient tolerably comfortable, another should be tried. A liberal supply of wine should be allowed; the same in gangrene: both erysipelas and gangrene are generally preceded by bilious vomitings and very often by diarrhœa. During the sloughing of gangrene, at its commencement, nitric acid lotion, a drachm to a pint, may be used with benefit; later, some antiseptic should be applied to the part: one-third liquor calcis chloratæ to two-thirds water; or Condy's fluid properly diluted. Charcoal may be thickly applied, and covered with a linseed-meal poultice. A poultice made of beer grounds some recommend, others have a preference for carrot poultice; we generally trust to the solution of chloride of lime, or Condy's fluid.

When there is any gonorrhœal discharge from the genitals, either in the male or female, the bidet should be used, if possible, twice a day at the least, or some other means should be resorted to to keep the parts affected cleansed; if this be neglected, gangrene of the genitals is very likely to occur. The patient, unfortunately, is not well able at these times to use the bidet himself or herself, and the cleansing of these parts is a very unpleasant office for another to perform for them, and hence it is very likely to be neglected unless the medical attendant is very strict in enforcing his injunctions on the subject of cleanliness, and makes a point of inquiring daily if the bidet has been used.

Pleurisy is one of the most dangerous complications that can arise in the advanced stage of Smallpox. It soon, generally, carries off the patient. Bleeding is useless, if tried, and is, in fact, practically found to do more harm than good, and should be considered as inadmissible. The best plan to adopt—we believe, indeed, the only one we have seen to do good, and we have seen many tried—is at once to put a large blister on the side, and give a full opiate, forty minims of the solution of hydrochlorate of morphia, previously alluded to, or a like dose of tincture of opium; the dose to be repeated in twelve hours unless the pain has very much subsided. Wine, if the patient has been taking any, had better, perhaps, be withdrawn.

Pneumonia, like pleurisy, arising in the advanced stage of Smallpox in a person previously debilitated by an exhausting disease, does not admit of, and certainly will not be benefited by, any active treatment. Very likely, on carefully examining the chest, some consolidation of the lungs will be discovered. A blister should be applied, and five grains of blue pill given every night, or night and morning, for a few days; acetate of ammonia at intervals; an opiate at night, if very restless, and beef-tea as diet.

Bronchitis is another of the dangerous inflammations occasionally met with in the advanced stage of Smallpox, hardly ever admitting of anything but palliative treatment, yet likely to be fatal. Counter-

irritation promises to be useful, and should be tried; and the inhalation of the steam of water, through a proper inhaler, always gives some relief. The lowering system, with repeated doses of calomel, &c., recommended by some writers, does not do well, and should be avoided.

Variolous ophthalmia, and ulceration of the cornea, are amongst the most serious results of confluent Smallpox. Bleeding, here again, used to be recommended, but the practice was bad: we soon saw it did a deal of harm, and was inadmissible. Quite the opposite mode of treatment, in our opinion, is indicated. The patient should be put on as generous a diet as can be borne, and allowed port wine, two or three glasses a day, and take quinine or liquor cinchonæ twice or thrice daily. To the eye, the following application may be made: ℞—Fot. papav. lb. j., pulv. aluminis ʒ j. pro fotu. Ung. cetacei to be applied each night between the eyelids. We sometimes touch the ulcer with nitrate of silver, scraped to a point. The eye should be fixed with a speculum—the one shown in Hey's Surgery answers well—and an assistant should be ready with some olive oil, in a grooved director, to drop into the eye, immediately after the caustic has been applied. Or the ulcer may be touched, by means of a camel's-hair pencil, with a solution of nitrate of silver, a scruple to the ounce.

The conjunctiva, in the advanced stage of Smallpox, often as late as the third week, becomes inflamed, and after a few days a small ulcer may very likely be observed on the cornea. In such cases, as soon as observed, a blister to the temple is nearly always of decided benefit. Perhaps a second may be required—it often is. Should the conjunctival inflammation continue and the ulcer remain stationary, a solution of nitrate of silver, two grains to the ounce, may be dropped into the eye every second day, two or three times, with a large camel's-hair pencil. Should the eye seem irritable, perhaps some vinum opii sine aromat. (to be had at Savory and Moore's) dropped within the lids, once or twice a day, may be serviceable. In nearly all cases, when lotions are not being applied to the eye, a green shade should be recommended to be worn. Scrofulous inflammation occurs after Smallpox, but it will be readily recognized by the great intolerance of light, and by its occurring chiefly in children.

From the earliest periods in the history of Smallpox strenuous efforts have been made to prevent the "pitting" that takes place from this disease. It must be confessed that it disfigures the countenance often terribly, and gives a very common expression to the handsomest face. We need not wonder then at the anxiety of friends, as well as of the patients themselves, that something should be done to prevent, as far as possible, future disfigurement. Some good can be effected, but when the disease is very severe the mischief arising from this cause cannot be wholly avoided. Velpeau recommended some years since that each vesicle should be opened and cauterized with a stick of nitrate of silver scraped to a point; to do good the operation should be performed on the *third* or *fourth* day of eruption. In the most confluent cases, those likely to produce the greatest disfigurement, the proceeding is scarcely practicable: it may be in semiconfluent cases. Mr. Higginbottom recommends the whole face to be washed with a strong solution of nitrate of silver, eight scruples to the ounce of water. We think this is much too strong, and that it will blister the whole surface; if used, half the strength will be enough. A mercurial plaster is used at the Children's Hospital in Paris, the form for which is given by Dr. Aitken;[1] it is a modification and simplification of the emplastrum vigo cum mercurio.[2] It consists of twenty-five parts of mercurial ointment, ten parts of yellow wax, six parts of black pitch. This application has good effect, but is most suitable for use in semiconfluent cases, or those barely confluent, where the patient can be prevailed on to use a little care in the management herself; in severely confluent cases the application would soon be rubbed off by the patient's restless movements.

The application recommended by Dr. Graves, a few years since, of a solution of gutta-percha in chloroform, did no good, and by confining the discharge under the coating of gutta-percha, produced a most offensive condition of the patient. What we do generally is this—wait until the pustules have been discharged, and the discharge has begun to dry, then put on some of the *best* olive oil, or a mixture of one-third glycerine and two-thirds of rose-water; some of this may be applied once or twice a day, for a few days, until the scabs begin to loosen. Cold cream and oxide of zinc, or olive oil and lime water, form good applications; or if the discharge is thin and excoriating, calamine mixed with olive oil. The patient should be warned not to allow the scabs to dry and remain some time on the nose, and other parts of the face, particularly on the forehead and near the end of the nose; when this takes place, the dry scabs themselves leave deep marks in the skin, worse than the eruption of Smallpox itself. The pain of removing the dry scabs is sometimes considerable, and the patient can

[1] Science and Practice of Medicine, vol. i. p. 263. Third Edition.

[2] See form in "Diseases of the Skin," by Erasmus Wilson, F.R.S. 4th Edit. p. 496.

hardly be prevailed on to take them off, or allow others to do so. In common the pits from the eruption are not deep at first, just after the patient has got well, and we may deceive ourselves by thinking our efforts to prevent disfiguration have been attended with considerable success. The disease leaves a peculiar brown stain on the skin at first, which soon wears off, but the pitting is more perceptible a twelvemonth or so after the patient has got well.

One or two warm baths towards the end of the treatment should be enjoined in all cases of Smallpox.

MORTALITY FROM SMALLPOX.—Two circumstances, wholly different in kind, influence very much the mortality from Smallpox, as will be seen on referring to Tables I. and II. These circumstances are, the age of the patients, and the confluent form of the disease. Infancy and advanced age are unfavorable periods for undergoing Smallpox. Children under 5 years of age die at the rate of 50 per cent.; and adults above 30 years die in still larger proportions. See Table II. Patients estimated at all ages, as they come to the Smallpox Hospital, die at the rate of 50 per cent. from confluent Smallpox; 8 per cent. from semiconfluent, and 4 per cent. from the distinct forms of the disease. See Table I. The most favorable time for taking Smallpox is from 10 to 15 years of age: beyond 60 years of age hardly any who take it escape dying.

Sydenham was fully aware of the dangerous day in Smallpox; the eleventh he says, which is the *ninth* day of *eruption*, as shown in the accompanying Table, No. III. The notion of the old authors of the critical days in Smallpox being the 7th, 14th, and 21st, is wholly wrong. The critical days, in fact, are really from the 8th to the 13th day, every one of these days being critical; but death may take place at any period, as seen in Table III.: the extremes being the 2d day in one instance, and the 168th in another. Patients may even die of the severity of the blood-poison from Smallpox before any eruption has appeared on the skin.

The following Table, No. III., formed from the Register of the Smallpox Hospital for 10 years—1855 to 1864—shows the days of eruption on which 987 cases proved fatal: by adding two days to any given number the period of illness may be known. Two-thirds of the fatal cases, it will be observed, took place during the second week of eruption:—

TABLE III.

Showing the days of eruption on which 528 unvaccinated, and 459 vaccinated cases proved fatal, from Smallpox, at the Smallpox Hospital, London, for the ten years 1855 to 1864 inclusive, and occurring amongst 1537 unvaccinated, and 5622 vaccinated cases. All patients having antecedent or superadded diseases of a fatal character have been excluded from the list, so as to represent the deaths from Smallpox alone, as accurately as possible.

	Unvaccinated Cases. Died on the		Vaccinated Cases. Died on the	
1st week	2d day of eruption	0	2d day of eruption	1
	3d	2	3d	2
	4th	9	4th	7
	5th	15	5th	7
	6th	18	6th	17
	7th	27	7th	23
		71		57
2d week	8th	53	8th	32
	9th	67	9th	50
	10th	52	10th	57
	11th	60	11th	64
	12th	52	12th	39
	13th	39	13th	28
	14th	27	14th	36
		350		306
3d week	15th	21	15th	13
	16th	17	16th	12
	17th	8	17th	8
	18th	10	18th	7
	19th	5	19th	8
	20th	6	20th	5
	21st	0	21st	5
		67		58
4th week	22d	2	22d	4
	23d	2	23d	1
	24th	0	24th	4
	25th	3	25th	4
	26th	4	26th	4
	27th	3	27th	3
	28th	7	28th	1
		21		21
	Upwards of four weeks	19	Upwards of four weeks	17
		528		459

Morbid Appearances.—In all cases of death from Smallpox, the skin will exhibit, of course, different diseased appearances, according to the stage of illness at which the person has died. The most striking morbid results of internal parts are those displayed on opening the larynx and trachea, with its branches. These phenomena are peculiar to Smallpox. When the air-passages have been much affected by the disease, and when death has followed on the eighth or ninth day, the mucous membrane is found to be very much congested and inflamed, the epithelium in some instances separated, caused probably, Dr. Petzholdt thinks, by fluid effused between it and the mucous membrane, so as to produce vesication. It is also covered with a very viscid mucous secretion of a brown color. After this is removed, the membrane appears thickened, pulpy, and in some instances ulcerated.

The next most remarkable morbid condition found in the dead from Smallpox, is the state of the chest after pleurisy. This can hardly be said to be peculiar to the disease, because a very similar state occurs after common inflammation. One side of the chest only is affected with variolous pleurisy; except, perhaps, in some very rare instances. The cavity of the chest is found filled with sero-purulent fluid; flakes of coagulable lymph floating in it; adhesions here and there between the pleura costalis and pleura pulmonalis, and the lung on that side of the chest rendered unusable from the pressure of the effused fluid.

Considerable difference of opinion has existed as to whether the pustules of Smallpox are ever to be found on the lining membrane of the alimentary canal. We do not believe that they are; at all events we have never found them there. If they ever exist, it is in some such rare cases as that reported by Dr. Patterson,[1] in which he believes he observed pustules on the mucous membrane of the colon. Sir Gilbert Blane, Rostan, and others have reported cases to a similar effect. The majority of writers on Smallpox believe that ulcerated spots on the mucous membrane of the intestines are due to other causes. Sir Thomas Watson says:[2] "It is affirmed by some writers that the pustules of Smallpox occur in various internal parts of the body, and especially upon the mucous membrane of the intestinal canal. I believe this to be a mistake. The enlarged solitary follicles often put on very much the appearance of pustules."

[1] Edinburgh Monthly Journal of Med. Science, Feb. 1849, p. 549.

[2] Principles and Practice of Physic, 4th Edition, p. 862.

"Many pathologists," writes Dr. Gregory,[1] "have expressed their belief that true variolous pustules have been found in the gastro-enteric mucous membrane. Others, again, among whom may be mentioned Cotunnius, Wrisberg, and Reil (who have paid great attention to the subject), are of opinion that this structure is incapable of developing variolous pustules, and that the appearances so described are in reality inflamed, enlarged, or ulcerated follicles, with petechial patches, similar in all respects to what are found in the common forms of idiopathic or typhoid fever. This pathological principle is fully borne out by the experience of the Smallpox Hospital. We may add, however, that even these appearances are very rare, and that the freedom of the abdominal viscera from urgent symptoms during life, and from all trace of disorganization after death, is a remarkable feature in the disorder. Inflammation may, indeed, originate from accidental causes in any internal organ during the progress of Smallpox, and its effects will be seen after death; but these are not to be confounded with the specific and acknowledged effects of the variolous poison upon the skin and mucous membranes of the throat and chest."

If vesicles of Smallpox are found on the gastro-intestinal mucous membrane, their course must be very similar to that described in the following quotation from Petzholdt, in the Brit. and For. Med. Rev. vol. v. p. 473: "There appear on the lips and inner sides of the cheeks, small white spots, of a round or oval form, the centre of which is very frequently somewhat darker in color. The epithelium is at these places much softened, and at length rises so as to form a small white vesicle, which is at no period transparent, the softened epithelium remaining always opaque and white; it is incapable of any great expansion by the fluid collecting beneath it, and soon bursts. The subjacent mucous membrane is to be seen at some points eroded on its surface. The course of such a pock is, consequently, very brief; the constantly moist state of the mouth rendering its actual filling with pus and desiccation, with the formation of a scab, altogether impossible."

When the lungs have become inflamed during or after the secondary fever of Smallpox, the morbid consequences will be found to be such as are observed after congestive pneumonia.

Bronchitis occurring during the progress of Smallpox leaves results similar to those seen after bronchitis generally. But we believe we have repeatedly observed, both during life and after death,

[1] Library of Medicine, vol. i. p. 308.

the air-passages, larynx, trachea, and bronchi, all or some parts of them,—the larynx and trachea especially,—affected with *erysipelas*, leaving such morbid traces as might be expected after this low form of inflammation.

ANATOMICAL CHARACTERS OF THE VARIOLOUS POCK.—The variolous pock has been carefully examined with a view of describing its structure, by John Hunter, Dr. Adams, Petzholdt, Erasmus Wilson, Dr. Gustav Simon, and others. It is not so easy as might be supposed to fix on the exact spot where the variolous pock first begins to be formed. On examining a piece of skin of a person who has died on the third day of the eruption, there will be found to be patches of a whitish opaque substance between the epidermis and the true skin. These patches adhere firmly to the true skin, but, as Petzholdt says, they may be removed by syringing the part carefully with water, leaving the true skin nearly free from the new substance. If the epidermis, which includes the cuticle, properly so called, and the rete mucosum[1] "be examined when the eruption of Smallpox first breaks out, its undermost layers are found to be softened, almost spongy, and as if filled with a fluid. If a circular incision be made into the skin round the circumference of a papula, this, being loosened by the cut from its lateral connection with the skin, can be removed pretty easily with the pincers, in the form of a little knot. This experiment shows that, at the period of the disease in question, the connection of the cuticle with the cutis is nearly destroyed at those parts of the skin which are affected, whilst a perpendicular section affords us a ready opportunity of satisfying ourselves that there is no cavity beneath the cuticle. During the growth of the pustules, the spongy softening of the cuticle is increased; a still greater quantity of fluid collects between the substance of its lowest layers; there at length arises a small cavity filled with fluid, and, by the increased accumulation of this fluid the cuticle is gradually pushed upwards." "When the thin covering of a part of the skin occupied by a pock is removed, the cutis does not come immediately into view, but it is covered by a substance, varying in color and consistence according to the degree of ripening of the pustule. At the time at which the formation of the cavity or hole described in the preceding paragraph commences, the fluid that covers the cutis is clear; at a later period it is turbid, more tenacious, and at length it becomes pure pus. If all these matters be removed, which is best done by a pretty strong stream of water from a small syringe, so as not to injure any of the subjacent parts, the following appearances can be seen with the aid of a microscope: In all the pocks where pus has formed, there remains some of it behind, which cannot be washed off; and if we employ for these investigations portions of skin that have had their vessels filled with red coloring matter, it can be seen with the naked eye that the pus is, as it were, wedged in between the bundles of vessels, and is retained by them and between them."

The depression in the pocks Dr. Petzholdt thinks is caused by the ducts of the cutaneous glands, which are ruptured as the pustules fill with pus and maturate, but which, in the early period of the eruption, bind down the cuticle to the cutaneous glands, and thus produce the pit or umbilicus.

Sir Thomas Watson says:[1] "Without going minutely into the anatomy of the pustules, you may distinctly see if you closely examine them when they are five or six days old—you may see, at least, in many of them—two colors, viz., a central whitish disk of lymph, set in, or surrounded by, a circle of yellow puriform matter. In truth, there is, in the centre, a *vesicle*, which is distinct from the pus. You may puncture the vesicle, and empty it of its contents, without letting out any of the pus: or you may puncture the part containing the pus, and let *that* out, without evacuating the contents of the vesicle. The vesicles have even, by careful dissection, been taken out entire; and they are said to consist of several little cells. It is most probable that the lymph contained in this separate vesicle is the purest part of the variolous poison."

Mr. Erasmus Wilson writes:[2] "When a well-formed and mature pustule is examined by dissection, it is found to be divided in its interior by a transverse septum into two chambers, both containing pus. The upper chamber is the larger of the two, and they communicate with each other, to a greater or less extent, by the rupture of the transverse septum around its marginal border. The epidermis forming the superficial boundary of the pustule is the segment of a sphere, and continuous by its circumference with the cuticle covering the adjoining skin. The transverse septum is a layer of false membrane, of a whitish color, which was deposited on the derma at an early stage of the pustule. Subsequently this layer becomes separated from the derma, and

[1] British and Foreign Med. Review, vol. v. p. 470.

[1] Principles and Practice of Physic, 4th Edition, p. 865.

[2] On Diseases of the Skin, 4th Edition, p. 489.

raised by the formation of pus beneath it, and at the same time it is broken around its edges, and permits the pus of the deeper cavity to communicate with that already contained in the superficial chamber. In consequence of the peculiarity in the mode of its production, this layer of false membrane generally retains permanently the umbilicated form of the primitive pustule, and is thinner at the centre than towards its circumference. When the septum is removed, the deep chamber is brought into view, and the depressed and sometimes ulcerated base of the pock exposed. The surface of the base is of a bright or purplish-red color, and highly vascular.

"Some difference of opinion subsists with regard to the cause of the umbilicated appearance of the pustule of Variola during its early stages. Dr. Heming many years since attributed it to the perforation of the pustule by the efferent duct of a sebiparous gland. Velpeau, who believes that the principal seat of Smallpox is the follicles of the derma, would, I suppose, entertain the same opinion. Other writers believe it to be produced by the pores of the skin, and Rayer refers it to the attachment of the false membrane. I agree with Velpeau that the follicles of the skin are the primary seat of the vascular congestion, that this congestion gives rise to the production of the papules or vari, and consequently that the epidermal sheath of the follicle is the probable cause of the umbilication of the Smallpox vesicle. When the vesicle is examined at its height of development, it is found to be multilocular in structure, and, when divided by a transverse section, exhibits an appearance which Gendrin has compared to a spice-box, while Bosquet likens it to a severed orange."

Dr. Gustav Simon[1] takes a different view of the subject: "I have found that variolous pustules are not always constituted entirely alike. In many cases where a central depression clearly existed, the epidermis was entirely raised from the subjacent cutis; and only at the spot corresponding with the umbilicus were both membranes united by a thin, whitish cord, which, as the microscope evidently showed, was a hair-sac. Upon the under surface of the epidermis, and for the most part also upon the upper surface of the cutis, was found a thin layer of a whitish mass, which, when looked at with the naked eye, possessed the characters indicated by Rayer;" which are, that "there exists in the Smallpox pustule, between the cutis and the cuticle, a false membrane, which in shape resembles a truncated cone, and has a thickness of half a line." "The layer fixed to the epidermis was not connected with that lying upon the cutis; nor when the latter layer was absent, with the cutis itself; but the hair-sacs, ascending from the cutis to the epidermis, alone connected the two membranes."

"Other vesicles manifested a structure somewhat different from the above. In these also there appeared beneath the epidermis a white substance; but this, at the spot where externally the umbilicus was visible, adhered to the surface of the corium, so that the epidermis seemed fixed to the cutis by this white mass alone. Now as regards the white mass beneath the epidermic covering of the vesicles, and often also apparent upon the surface of the cutis, which most modern writers take for a false membrane, it consists, for the most part, of the deeper softened layers of the cuticle," &c. "As we are accustomed to give the name of false membranes to layers of coagulated fibrine, we cannot include the above-described white mass under this appellation; for, as I have before shown, the epidermic elements always form its major part."

"The fact that the described white mass, at certain spots of the vesicle, is prolonged uninterruptedly from the under surface of the epidermis to the cutis, while at other points this connection is interrupted by the dissociation of the under layers of the epidermis, or by the complete separation of the cuticle from the cutis—this fact, I repeat, is the occasion of the little divisions, or compartments, mentioned by most authors. These are usually of unequal magnitude and irregularly arranged; but sometimes I have seen them arranged with tolerable regularity. In cases of the latter kind, the white mass extended itself from the middle of the vesicle in the shape of little septa, like the rays of a circle, to the periphery, so that six or eight chambers of tolerably equal size were formed. In the middle of the vesicle, from which the irregularly arranged septa proceeded, a hair-sac was sometimes found. Frequently no separate compartments at all existed. In vesicles of this sort the epidermis was connected to the cutis in the middle by a thin white cord, or over a larger space; and around this centre ran a canal, into which the white mass extended, in the form of little clusters or lamellæ; but the canal was at no point interrupted by a complete partition. Rayer appears to have observed the same thing."

The reviewer says:[1] "That the production of an umbilicus is not dependent on the presence of a hair-sac or sebaceous

[1] British and Foreign Med. Rev., vol. iii. 1849, p. 350.

[1] British and Foreign Med.-Chir. Rev., vol. iii. p. 352.

follicle, is easily proved; for it is well known that neither exists in the hollow of the hand and foot: yet in children, in whom the thin epidermis permits it to appear, the central depression is frequently excellently marked. A remarkable peculiarity, first noticed by Rayer, characterizes the variolous vesicle, formed beneath the horny cuticle, which, in the adult, invests the palm of the hand and the sole of the foot. On removing the epidermic cap of the vesicle and wiping off the fluid collected beneath, he observed that in the centre of the denuded corium a little elevation existed, while the circumference was visibly depressed below the level of the neighboring healthy cutis. Dr. Simon has examined these points microscopically, and he finds that the central elevation consists of a file of papillæ of normal or nearly normal size and condition, while the depressed margin is paved with papillæ, bent down or flattened. The explanation is simple. At the centre of the vesicle, an organic connection (from some yet unexplained cause) exists between the cutis and cuticle, and at this point no fluid is effused; but around this centre, exudation of fluid occurs without impediment, and tends to force the cutis and cuticle asunder. In other localities the cuticle yields, and rises everywhere, except at the umbilicus of the vesicle; but there the texture of the cuticle is so dense, and presents such great resistance to the distending power, that the cutis itself yields first, and sinks below the ordinary level." "Of the anatomy of pustules, Dr. Simon remarks, that the pus frequently exists between the cutis and cuticle, the process of suppuration commencing upon the surface of the cutis; but that frequently, also, the suppuration begins in the tissues of the cutis, extending thence beneath the epidermis."

The following is Wedl's view of the subject:[1] "External integuments.—Exudations in this situation are particularly fitted for study, being accessible to observation even during life. The most frequent are those which take place in the cutaneous *papillæ*, in which they are either confined to small limited districts, within which the exudation takes place around isolated groups of *papillæ*, or are more extensive. An instance of the former kind, or of a limited exudation, is afforded in Smallpox, in which the spots are at first filled with a limpid fluid, containing nothing but molecules, and do not become pustules till afterwards, when pus-corpuscles are developed in the hyaline exudation. The transudation takes place from the capillary system of the *papillæ*, the exudation as it is poured out gradually accumulating between the under surface of the *epidermis* and the upper surface of the *corium*. But since the process is confined to limited groups of *papillæ*, the *epidermis* covering the latter is raised in the form of a transparent vesicle, whilst the spot at which the hair escapes from its sheath, together with the excretory duct of the sebaceous follicle, remains depressed, and constitutes the central *pit* of the vesicle. In those parts of the skin where no hairs nor sebaceous follicles exist, as in the palm of the hand and sole of the foot, the exudation deposited around a point where several of the deeper grooves in the *corium* meet, may cause a similar pit, since in a situation of this kind the *epidermis* constitutes a stronger layer, and is of closer texture. When the puriform fluid in the pustules begins to dry up, the pit becomes shallower and wider, owing to the subsidence of the swelling. In the integuments of a subject dead of Smallpox, it is easy to perceive that the vessels of the *papillæ* are more or less injected, when the skin has been macerated long enough to allow of the removal of the *epidermis*, beneath which the isolated patches of vascular injection are immediately apparent. Perpendicular sections show that the *papillæ* are the constant and principal seat of the injection; and it is from their vessels also that the hemorrhage takes place, in cases of petechial Smallpox."

[1] Rudiments of Pathological Histology, by Carl Wedl, M.D. Translated by George Busk, F.R.S., Sydenham Society, p. 206.

Smallpox after Vaccination.—Varicelloid, and Modified Smallpox.

Vaccination was announced to the public by Jenner, in 1798. The then prevalent practice of inoculation for Smallpox gave origin, no doubt, to the idea in the mind of Jenner, of performing a similar operation with vaccine lymph; coupled with the popular belief among the peasantry of Gloucestershire, that once having taken the disease from the cow, in milking these animals, such persons remained ever afterwards proof against the infection of Smallpox. Great hopes were entertained at first, and for some years, that all who availed themselves of vaccination would remain secure against Smallpox for the remainder of their lives. Independently of the popular belief that cowpox afforded protection against Smallpox, it was thought by Jenner and the medical profession generally, that as there was great similarity, if not identity, between vaccinia and Variola, and as Variola was believed to occur but seldom a second time, the same law it was imagined would govern the two diseases—that those who had had the vaccine disease would remain ever after, or nearly so, secure against

Smallpox; that they would enjoy the same immunity as those who had already had Smallpox, or at least as those who had been inoculated for this disease.

Unfortunately this doctrine has not been altogether realized in practice. It has before been shown, p. 139, that at a time when there were probably about as many persons in England who had had Smallpox, or had been inoculated for Smallpox, as had been vaccinated, the numbers of those admitted into the Smallpox Hospital with Smallpox for a series of years, were but as 1 per cent. after Smallpox or inoculation, to 53 per cent. after vaccination. Notwithstanding this apparent drawback of vaccination from the first statements made about it, it was the greatest discovery in relation to disease ever made by man for the preservation of human life. The misfortune was that too much had been promised for it at first. Another great misfortune was, greater perhaps practically than the first, the public were taught to believe that the operation was so simple, and required so little knowledge and care, that anybody might perform it; and thus it has been in a great measure left to chance in England. It has never been taught by appointed teachers until five or six years ago, and even now it has not been taken up in a proper spirit, and treated by those in authority at the examining boards as it ought to be, and from its intrinsic importance really deserves to be. Nearly all that has been done in England, in regard to the more effectual performance of vaccination, and teaching of students, has been done at the suggestion and earnest entreaty of Mr. Simon, the able and zealous Medical Officer of the Privy Council; not, as we might reasonably have expected, by the Councils of our medical and surgical Corporations, who have the supervision and direction of medical education. Great care is given to teaching and learning the capital operations, as they are called, which not one practitioner in twenty through the whole country ever performs; no care, or next to none, to teaching and learning the other, which nearly all, when in practice, will have to perform frequently. Had certificates, fortunately, of having received instruction in vaccination been required of students at the examining boards, as they ought to have been, forty or fifty years since, hundreds of persons who have died, and thousands still living who have been badly vaccinated, and will still die of Smallpox after vaccination, might have been saved. As medical and surgical practitioners, our object should be to save all the lives we can by our art, no matter by what means; and if a little operation —little apparently in practice, but very important in its results—well performed can save many lives, as most certainly it can, and prevent much suffering and sorrow, it should surely always be done with the greatest care, and in the best known way. The success of all operations depends on nice care and management. Operations for hernia and for stone, for instance, if roughly, carelessly, and badly done, end badly; so it is with vaccination: and so far as the public are concerned, it is quite as objectionable to them, no doubt, to die of Smallpox because they have been carelessly and badly vaccinated, as it would be to them to die of hernia or stone because the operations for these complaints, respectively, had been badly performed. In the latter cases, the day of retribution would come immediately; in the former, unfortunately for its correction, it is delayed for perhaps twenty years, or more; otherwise, it would soon be set right. The operation, as an operation, has not been properly estimated from the first introduction of it in England, and it should be looked upon as a blot on our polity that vaccination has been worse performed, *generally*, in England, its birthplace, than in any country in Europe.[1]

Some cases of Smallpox after vaccination were brought forward a few years after its first introduction into practice; and, in 1818, a work appeared by Dr. Monro, of Edinburgh,[2] treating particularly on the subject. In 1819, nineteen cases of Smallpox after vaccination were admitted into the Smallpox Hospital, London, according to the Report of Dr. Ashburner, then physician to the hospital, to the Court of Governors of the hospital held at the end of that year. In 1820 the work of Dr. John Thomson[3] was published, describing the disease which had lately prevailed in Scotland, and naming it a "Varioloid Epidemic." In 1824 Smallpox after vaccination prevailed to a great extent in Sweden. In 1825 Smallpox was epidemic in London, and attacked several persons who had been vaccinated, 147 of whom were admitted in that year into the Smallpox Hospital. In 1828 there was a severe epidemic of Smallpox in Marseilles, when about 2000 were attacked who had been vaccinated. Between July 1831 and June 1836, as reported by Dr. Heim, 955 persons were attacked with Smallpox after vaccination in the kingdom of Wirtemberg, of whom 75 died, or 7·8 per cent. From this period

[1] See Med.-Chir. Trans., vol. xxxvi. p. 381.

[2] Observations on the different kinds of Smallpox, and especially on that which sometimes follows Vaccination. Edinburgh, 1818.

[3] An Account of the Varioloid Epidemic, which has lately prevailed in Edinburgh and other parts of Scotland, 1820.

cases of Smallpox after vaccination have kept gradually increasing in numbers, until they now amount to four-fifths of the admissions into the Smallpox Hospital.

The introduction of vaccination has rendered the diagnosis and course of Smallpox, and consequently the study of the disease, far more intricate than it used to be in former days, and now is in the unvaccinated. The phases of the disease have been made by vaccination far more numerous than they were before. A large majority of the cases of Smallpox occur at the present time (1865) after vaccination. Thirty years since, from 1835 to 1845, the admissions of patients into the Smallpox Hospital were 44 *per cent.* of Smallpox *after vaccination;* from 1845 to 1855, 64 *per cent.;* from 1855 to 1865, 78 *per cent.;* and during the last two years (1863–4), 83 and 84 *per cent.* respectively. It will therefore be seen that they are gradually increasing in numbers; and, now that vaccination has been made by law compulsory in England, the percentage of cases of Smallpox after vaccination will no doubt go on increasing, until it will be rather a rare circumstance to see Smallpox in the unvaccinated, at least in the adult. Unfortunately, the number of admissions into the hospital keeps also increasing, owing principally, doubtless, to the rapid increase of the population of the metropolis, thus :—

Admissions into the Smallpox Hospital,		1835 to 1845—3494.
"	"	1845 " 1855—4546.
"	"	1855 " 1865—7326.

Now there can be no doubt that what has been observed for the last thirty years at the Smallpox Hospital, London, in relation to the occurrence of Smallpox after vaccination, has been going on, in much the same way, all over the country, wherever Smallpox has prevailed.[1]

The disease, as modified by vaccination, received the name of "varioloid," first suggested for it by Dr. John Thomson, of Edinburgh, who wrote a work on the subject in 1820.[2] This word "varioloid" has been adopted generally throughout Europe by writers on Smallpox—in France, Germany, Denmark, &c. &c.—in treating of Smallpox as modified by vaccination. It appears to us not to have been well chosen, inasmuch as the disease is not simply "like" Smallpox, as the name implies; it *is* Smallpox: it will give the disease in the most severe form, in the natural way, by infection, to the unvaccinated, and will produce Smallpox by inoculation just as a case of Smallpox uninfluenced by vaccination will do. There is not a good name, perhaps, for it at present. When the disease is highly influenced, and altered in its course, rendered mild, by vaccination, we call it, at the Smallpox Hospital, *Variola Varicelloides*[1]—Variola like Varicella, which is a better term than "varioloid," the meaning of which is simply, "like Variola;" not a very satisfactory way of explaining what is meant. Perhaps the best term for it is, "Smallpox modified by vaccination," when it is modified; but it is not always modified in persons who have been vaccinated. Besides, there are various degrees of modification, when it is modified; it is not always even like Varicella unfortunately, but often hardly modified at all, and such patients frequently die. Smallpox after vaccination has, in fact, various degrees of severity and modification, from the slightest form in which there is none, or hardly any eruption at all, to the most severe confluent cases, closely, often exactly, resembling the disease in the unvaccinated; and it also assumes the petechial and malignant types after vaccination just as in the unvaccinated state. All this depends in a great measure on the way in which patients have been vaccinated. Those who have been fortunate enough to have been vaccinated in four or more places with lymph that leaves good, easily perceptible cicatrices, have almost invariably a slight form of Smallpox when it occurs; but those who have only one or two marks from vaccination, such as are hardly visible, will probably have a severe form of the disease; and those who have no marks at all are in still worse circumstances. Now, although this rule holds good *generally*, almost *invariably*, still it is not an invariable rule; and perhaps more exceptions will be found—we may say will certainly be found—in those who have been indifferently vaccinated, than in those who have been well vaccinated. Persons seemingly indifferently vaccinated will oftener afterwards have a light form of Smallpox than well-vaccinated persons will have a severe form of the disease. So far it is fortunate. But what we contend for, and always have contended for, is that, if possible, all should be vaccinated in the *best way;* at least the attempt should be made to vaccinate all in the best way, that there should be as little as possible of hap-hazard vaccination, done with a view

[1] See Report of the Smallpox and Vaccination Committee of the Epidemiological Society, ably drawn up by Edward Cator Seaton, M.D., Hon. Sec. to the Committee, 1853. Also a valuable contribution by the same author, "On the Protective and Modifying Powers of Vaccination,"—a pamphlet, reprinted from the Journal of Public Health and Sanitary Review, 1857.

[2] An Account of the Varioloid Epidemic of Scotland.

[1] [Varioloid is the term commonly used in America.—H.]

that if the operation takes effect badly it can be done again. By such a proceeding persons often take vaccination badly, and cannot be made to take it properly afterwards; the imperfect success prevents its taking fully again, and yet some day they may take Smallpox severely, and perhaps die of it. Every effort should therefore be made that there may be as few imperfect vaccinations as possible.

It is, however, undeniable that a few cases occur even to the best vaccinators that do not take the vaccination well. Children sometimes fall ill from other causes just after being vaccinated. There cannot always be good subjects at hand to vaccinate from; the weather and other circumstances interfere with the regular attendance of those who have been vaccinated to afford supplies of lymph for the vaccination of others; but careless and bad vaccinators avail themselves of these untoward circumstances as an excuse for having frequent failures. Great care and great nicety of management are requisite for uniform or almost uniform success in vaccinating, and without the observance of this nice care and management many cases of vaccination do badly, or do not take at all: after two or three failures the friends of a child are led to think it cannot be got to take the vaccination, and they neglect to have further trials made, and ultimately the child takes Smallpox, perhaps severely; such results are of frequent occurrence.

TABLE IV.

Analysis of all the cases of Smallpox after Vaccination, admitted at the Smallpox and Vaccination Hospital, London, for a period of 20 years, viz., from 1836 to 1855, inclusive, showing, from a careful examination of the cicatrices, the relative amount of security given by the number of vesicles produced at vaccination; and, judging from the character of the cicatrices, the probable state of activity and efficacy of the lymph used for Vaccination.

Patients admitted with Smallpox.	Number of Patients.	Character of Cicatrices.	Cases.	Results. Discharged.	Died.	Died affected by superadded disease.	Rate per cent. of Mortality from Smallpox, after deducting entirely the cases affected by superadded disease.
1. Having one vaccine cicatrix . .	2001	good	1032	978	54	15	3·83 } 7·73
		indifferent	969	835	134	21	11·91 }
2. Having two vaccine cicatrices .	1446	good	873	841	32	12	2·32 } 4·70
		indifferent	573	516	57	10	8·34 }
3. Having three vaccine cicatrices	518	good	307	300	7	4	0·99 } 1·95
		indifferent	211	202	9	2	3·34 }
4. Having four or more vaccine cicatrices	544	good	358	356	2	0	0·55 } 0·55
		indifferent	186	183	3	2	0·54 }
5. Stated to have been vaccinated, but having no cicatrix	370	.....	370	269	101	18	23·57
6. Stated to have been vaccinated, but particulars of cicatrix not recorded	17	.....	17	14	3		6·66
	4896		4896	4494	402	86	6·56

Notes.—A good vaccine cicatrix may be described as distinct, foveated, dotted, or indented, in some instances radiated, and having a well, or tolerably well, defined edge.

An indifferent cicatrix—as indistinct, smooth, without indentation, and with an irregular and ill-defined edge.

Aggregate mortality with *good* vaccine cicatrices, from Smallpox alone, uninfluenced by other diseases, 2·52 per cent.

Aggregate mortality with *indifferent* vaccine cicatrices, from Smallpox alone, uninfluenced by other diseases, 8·82 per cent.

There is found to be a mortality of about 2 per cent. in vaccinated as well as unvaccinated patients, from Smallpox being complicated with antecedent or superadded diseases.

From the foregoing Table, No. IV., it will be seen that nearly five thousand cases of Smallpox after vaccination are reported on, each case having been carefully recorded at the time in the hospital register, and the whole subsequently arranged under different headings, so as to show the value of the different modes of vacci-

nating, in persons who have had Smallpox after having been vaccinated several years previously.

Three-fourths of the cases had taken the vaccination in but one or two places, and among these, by far the largest proportionate mortality from Smallpox has fallen; and what should also be strongly impressed upon the memory is, that by far the largest amount of suffering and disfigurement have been produced in those in this category who recovered. It is not simply those who died in the one class of cases that suffered more than others to which we are desirous of drawing the attention, but to those belonging to this class who escaped dying that also suffered and were disfigured, a great deal more than those in another class in whom the vaccination had taken effect in four or more places and recovered. This is very important to keep in mind. By vaccinating so as to take effect in four or more places, we not only save life, but prevent a great deal of suffering and subsequent damage to the appearance of the person, which in females, at least, is of great consequence, and not always quite a matter of indifference to males.

It will be observed that of 544 cases having four or more vaccine cicatrices, only half of 1 per cent. died of Smallpox, or 1 in 200; whereas of 969 cases; with only one indifferent vaccine cicatrix, just upon 12 per cent. died; in each instance antecedent or superadded disease has been deducted, so as to leave the death, as far as could be known, purely the result of Smallpox. Here, then, is a very important practical point to bear in mind when vaccinating: if only one indifferent cicatrix remains from the operation, such persons, taking Smallpox in after life, die at the rate of 12 per cent.; on the other hand, if four or more cicatrices remain, only half of 1 per cent. will die of Smallpox.

It should be further observed, that of 370 persons who believed themselves vaccinated, but who had no cicatrix to show for having been vaccinated, but who trusted to it for their protection, they died of Smallpox at the rate of 23½ per cent. This again is a very important practical point to bear in mind; persons having no cicatrix remaining from vaccination should by all means be urged to be re-vaccinated, else they may very likely some day fall a prey to Smallpox; such persons are in a very unsafe position.

Now, what has been going on in London for years past, as seen in patients who have been admitted with Smallpox at the hospital, has been going on also, there can be no doubt, all over the country, in persons who have gone through the disease elsewhere—a very large majority of the patients admitted at the hospital are not Londoners, but persons who have come to London from the country, to serve in the capacity of servants, or are employed in different ways of business. They were vaccinated in the country.

The security of vaccinated persons will be seen by Table IV. gradually to rise, not only from the number of cicatrices produced at vaccination, but also according to the *quality* of the cicatrices, agreeing with the description given, in the notes of the Table, of the characteristics of good and indifferent cicatrices. Active vaccine lymph, such as leaves clear permanent cicatrices, is evidently indicated as the most desirable to select for use in vaccinating. Another recommendation is, that it takes effect more readily at the time of using it. Long humanized lymph requires a more dexterous hand to produce anything like uniform and perfect success.

In the notes of Table IV. it will be seen that the aggregate mortality in persons who have Smallpox is much less in those with good, than in those with indifferent vaccine cicatrices: thus taking all the cases together, regardless of the number of cicatrices, those with *good* cicatrices died at the rate of 2½ per cent. only; whereas those with *indifferent* cicatrices died at the rate of 8¾ per cent.: this is also a very important subject to remember, carrying with it, as it does, the increased amount of danger and suffering when Smallpox arises. One really good, circular, radiated, and indented cicatrix, is worth two or three indifferent cicatrices generally, such as can hardly be seen. With good, active, eighth-day lymph, an expert vaccinator will hardly ever fail; certainly not above once in 150 times. We may, indeed, confidently state, from those on whom we can rely, and from our own experience in vaccinating, that the failures will not be nearly so often as once in 150 times; they should be very rare occurrences indeed, in good hands.

In the course of years, vaccine lymph becomes humanized, by passing many times through the subject, and can only be kept in a good state of efficiency by having many subjects constantly to select from for its continuance, and even then the cicatrices it leaves, after many years' use, are not so good as they were formerly; this is a point on which we can bear witness from our own experience, and it is shown by the above Table how very important it is to have lymph that leaves good permanent cicatrices.

Out of large numbers of cases of Smallpox after vaccination, viz. 1958, admitted into the Smallpox Hospital during the years 1863 and 1864—Smallpox having been epidemic in London throughout these entire years—the mortality after vaccination shows a considerable increase.

viz. from 6·56 per cent. as given in Table IV., for twenty years—1836 to 1855—to a mortality of 9·2 per cent. out of 1958 cases for the years 1863–4; all patients manifesting antecedent or superadded diseases having been deducted from the calculation as in the former instance, in making Table IV.

It is a question that may be fairly and properly entertained, and deserves very mature deliberation, whether we ought not to resort more frequently than has hitherto been done to supplies of lymph from the cow. A good stock should not be carelessly given up; with many subjects constantly to operate on, and choose from, it can be kept for many years in a good active state of efficiency; and, however many may be vaccinated, the lymph can only make the circuit of the human body fifty-two times every year, yet this multiplied by twenty or thirty, as the case may be, according to the number of years, makes the total number considerable. The able and interesting researches of Mr. Ceely of Aylesbury,[1] by which he established the practicability of obtaining lymph from the cow, by inoculating these animals with the virus of human Smallpox, has secured to us the means of procuring fresh supplies of vaccine lymph should it be difficult to meet with the disease in its natural state in these animals.

Mr. Badcock, formerly of Brighton, now of Camberwell Grove, informs me that he has succeeded thirty-seven times during the last twenty-five years, in inoculating the cow with the virus of human Smallpox.

The subject of deterioration of vaccine lymph will be of course a disputed point. It will, perhaps, be argued that diseases of this class do not alter; that they remain perpetually the same; that Smallpox, measles, scarlatina, &c., are the same now as they were originally, and ever will be. This argument may be regarded as true generally, but it should be remembered that vaccinia is not genuine Variola, even when we know the disease has been produced in the cow by inoculation with the virus of human Variola. In passing the disease through the cow it thereby undergoes alteration, and is not afterwards communicable to man by infection as true Variola is; nor is vaccinia one of the diseases natural to man, but is only taken by him through the agency of inoculation: therefore, on this account, it is likely from a variety of circumstances to undergo degeneration, as it is a disease produced at will—artificially—not taken when the body is most prone to receive it, as we may suppose the body is when disease is taken in the natural way. We feel bound, however, to state we have frequently produced, lately, with lymph brought into use by Jenner more than fifty years since, vaccine vesicles which, on comparison, exactly correspond with the vesicles sketched in Jenner's original work, explaining and illustrating the vaccine disease; but we also feel bound to state for consideration, reflection, and practical deduction, the facts with regard to Smallpox after vaccination which have come before us at the Smallpox Hospital for the last thirty years.

According to a statement of Dr. Sanderson,[1] as reported to him by the local medical practitioners, the cows still suffer from the true cow-pox every spring, or nearly so, in the neighborhood of Berkeley, Gloucestershire, the birthplace of Jenner.

Every now and then a patient is admitted into the Smallpox Hospital who has been vaccinated *after* the symptoms of Smallpox have appeared, the disease being known to be Smallpox when the vaccination was performed. Several patients were thus admitted a few years since from one of our largest west-end London parishes These vaccinations must have been done under a total misapprehension of the powers of vaccination to control Smallpox. It does no good whatever when so performed, and should not be repeated, as it only tends to bring vaccination into discredit with the public. The facts of the case are these:—Vaccination to be effective should have gone on to the stage of areola before there is any illness from Smallpox. It has before been stated that when Smallpox has been taken into the system there is twelve days' freedom from illness generally, forty-eight hours' illness, and then the disease begins to appear on the skin. The areola of vaccination is not fully formed until the ninth or tenth day of the progress of the vaccine vesicles, on those who have never been vaccinated before; so that unless there has been time for the areola to be formed after the vaccination, before the illness produced by Smallpox begins, the vaccination will not be of the least benefit. The progress of vaccination is generally—not always—interrupted as soon as the illness from Smallpox commences, but as it never can be exactly known when Smallpox is taken, so long as persons are well in a house where it exists it will be right to vaccinate or re-vaccinate, as the case may be, as soon as possible, all likely to contract the disease; but this is a very different thing from vaccinating *after* the

[1] Observations on the Variolæ Vaccinæ, and Variolation of Cows, by Robert Ceely, Esq., Surgeon to the Buckinghamshire Infirmary. In Transactions of the Provincial Medical and Surgical Association, 1840.

[1] Sixth Report of the Medical Officer of the Privy Council, with Appendix, 1863, p. 213.

symptoms of Smallpox have actually commenced. Example:—Suppose an unvaccinated person to inhale the germ of Variola on a Monday: if he be vaccinated as late as on the following Wednesday, the vaccination will be in time to prevent Smallpox being developed; if it be put off until Thursday, the Smallpox will appear, but will be modified; if the vaccination be delayed until Friday, it will be of no use, it will not have had time to reach the stage of areola, the index of safety, before the illness of Smallpox begins: this we have seen over and over again, and know it to be the exact state of the question. Re-vaccination will have effect two days later than will vaccination that is performed for the first time, because re-vaccinated cases reach the stage of areola two or three days sooner than in those persons vaccinated for the first time. Four or five years since, Smallpox occurred in a family at Richmond, consisting of a man and his wife, and their niece. We received the account of the occurrence from the man, the only one of the three admitted into the hospital. The niece was the first attacked with the disease, and died. About a fortnight afterwards the woman was attacked, and died; both at Richmond. During the woman's illness the man was vaccinated, and he had five large vaccine vesicles on his arm, *without any areola*, on his admission, in the early stage of Smallpox, at the hospital. The progress of the vaccine vesicles was arrested by the Smallpox; the man went through confluent Smallpox, wholly unmodified, and died. Now had the vaccination been performed one day sooner, the Smallpox would have been modified—two days sooner, and the man would have been saved from it altogether: this shows the necessity of performing the vaccination as promptly as possible in houses where Smallpox exists. Three weeks or a month had been lost after the first outbreak of Smallpox in the house before the vaccination was performed on this man.

Re-vaccination.—It is found on examining large numbers of persons attacked with Smallpox after vaccination, that the majority of those attacked are from eighteen to twenty-five years old, and that they had been almost invariably vaccinated in infancy.[1] This may be partly accounted for, no doubt, by persons of this age coming to London from the country to act as servants, in shops, &c. But it would seem that all persons at this age become more liable to take the disease then than earlier, or later in life; they become, in fact, more susceptible to it at this age than sooner after vaccination. It would, therefore, seem to be a wise course to pursue to recommend all persons on reaching adult age, especially if about to change their place of residence, to be examined as to their probable security against Smallpox. If they have four or more good cicatrices from vaccination they are tolerably safe; if, on the other hand, they have but one cicatrix, and that such as can hardly be seen, or no cicatrix at all, such persons had better be re-vaccinated as a matter of precaution. These remarks apply especially to persons on passing from one part of the world to another, more particularly if the climate be very different from the one where they have been living. Any change from either a hot to a cold, or colder climate, renders persons liable to contract Smallpox. Persons coming from India to England should be re-vaccinated. Vaccination, judging from the cicatrices, does not appear to take effect so well in India as in England. At certain seasons of the year there, during the hot season, the vaccine lymph is said to suffer deterioration. However this may be, on examining persons who have been vaccinated in India, the cicatrices left by vaccination will generally be found to be very indifferent. Further, it would seem, that persons who have been inoculated for Smallpox or have had Smallpox, are liable to be attacked with this disease a second time, under the circumstances mentioned, and that a predisposition to contract the disease is renewed by any great change of climate, either to a hotter or colder temperature.

In commendation of re-vaccination we may state that but very few patients have been admitted with Smallpox into the Smallpox Hospital, who stated that they had been re-vaccinated with effect, and that these few have had Smallpox in a very mild form. For just upon thirty years we have re-vaccinated all the nurses and servants who had not had Smallpox, on their coming to live at the Smallpox Hospital, and not one of them has contracted Smallpox during their stay there. Re-vaccination has been extensively practised for some years past, at stated intervals, and seemingly with good effect, on the troops of some of the foreign armies, more especially and perseveringly on the armies of Germany. Combining all these circumstances together, we therefore feel perfectly justified in recommending re-vaccination for extensive adoption, after adult age, in England; especially to persons who appear, from their cicatrices, to have been but indifferently vaccinated in infancy.

For a very masterly summary of the effect produced by vaccination on the inhabitants of different parts of the world, and a thorough investigation into the whole subject of vaccination and Smallpox, at home and abroad, we earnestly recom-

[1] See Med.-Chir. Trans., vol. xxxvi. pp. 377, 380.

mend to all persons interested in vaccination, the perusal of the Blue Book, by John Simon, Esq., Medical Officer of the Privy Council, entitled "Papers relating to the History and Practice of Vaccination. 1857." It is to be had of Messrs. Longman and Co.

Epidemic Diffusion of Smallpox.—Epidemics of Smallpox begin generally, in London, in the autumnal period of the year. The moist weather of the autumn seems to be favorable to the spread of the disease. When, however, it has once been set going, it continues often throughout all the seasons of the year, quite uninfluenced either by the frosts of winter, or the heat of summer. A larger number of patients have, on several occasions, during epidemic outbreaks of the disease, been admitted into the Smallpox Hospital during the month of May than in any other month of the year. The present epidemic of Smallpox has lasted an unusually long time; it has now (September, 1865) been going on continuously for three years. From 1796 to 1825 there was not any epidemic of Smallpox in London. After that came the epidemic of 1838. Since then epidemics have followed in rapid succession. There was one in the winter of 1840–1. Smallpox was epidemic again in 1844–5, in 1848, in 1851–2, in 1854–5–6, in 1859–60, and in 1863–4–5. The epidemic of 1844 began in May; all the rest had their commencement in the autumnal period of the year. We are quite unable to explain why the disease becomes at times epidemic, then culminates, and then declines; nothing has yet occurred to lead us to the solution of this question. The decline would not seem to be for want of subjects to act on, as of late years fresh epidemics have commenced two or three years only after the preceding epidemics had ceased, and the disease has attacked numerous persons of adult age who were residing in the same place when the disease was raging there but a short time before, but who missed taking it then. A probable explanation of this would be that during the epidemic that had just before passed, these persons were not so susceptible to the influence of the variolous poison, although they became so a short time afterwards; this would seem to be the most obvious explanation of the occurrence, whether it is, or is not, the true one.

Inoculation for Smallpox.

It is a remakable fact in the history of medicine and the treatment of disease, that the inventor of variolous inoculation should be unknown: even the place where inoculation originated is unknown. What we do know for certain is that inoculation was practised at Constantinople in 1700. Dr. Gregory[1] writes: "About the year 1703, rumors of the great success of this operation attracted the attention of Dr. Timoni, a Greek physician, who had studied and graduated at Oxford. He subsequently settled at Constantinople, and being convinced of the importance of the discovery, wrote an account of it, in 1713, to his English correspondent, Dr. Woodward, which in the following year was published in the Philosophical Transactions. In 1715, Dr. Pylarini, the Venetian consul at Smyrna, having also learnt the success of this Turkish practice, published an account of it at Venice. A notice of this work appeared in the Philosophical Transactions for 1716, and these favorable accounts were fully corroborated by the reports of Mr. Kennedy (an English surgeon who had travelled in Turkey) in his Essay on External Remedies, published in London in 1715.

"No notice, however, was taken of these important facts by any English physician, and the idea of transplanting or engrafting Smallpox (as the process was called) was well-nigh forgotten in London, when the celebrated Letter of Lady Mary Wortley Montague appeared, which described the practice in so lively a manner as to attract public attention.[2] 'The Smallpox,' she writes, 'so general and so fatal amongst us, is here entirely harmless by the invention of engrafting, which is the term they give it. There is a set of old women who make it their business to perform the operation. Every year thousands undergo it, and the French Ambassador observes pleasantly that they take the Smallpox here by way of diversion, as they take the waters in other countries. There is no example of any one that has died of it, and you may believe I am well satisfied of the safety of the experiment, since I intend to try it on my dear little son. I am patriot enough to take pains to bring this careful invention into fashion in England.'

"She kept her word, and to the spirit and enterprise of this lady the introduction of inoculation into this country is altogether due; her own daughter was reserved to be the first example of inoculation in England. This event occurred in 1721, and its success was complete."

Through the advice of Drs. Timoni and Pylarini, inoculation was begun in America in 1721, by Dr. Boylston; 244 persons were inoculated, and six died. About the same time, in England, a son of Lord Sunderland, and the butler of Lord Bathurst, both died of inoculation. This brought the practice into discredit, and it was not until the middle of the last cen-

[1] Cyclop. of Pract. Med., vol. iii. p. 748.
[2] Vol. ii. Letter xxxi., dated April 1, 1717.

tury that it was revived. In 1746, the Smallpox and Inoculation Hospital, London, was founded, that the poor might partake of the benefits of inoculation, which had hitherto been confined in a great measure to the rich. Inoculation was continued there until 1822. Dr. Gregory went carefully over the records of the hospital for this period, and found that only three in a thousand died of inoculation. The inoculated disease was usually very mild, but not invariably so. The great objection to it was, that it spread Smallpox just as the natural disease did. It could be set going anywhere by sending in a letter a bit of cotton-thread dipped in variolous lymph for the purpose of inoculation; so that, although the practice was of great advantage to individuals, it was very destructive to the public at large, and the general mortality from Smallpox was thereby greatly increased.

About the time the hospital was founded, two brothers, Robert and Daniel Sutton, one practising at Bury St. Edmunds, in Suffolk, the other at Ingatestone, in Essex, by carrying out more fully than had been done before, the practice of treating Smallpox suggested by Sydenham, improved amazingly the mode of managing those under inoculation. Their practice was adopted by Baron Dimsdale, who obtained great celebrity as an inoculator. It consisted in giving purgative medicine, spare diet, and exposing the patient freely to cold air in the day, and making him sleep in a large airy apartment at night.

"*Phenomena of Inoculation.*—On the second day after the operation, if the part be viewed with a lens, there appears an orange-colored stain about the incision, and the surrounding skin seems contracted. On the following day a minute papular elevation of the skin is perceptible, which on the fourth day is transformed into a vesicle with a depressed centre. The patient perceives an itching in the part. On the sixth day, some pain and stiffness are felt in the axilla, proving the absorption of the virus into the general mass of blood. Occasionally on the seventh, but oftener on the eighth day, rigors occur, accompanied sometimes with faintishness, sometimes with pain of the back, headache, or vomiting. The patient complains of a disagreeable taste in the mouth, and the breath is offensive, soon after which the eruption shows itself."[1]

In 1840, an Act of Parliament was passed, rendering variolous inoculation unlawful in England; the penalty for infringing this law is a month's imprisonment.

Still, however, under certain unfortunate circumstances inoculation might be justifiably had recourse to. First, for instance, when Smallpox breaks out on board ship, and there is not any vaccine lymph at hand, or a probability of any being soon obtained, and persons are present who have neither been vaccinated nor have had Smallpox. Secondly, when Smallpox occurs in our colonial possessions, and several months must elapse before vaccine lymph can be procured from England, or elsewhere. Under these circumstances, on board ship where Smallpox exists, or in houses in the colonies where unvaccinated persons are unavoidably compelled to remain in close proximity to Smallpox, inoculation of them would be perfectly justifiable. Then it will have to be considered and determined, as so many persons now take Smallpox after vaccination, and the badly-vaccinated suffer so severely and fatally from Smallpox, whether the vaccinated who have been badly vaccinated should have their vaccination tested by inoculation. It may be pretty well known, by attending carefully to the previous statements in this article, whether persons have been well vaccinated or not. If they have four or five good vaccine cicatrices, readily discernible, they will most likely have Smallpox in a mild form if they take it, and need not be inoculated. If, on the contrary, they have only one cicatrix that can but just be seen, or no cicatrix at all, they will probably have Smallpox, if they take it, in a severe form. Rather than run the risk of contracting a dangerous attack of Smallpox by inhalation, it would, perhaps, be wiser that they should have their vaccination tested by inoculation. It is no doubt a choice of evils, but the evil is likely to be much less of having Smallpox by inoculation than of having it by inhalation.

In ships, or in houses in the colonies, then, where there is Smallpox, and where no vaccine lymph can be obtained to vaccinate those who have not been vaccinated or to re-vaccinate those who have been badly vaccinated; and where the inmates cannot get away, no further harm would be done to the public by inoculating those who are presumably unsafe, and unavoidably obliged to be resident in such ships or houses. The greatest objection, as before stated, to the practice of inoculation, was from its spreading Smallpox by infection, to other persons; but this objection would not be valid under the conditions above specified — these conditions and limitations being observed, inoculation might still fairly be considered to be admissible and justifiable, rather than to allow unvaccinated or badly-vaccinated persons to take Smallpox in the natural way.

Whenever, after carefully weighing all

[1] Gregory, op. cit. p. 750.

the circumstances of difficulty and danger, it is determined to inoculate, the variolous lymph for inoculation should be taken when *limpid*, and on the *fifth* or *sixth* day of eruption, and when practicable, it should be chosen from a mild form of disease, and inserted into the arm in but one place, the object to be aimed at being to give Smallpox in the mildest possible way.

VACCINATION.

By Edward Cator Seaton, M.D.

Vaccination, or Vaccine Inoculation, is the process by which a peculiar specific disease—Vaccinia or the Cow-pox (from the Latin, *vacca*, a cow)—is introduced into the human system with the view of protecting it against an attack of smallpox.

The Cow-pox never originates spontaneously in man, but (as its name implies) is a disease natural to, and it is not unfrequently seen in, the cow; particularly, and indeed as a disease of spontaneous origin almost exclusively, in the milch cow. As observed in that animal it is a vesicular affection, occurring either casually or as an epizootic, the vesicles manifesting themselves chiefly or entirely on the teats and udder. It has a very precise and definite course. About four days from the probable period of invasion, without any or with scarcely any apparent general indisposition, small, red, rather tender papules appear near the udder and on the body of the teats. These become developed into vesicles, the most characteristic of which are soon seen to have an elevated margin, a central cup-like depression, and by the eighth or ninth day generally a pale rose or light damask areola, not more than a line or two in width, about the base. In two or three days more the disease has reached its acme; the areola has extended to four or five lines, with circumscribed induration of adjacent skin and subjacent cellular tissue: the vesicles, if they have not already burst, are turgid with lymph which, before perfectly clear, is now becoming opaque. By the twelfth day the lymph is more decidedly turbid and a process of desiccation and incrustation has begun, which in five or six days more is complete. The crusts so formed separate spontaneously at from the twentieth to the twenty-fourth day from invasion, leaving behind them permanent slightly-depressed cicatrices. From traction in the process of milking, the vesicles when fully formed very generally burst, and the lymph which exudes from them is frequently found to produce sores of a definite and similar character on the hands of the milkers, who, in their turn, transfer the lymph to, and become the means of infecting, other animals in the dairy.

A popular notion which existed in the county of Gloucester, as in some other dairy districts, that milkers who had thus been infected were afterwards insusceptible of smallpox, attracted about a century ago (in 1768) the attention of Edward Jenner, at that time apprenticed to a surgeon at Sodbury, near Bristol. The impression then made on his mind was never effaced. At a very early period of his medical career he set himself to investigate the truth of the popular belief; and, in the course of inquiries and reflections which satisfied him on this point, his genius conceived the idea that it might be possible to propagate the Cox-pox at will in man, first by directly inoculating it from the cow and then from one human subject to another, and that therewith protection against smallpox might be imparted in perpetuity. Many years, however, elapsed before he was able to put this idea to experimental proof. But when at length he communicated to the world (in 1798), in his Inquiry into the Causes and Effects of the Variolæ Vaccinæ—"that masterpiece of medical induction," as it has justly been called[1]—his great discovery of Vaccination, he had already established on a sufficient basis of observation and experiment the following among other points relative to the Cow-pox: (1) That this disease casually communicated to man has the power of rendering him insusceptible of smallpox; (2) that the specific Cow-pox alone, and not other eruptions affecting the cow, which might be confounded with it, had this protective power; (3) that the Cow-pox

[1] Simon, Letter prefixed to Papers relating to the History and Practice of Vaccination, p. xii.

might be communicated at will from the cow to man by the hand of the surgeon, whenever the requisite opportunity existed; and (4) that the Cow-pox, once ingrafted on the human subject, might be continued from individual to individual by successive transmissions, conferring on each the same immunity from smallpox as was enjoyed by the one first infected direct from the cow.

It is in this transmission of Vaccinia from one human subject to another that the practical usefulness of Jenner's discovery lies; and since the process was first made known, all ordinary vaccinations have been thus carried on. Indeed, although at convenient opportunities, or on grounds which will be hereafter stated, lymph has at various times since Jenner's day been obtained direct from the cow, and new stocks, as it were, thus introduced into practice, we may safely say that the great bulk of the vaccinations of the present day, in this country at least, are performed with lymph transmitted from the early direct vaccinations of Jenner himself.

In endeavoring to give a brief account of the present state of our knowledge in regard to Vaccination, I shall (1) describe the phenomena of Cow-pox as observed in the human subject; (2) describe the mode or modes of inducing these phenomena, or of vaccinating; (3) review the evidence we have of the extent and degree of protection against smallpox which Vaccination affords; (4) state the pathological relations of variola and vaccinia; and (5) inquire whether Vaccination, in protecting mankind against one disease, is, or may be, the means of introducing others—whether, in short, the good that it is known to effect is counterbalanced by any, and what, degree of possible harm.

I. PHENOMENA OF COW-POX IN THE HUMAN SUBJECT.—The symptoms which Cowpox manifests in the human subject resemble very closely those already described as observed in the cow. If some vaccine lymph be taken on the point of a lancet, and inserted by puncture on the arm of an infant who has not before been vaccinated, no particular local effect is noticeable for the first two days; but if the vaccination be about to succeed, by the end of the second or by the third day a slight papular elevation is perceptible, which, by the fifth or sixth day, has become a distinct vesicle of a bluish-white color with a raised edge, and a peculiar, central, cup-like depression. By the eighth day (the day week from the insertion of the lymph) this vesicle has attained its highest perfection, is plump, round, and more decidedly pearl-colored; the elevation of its margin, and the depression of its centre, are more marked. At this date, or sometimes a few hours earlier, a ring of inflammation, termed the areola, begins to form about its base, and, for the next two days, continues to spread. It is circular, and, when fully developed, has a diameter of from one to three inches, and is often attended with considerable hardness and swelling of the subjacent connective tissue. The establishment of the areola demands always the attention of the practitioner and student, as the anatomical evidence that the Cow-pox has produced its specific effect on the constitution. Other proofs of the constitutional influence of the Vaccination are, at this period of its course, generally afforded in the child's restlessness and heat of skin, with (frequently) derangement of the stomach and bowels, and with (sometimes) swelling of the axillary glands. But these general symptoms, though seldom altogether absent, are often exceedingly slight; nor, provided the areola be properly formed, are they to be looked upon as indispensable for the protective effect of Vaccination. After the tenth day the areola begins to fade, the vesicle begins to dry in the centre, the lymph remaining in it becomes opaque and concretes, and by the fourteenth or fifteenth day, a hard brown scab is formed, which contracts, dries, blackens, and, from the twentieth to the twenty-fifth day, falls off, leaving a cicatrix commonly permanent, and which in character is circular, somewhat depressed, foveated or indented with minute pits, and sometimes radiated.

Occasionally certain constitutional symptoms beyond those already described are observed. In young children of full habit, especially in hot weather, about the ninth or tenth day, when the areola is at its height, an eruption of roseola will sometimes take place, chiefly on the extremities; sometimes the eruption has a papular form (vaccine lichen), and sometimes it is vesicular. These eruptions are generally very transitory; their ordinary duration does not extend beyond a week; and they very seldom indeed last beyond the falling of the scab.

When the mode of inserting or applying the lymph has not been by single puncture, or by such abrasion of the skin as would raise a single vesicle, but by two, three, or more punctures close-set together, or by scarifications or abrasions over some extent of surface in the manner that will hereafter be described, so that two, three, or more vesicles are developed in close proximity, these usually coalesce, and a vesicle results, the compound character of which is very obvious: or there may be a crop of coalescent vesicles, each having its distinct head with characteristic depression. These compound vesicles and crops are round, oval, or of irregular outline, according to the manner in which

they have been induced, and the shape of the resulting cicatrices varies accordingly.

The clear, smooth, supple, delicate skin of the infant is peculiarly adapted to manifesting in perfection the local characters of the vaccine disease. In elder children, and much more in adults, though the phenomena of primary vaccination are essentially the same as in the infant, the vesicle is often wanting in that plumpness, sharp definition of edge, and beautiful lustre which the experienced vaccinator delights to see. And, in the adult, the course of the disease is frequently somewhat retarded, the areola is apt to be more diffuse, and swelling of the axillary glands is more frequently observed.

When lymph is employed that is derived directly or very recently (as within three or four removes) from the cow, the course of the disease is generally retarded at various stages. Papulation is sometimes deferred till the seventh, eighth, ninth, or even the tenth day, and the areola is not complete till from the eleventh to the fourteenth or even the sixteenth day. The areola, when at its height, is more indurated than is observed in Vaccination with ordinary humanized lymph, and is said to decline and revive, continuing to exhibit a brick-red or purplish hue while the hardness remains.[1] The papular and vesicular eruptions, which have been above referred to as occasionally attending Vaccination at this stage of its course, are more frequently seen. The vesicles themselves are commonly not more developed than those produced by ordinary lymph. Desiccation is generally prolonged, and the crust is often retained till the fourth or fifth week.

Such are the ordinary phenomena induced by primary Vaccination in the human subject. Their course, however, is sometimes modified; and may be (*a*) simply retarded, (*b*) simply accelerated, or (*c*) altogether irregular and spurious. (*a*) *Retarded Cow-pox.*—The most simple and frequently seen form of retardation is a mere delay for a day or two in the course of the vesicle; by the eighth day it has not more size or development than ordinarily is met with on the sixth, and the areola does not form till the tenth day, or even later. This amount of retardation is probably more frequently met with in cold than in hot weather, especially if the cold be accompanied with a dry easterly wind. Sometimes the delay is much longer; at the end of a week from the Vaccination, when the child is brought for inspection, so little of result is seen that it is a question whether the Vaccination has not altogether failed: some fresh lymph is inserted on another spot, and, as the vesicle of the new Vaccination rises, that of the first Vaccination is seen also to develop itself, and the two run their course at the same time. Bousquet refers to a case (not in his own practice) in which this revival of a dormant Vaccination took place, it was said, at the end of three weeks.[1] I have never seen any case in which the vesicle rose after such a delay as this; but I have seen cases in which, without any recourse to a second Vaccination, vesicles have risen a week after the usual period, and when success had ceased to be looked for. Retarded Cow-pox is much more frequently seen when Vaccination is performed with dry lymph than when it is done direct from the arm: no doubt because, in the former case, the lymph, when deposited on the cutis, is often in an undissolved, or imperfectly dissolved, state. Mere retardation of phenomena, if these phenomena be regular in their character, does not in any way interfere with the protective value of the Vaccination. Retardation of Vaccination at various stages of its course is often independent of any obvious cause; but sometimes it can be traced to certain conditions of the system, as the incubation of other diseases. If, for instance, vaccine lymph has unwittingly been inserted in a child who happened to be incubating measles or scarlatina, and the Vaccination has succeeded, the vesicles will usually, when the rubeolar or scarlatinal symptoms set in, be arrested in their course, and no areola will be formed till the measles or scarlatina have subsided.[2] Again, when Vaccination has been performed with effect on a child who is incubating smallpox, the vaccine vesicles usually proceed in the ordinary way till the smallpox manifests itself, and, even after this, may undergo some further local development, the vaccinia and variola apparently going on together; but unless, before the smallpox appeared, the Vaccination had already reached the stage of areola, its progress to that stage will be arrested, in which case it will have no effect in modifying the smallpox. (*b*) *Accelerated Cow-pox* is to be regarded with much more suspicion than retarded Cow-pox, spurious vaccination having generally an accelerated course. There may be, however, simple acceleration; a course some twelve or twenty-four hours in advance of the usual course, the vesicle on the eighth day being in the state in which it is usually seen on the ninth, the areola nevertheless being regular, and the crust being subsequently duly formed. In such case the value of the Vaccination is not

[1] Ceely, Observations on the Variolæ Vaccinæ in Trans. of Prov. Med. and Surg. Assoc., vol. viii. p. 346.

[1] Nouveau Traité de la Vaccine, p. 176.

[2] Jenner notices this in one of his early tracts, Continuation of Inquiry, p. 31.

impaired. (c) *Irregular and Spurious Cow-pox.*—Vaccination sometimes runs an entirely irregular course, the varieties of irregularity being considerable. In one form the most frequent, the course resembles that of a revaccination (as will hereafter be described) instead of that of a primary Vaccination; the vesicle begins with itching and irritation; it is acuminated or conoidal, instead of being flat and with central depression, and it contains straw-colored or opaque fluid, instead of clear lymph; it has an early or irregular areola, which is at its height by the fifth or sixth day, and when seen on the eighth day is far on the decline; there is then a small scab on the surface which usually drops off by the tenth day. Or the local effect may have been even less than this, and on the eighth day there may be nothing remaining but a very thin scab or scale just about to detach itself, and which at the lightest touch falls off. In other cases, the vesicles, when seen on the eighth day, are found to have burst, and to present an irregular pustuloid or scabby appearance, or to be so many open sores. It would be difficult, and is quite unnecessary, to give a verbal description of each form of irregularity that may be observed; *the one important practical fact being that a Vaccination presenting any deviation from the perfect character of the vesicle, and the regular development of the areola, is not to be relied on as protective against smallpox.* A spurious and irregular course of the vaccine vesicle, no doubt, sometimes occurs without assignable cause, and in the most experienced hands, but it is far more usually due either to the use of lymph not well chosen by the vaccinator, or to something amiss in the state of the child vaccinated. Thus, it may be seen in various forms on several children vaccinated from the same source; or it may occur in children who are vaccinated from the undeniably good sources, but who themselves, at the time, are suffering from intertrigo or other affections. A spoilt and broken appearance of vesicle, which is frequently on the eighth day ascribed to rubbing or mechanical irritation, is often really an irregularity arising from one or other of these causes; and the best proof how much such irregularities depend on the care and skill of vaccinators is the frequency with which they are seen in the hands of some vaccinators, and their extreme rarity in the hands of others. Parents, however, should always be directed to be very careful to keep the vesicles uninjured in their progress, and to avoid the premature removal of the crust.

The local and general symptoms of Vaccination seldom call for any treatment. Occasionally, however, they run an active course; the arm becomes inflamed; erythema, or sometimes true erysipelas, develops itself; the vaccine vesicle degenerates into a purulent ulcer, or sometimes into a sloughing sore, leaving a cicatrix which has none of the characteristic pittings, but is simply a puckering, or a flat, smooth, shining scar. The treatment of such cases would be the treatment of erysipelas or of inflammation from any other cause. It is needless to say that degeneration of the vesicle thus taking place deprives the Vaccination of all protective power.

Phenomena of Revaccination.—In the majority of persons the regular phenomena of Vaccination, such as we have described them, can only be produced once in a lifetime; any subsequent introduction of lymph either failing to produce any local effect whatever, or (much more commonly) producing a modified effect, resembling one of the forms of spurious Vaccination. The absence of effect is relatively most common in the child, the spurious effect most common in the adolescent and adult. This spurious effect consists either in a papule, or (more often) in an acuminated vesicle, with a hard and irregular areola. The symptoms begin early, reach their height by the fifth or sixth day, and then decline. The scab, small and imperfect, forms generally on the eighth day, and soon falls. There is usually much itching, and often considerable constitutional irritation. Severe constitutional symptoms are, out of all proportion, more frequent in revaccination than in primary Vaccination; and in very exceptional cases the vaccine lymph may act as an animal poison, giving rise to phlegmonous erysipelas: some still rarer cases have occurred of pyæmia, terminating fatally. In a certain proportion of cases the results of revaccination are the same as those of primary Vaccination, the vesicle, in shape and character, being in no degree distinguishable; in such cases the areola is sometimes small and transitory, and the scab on falling leaves a small inferior cicatrix; at other times the areola is perfect, and a good cicatrix is left. Normal vesicles resulting from revaccination are much more frequent in adults than in children, but I have seen them in the arms of children a few years old, who had excellent marks of their first Vaccination.

II. METHOD OF VACCINATING.—For the proper and successful conduct of Vaccination, special attention must be given to the state of health of the child to be vaccinated, to the selection of the lymph to be used in the Vaccination, and to the thorough insertion of it.

1. Except for pressing reasons, children should only be vaccinated when they are in good health. Especially must they be free from any acute disease, from diarrhœa,

or from any chronic disease known to interfere with the regular course of the vaccine vesicle. Of these diseases, the chief are herpes, eczema, and intertrigo. The latter affection, especially, so often modifies, or even completely spoils the course of Vaccination, that it is of great consequence, before proceeding to vaccinate a child, to see that there is no chafing behind the ears, in the folds of the neck, or in the groins. But cutaneous eruptions of all kinds are a contra-indication, and, as a rule, great attention should be given to having the skin perfectly clear, and free from them, before inserting vaccine lymph. Of course this rule is liable to be modified by circumstances; and especially when there is any immediate risk of smallpox, it becomes imperative to perform Vaccination notwithstanding these contra-indications.

Health permitting, all children should be vaccinated in very early infancy. Inasmuch as one-fourth of the deaths from smallpox in England occurs in children under the age of one year,[1] it is obvious that delay is attended with extreme risk; and this risk, it need scarcely be said, is greatest in large towns. Plump and healthy children, living in large towns, should be vaccinated when a month or six weeks old;[2] in more delicate children, the Vaccination might be postponed till they are two or three months old; but all, except those whose state of health positively contra-indicates Vaccination, should be vaccinated by the age of three months. This early period of life is also particularly suitable for Vaccination, as being usually free from the disturbing influence of teething. Circumstances connected with lymph-supply render a longer delay unavoidable in many instances in small towns and country districts; and, when smallpox is not present in the localities, such delay, if not too long continued, is not of material moment. But even children living in these districts should always be vaccinated within a few months, six or seven at the utmost, from birth.

Under circumstances, however, of direct exposure to smallpox, it should be well understood that no age is too early for Vaccination; infants have repeatedly been vaccinated immediately after birth, and thereby saved. In all cases of much risk, it is of the utmost importance to avoid any delay. The loss of a day may be the sacrifice of a life. The Vaccination may possibly be in time to prevent smallpox altogether; but even supposing the variolous infection to have entered the system before the Vaccination has been performed, still if *the Vaccination be only got to the stage of areola* before the smallpox manifests itself, it will exert its modifying power, and the child will be saved.[1]

2. The lymph to be used in vaccinating should be taken from healthy subjects, and from thoroughly characteristic vesicles. It *may* be taken as soon as the vesicle will yield it, when, though it can only be got in small quantity, it is effective. It is usually taken, and for the ordinary purposes of Vaccination is best taken, when the vesicle is fully formed, but either before the areola appears, or within a very few hours of its commencement. This, in regular cases, is on the day week from the Vaccination; the lymph is then easily obtained in sufficient quantity. It is a very great mistake, but one which is often made, to take it at a later period of vesicle than this, and when the areola is fully complete. No doubt it flows then more freely, and may be got in greater abundance; no doubt also (as alleged by those adopting this practice) it very often takes; but it does not do *this with anything like the same certainty*—a point of great consequence; and it is also more likely to lead to erysipelatous and spurious results. Prime lymph has always a certain degree of viscidity, and a thin serous lymph, even from a vesicle which is not advanced, is to be avoided. Babies are much better lymph-givers than elder children, or adults. Children of dark complexion, not too florid, with a thick, smooth, clear skin, yield the finest and most effective lymph.

3. A child and a vesicle fit for the purpose having been selected, the vaccinator in order to collect the lymph proceeds to open the vesicle by a number of minute punctures, which must be made on its surface and not round the base. The object

[1] Of 42,277 deaths from smallpox which occurred in England in the ten years 1856–65, 10,223 were in children under one year of age.

[2] If the parents of a child, apparently healthy, were known to have any taint of syphilis, it would, in the interests of Vaccination generally, be a matter of prudence to postpone the Vaccination, unless smallpox were imminent, till the age was passed within which hereditary syphilis mostly manifests itself, *i. e.* till the completion of the third month from birth. For if any syphilitic symptoms were to appear after the Vaccination, they would most likely be ascribed not to their hereditary cause but to the Vaccination.

[1] The incubative period of smallpox being twelve days, and the time requisite to carry Vaccination to the stage of areola being only nine days, it is manifest that even a person who has actually imbibed the infection of smallpox may, by Vaccination within the first three days after the reception of the infection, obtain the modifying benefits of Vaccination. A day more, and the Vaccination will be too late: whatever local effect it may produce, the smallpox will not be modified by it. (See the article Smallpox: see also the subject more fully treated in my "Handbook of Vaccination," pp. 102–106.)

in making many punctures is to open the various cells of the vesicle, and the reason for making these on the surface, and not round the base, is to obtain the lymph free from any admixture of blood. If by accident any blood be drawn, this must be allowed to coagulate, and then be carefully removed before taking the lymph: for it is a cardinal rule, never to be deviated from, that the inoculation must be with vaccine lymph, *and with lymph only*. When the cells of the vesicle are freely opened, the lymph soon exudes, and lies on the surface. The lancet or point to be charged is then dipped into it; or if a capillary tube is to be charged, the end of the tube is inserted in the liquid as it lies. On no account must there be any pressure or squeezing of the vesicle with the lancet to make the lymph exude: and when lymph ceases spontaneously to stand on the surface of a vesicle, that vesicle must be considered no longer usable for lymph supply. Very generally, however, when the lymph which has first exuded has been taken, and the surface of the vesicle left apparently dry, if the operator wait a minute or two, he will find there has been a fresh exudation of good usable lymph; and when he does not find this, he may often induce it in a way quite unobjectionable, by wiping very gently the surface of the vesicle with a soft wet linen cloth, thereby removing or dissolving the inspissated lymph which clogs the punctures. Vesicles of perfect character, and of the same size and appearance, differ very much in their yield of lymph; ordinarily, from a vesicle of such size as is produced by a single deep puncture, enough lymph may be got for the direct Vaccination of from four to six children, or for charging (*i. e.* for well charging, dipping once and again) six to eight ivory points. Some vesicles yield much more, but the caution already given against a thin, serous, too-readily-flowing lymph must be borne well in mind. When vesicles are compound, their yield of lymph is of course proportionally increased.

Lymph should in every instance (where practicable) be inserted direct from arm to arm. All processes for preserving and conveying lymph (valuable, and indeed invaluable, as they are for their own proper purposes) render in the long run the taking of the Vaccination a matter of inferior certainty:[1] and it is only in cases of necessity that Vaccination should be performed with lymph stored or conveyed. But a caution must be here interposed. The superior relative advantages of arm-to-arm Vaccination are so well known, that practitioners whose vaccinations are few, and whose opportunities of lymph-selection are therefore limited, will often be induced to take their lymph from second-rate vesicles, rather than lose the opportunity of vaccinating direct from an arm. This, which I know to be the source of much current inferior Vaccination, is a course which should never be adopted. The operator, *unless he has some preserved lymph that he can rely on and unless he can rely on his own hand to make preserved lymph take*, should put off the Vaccination till he has the opportunity of doing it from a thoroughly satisfactory vesicle. [In the United States, many practitioners have for a long time been in the habit of using the scab, instead of fluid lymph, for vaccination. The scab is allowed to come off spontaneously, about the nineteenth or twentieth day. When kept covered from the air, it will retain its specific character, usually for a month or more; and the vaccine pustules produced by it go through all the stages and appearances belonging to the Jennerian pustule. For the use of the scab, it is only necessary to mix a small portion of it with tepid water, into a paste, which may be applied to the arm in the same manner as the fluid lymph.—H.]

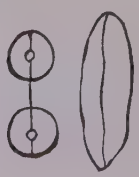
Various methods may be employed for inserting lymph—the essential part of all of them being either to introduce the lymph into the substance of the cutis, or to bring it well in contact with its absorbing surface. (*a*) One of the most commonly used is that by puncture. When it is intended to operate in this way, the arm of the child to be vaccinated should be grasped by the left hand of the vaccinator, so as to put the skin on the stretch, and a very sharp, perfectly clean lancet, well charged with the lymph selected, should be introduced by valvular puncture from above downwards, so that the lymph may gravitate into the wound. The lancet should not be held level with the skin, but at an angle of 45°, or thereabouts, and made *to enter the cutis*. If the lymph be thus well put in, it is retained by the valvular character of the puncture and elasticity of the skin; and any fear that the bleeding which ensues will cause the Vaccination to fail is quite chimerical. A minute and superficial puncture, on the other hand, does not unfrequently fail. In vaccinating by puncture, *not less than* five should be made, and they should be at a distance of half an inch from each other. Five or more punctures at this distance from each other can very well be made on one arm, or, if the operator prefer it, three or more may be made on each arm. In the manipulations by which Vaccination is effected by puncture, it is a very good plan to make each puncture a double one: thus, //. A finer and

[1] An account of the various methods of storing lymph and their relative advantages is given in my "Handbook of Vaccination," chap. viii.

larger, often oval, compound vesicle is thereby raised. For Vaccination by puncture no instrument is needed but a common lancet, very sharp: and many of the instruments which have been specially devised, are not, in my opinion, nearly so good. (*b*) A modification of the plan of vaccinating by puncture is that of multiple superficial punctures, or tattooing—a number of minute superficial punctures being made with the point of the lancet, thus,

and the lymph then spread over with the flat part of the lancet. The number of spots over which this tattooing should take place will depend, of course, on the extent of surface operated on at each spot; but in order that a local effect may be produced equal to that of five ordinary vesicles, tattooing over such a surface as is above depicted should be repeated on at least three spots. (*c*) Another modification of puncture, common in some of the northern districts of England, is that of first spreading the lymph on the arm of the child to be vaccinated, and then ripping up the cuticle with the point of the lancet

over a surface equal to a sixpenny-piece, or more, with, frequently, a second plastering of lymph afterwards; crops of vesicles are thus raised, close set together, and nearly always confluent, each vesicle, however, having its distinct depressed head. I have counted as many as eighteen or twenty such on one base: but the vesicles by the pressure they exercise on each other do not attain the size and development of separate vesicles. It is usual to make two crops, such as I have described, and this must be regarded as sufficient for full protection. (*d*) Vaccination is often performed, not by puncture, but by scratch; three or four longitudinal or transverse scratches, each about three-quarters of an inch long, are made at distances half an inch or an inch apart from each other, with the point of a lancet, or with a thick needle, and the lymph is rubbed on. In the course of each scratch two or three separate vesicles



will arise, or more frequently, one oblong compound vesicle will be produced. (*e*) Another and very excellent plan of scarification is adopted by many vaccinators, which consists in abrading the cuticle by a number of fine parallel scratches, thus,

or by further cross-scratch, thus,

Abrasion should only be carried so far as to make it certain, by the appearance of blood oozing, that the cutis is reached; any such oozing is then rapidly wiped away with the finger and the lymph plastered on. When the abrasions are made to cover such a space as is above indicated, it is sufficient to make two on each arm, or three on one arm, about three-quarters of an inch or an inch from each other—but when, as by some practitioners, the abrasions are made over smaller surface,

producing vesicles which are not more than equal in size to those of good puncture, there should be at least five such abrasions. Vaccination by scarification cannot be better done than with a common lancet; some practitioners, however, prefer the use of a scarifier or rake (invented by Dr. G. Weir), which consists of three or four needle points, inserted in a handle of ivory. The skin being held very tense, this rake is drawn lightly across in one direction, and then, if the operator pleases, crossed in another, the lymph being then plastered over.

In cases in which Vaccination has to be performed with preserved lymph, if the lymph has been preserved liquid, as in Husband's tubes, no other directions are requisite for the performance of the operation than those which have now been given. But if the lymph has been preserved by drying, as on points or glasses, it will be necessary that it should be revived or brought back to the liquid state in order that it may be taken up by the system. This is done by the use of a very minute quantity of water; if the lymph to be revived has been preserved on glasses, a very small drop of water is taken on the point of the lancet and well rubbed in with the lymph, which is then left for some minutes to soften; or, if the lymph has been kept on points, these should be very lightly dipped in water and then placed for a few minutes on the edge of a

book. Much care is required in thus reviving lymph: on the one hand, there must be moisture enough to bring the lymph back to the state in which it was when taken from the vesicle; on the other, it is important that the lymph be not too much diluted. For puncture-vaccination the revived lymph is either taken on the lancet-point and inserted as in a Vaccination direct from the arm, or, if the mode of preserving has been by points, a puncture is first made with a clean uncharged lancet and the ivory point is then itself inserted into this puncture and kept in position by the thumb for a short time, care being taken to press the thumb well down as the point is being withdrawn, and thereby insure that the lymph on it is well wiped off and left in the wound. In Vaccination by scarification or abrasion, the flat of the glass, or the flat of the point, may be rubbed over the scarified or abraded surface.

Of the different modes of vaccinating which I have described, and of numerous modifications of them which are met with in practice, is there any which can be more thoroughly relied on than another for infecting the system? This is a question very frequently put to me, and my reply always is that a person properly taught *ought* to be able to infect thoroughly by either of them; and that any one who has so learnt, and so habitually practises, either plan as to be able to rely on securing the results he aims at, should not—so much depends on habit—change it for any other. By every one of the processes I have described, I have seen the most perfect results produced by experienced vaccinators: certainly by no process more completely than by the one I first described—that of simple puncture. But unusual opportunities of observing Vaccination as generally practised have shown me that many, if not most, practitioners do not—for want of original instruction, and of attention to various small but essential points—by whatever process they adopt, succeed as they ought to do; and judging by the results in the cicatrices which their vaccinations leave, that, as a rule, those who vaccinate by simple puncture are by far less successful than those who vaccinate in either of the other ways described;[1] further, that the best marks, on the whole, as regards size, depth, and foveation, are those which result from the plan last of all described, viz.: that of cross scratch or abrasion over a sufficient surface.

And this plan is, in most hands, eminently more successful than puncture when dry lymph has to be employed.

Mr. Marson states that "with good lymph, and the observance of all proper precautions, an experienced vaccinator should not fail of success in his attempts to vaccinate above once in 150 times."[1] His own habitual success is, indeed, far greater than this; but so much, at all events, he considered might fairly be expected of any good vaccinator. That this standard is not too high is clear from inquiries I have made as to the results obtained at several large stations where Vaccination is well performed and where accurate records are kept at which I found that the average of failures to infect at the first operation did not exceed one in 170 cases. Good vaccinators expect also to raise a vesicle for every point of insertion; and, in their hands, cases in which only one or two vesicles result from four or five punctures or scarifications are as rare as total failures. But such results, it must be well understood, will not ensue unless patience and diligence be given to learn how to select lymph, and how to manipulate; nor unless practitioners, seriously considering the responsibility which rests upon them when they are proceeding to vaccinate, act with corresponding care. If they fail to infect their patients at the first, and still more if they fail at the second trial, it is very likely these will not again present themselves, and will remain unprotected. If, endeavoring to raise four or five vesicles, they raise only one or two, their patients do not get all the protection Vaccination is capable of affording. It has happened to me for many years past to see, all but daily, children growing into manhood and womanhood, who were unprotected against smallpox, not by their own or their parents' neglect of Vaccination, but by the want of skill of operators, who (often with two, three, and four operations) had failed to infect them. Mr. Marson also says, "Patients often present themselves with smallpox at the hospital, who state they have been cut five, six, or eight times, or more, for Cow-pox without effect." "This," he adds, "is a great evil. It would happen but rarely in careful

[1] Chiefly, I believe on account of the minute and superficial character of the puncture, and the very small quantity of absorbing surface exposed to the action of the lymph. This arises in great measure from a chimerical fear that if blood be drawn the lymph will be washed away. There is a kind of Vaccination by scarification which is equally imperfect in its results, when a sort of make-believe scratch like this | is made, and a little lymph put on.

[1] Analytical Examinations of Smallpox and Vaccination, Medico-Chirurg. Trans., vol. xxxvi.

hands." So far, indeed, are practitioners generally from having attained the standard which all should reach, that it is no uncommon thing to hear 5 and 10 per cent. of failures spoken of as a satisfactory result, while not a few think they do well if they succeed in four operations out of five: and constantly practitioners are met with, who do not in one-half the cases they vaccinate, and many of them not in a quarter, raise all the vesicles which they have attempted to produce.[1]

Insusceptibility to the infection of Cow-pox, even for a very limited period, is an excessively rare occurrence. For in the few cases in which good operators fail at the first operation to infect they rarely fail at a second trial, but now and then this is so, and even a third operation may not take. In such cases, if the operations have been properly performed, and lymph inserted direct from the arm, it may be assumed that there is temporary insusceptibility. Cases have been mentioned to me on good authority, but I have never met with such myself, in which this insusceptibility has remained for many years.

III. Protection afforded by Vaccination against Smallpox.—Persons who have once been successfully vaccinated are, as a rule, permanently protected against smallpox. A certain but indeterminate proportion of vaccinated persons will, however, be liable at some period or other of their lives, especially under epidemic influence, to take smallpox in a mild and modified form. A very much smaller proportion will be liable to take it in a severe, disfiguring, or fatal form. But the liability of any individual to take smallpox severely after Vaccination, and probably the liability to take it at all, will be inversely as the goodness and amount of the Vaccination.

The protection which Vaccination is capable of affording against smallpox was held by Jenner to be exactly that—neither more nor less—which an attack of smallpox, either taken naturally or induced by inoculation, was well known to confer against a subsequent attack of the same disease. Believing Cow-pox and smallpox to be identical, he considered that the system of a vaccinated person had already in fact passed through an attack of smallpox. He was well aware that this disease did in some individuals recur, and that the having passed through one attack was not in every instance a security against a future attack. He refers repeatedly in his writings to cases of this kind seen by himself and by other reliable observers, and states the claims of Vaccination thus: "Duly and efficiently performed, it will protect the constitution from subsequent attacks of smallpox as much as that disease itself will. I never expected it would do more; and it will not, I believe, do less." It was only, however, to *efficient Vaccination*, *i. e.*, to Vaccination which had gone through all its stages with perfect regularity and had given evidence of infecting the constitution, that he attributed this protecting power. Inefficient Vaccination, like inefficient inoculation, he knew would fail. Observations made since Jenner's day, with remarkable care and ability and on a scale which gets rid of all sources of fallacy, by Mr. Marson of the Smallpox Hospital, have conclusively established that, for *thoroughly* infecting the constitution, a certain *amount* of local affection is as necessary as a perfect *character* of vaccine vesicle. We must therefore so far extend the meaning of the words "due and efficient" performance of Vaccination, as to make it include amount as well as quality of vaccine influence: and with this extension, the experience of seventy years tends to show the correctness of Jenner's estimate.

The protective power of Vaccination, like that of natural or inoculated variola, shows itself in two ways: 1st, as regards the large majority of persons, in shielding the constitution against any future attack, however modified, of smallpox; and 2d, as regards the remainder, in general so modifying the subsequent smallpox that that disease is, as a rule, deprived of all danger to life, and does not on recovery leave behind it those disfiguring traces which are not the least of the terrors of unmodified variola.

1. On no subject is medical testimony more unanimous than on the very large immunity from attacks of smallpox which successful Vaccination will confer.[1] While there are few unvaccinated persons who do not at some period or another of their lives sustain an attack of variola, the vaccinated are, as a rule, entirely exempt from it. Precise data have never been collected on a sufficient scale to enable us to state with numerical accuracy what

[1] *Vide* Reports of Medical Officer of Privy Council, iii. iv. v. vi. and vii.

[1] See this testimony as given on a large scale in Report of Smallpox and Vaccination Committee of Epidemiological Society, 1853; Seaton on Protective and Modifying Powers of Vaccination, 1857; and Simon, op. cit. pp. lxxix. lxxx. The Epidemiological Society refer to answers received from above 2000 medical practitioners in England, besides evidence collected from abroad, affirmative of the protective value of Vaccination. Simon gives the answers, to the like effect, of 540 distinguished medical men, British and foreign (out of 542 of whom inquiry was made), to a question "purposely constructed to elicit the expression of every existing doubt on the protective influence of Vaccination."

proportion of persons may still be liable to take smallpox, who have already had variola or who have been successfully vaccinated. The proportion in both cases will vary, of course, with the degree of exposure to the infection, and will no doubt also be greatly dependent on the presence or absence of epidemic influence. We have, however, as regards one limited class of the population some very precise facts. The records of the Royal Military Asylum at Chelsea show that 5,774 boys were admitted into that institution in the course of the forty-eight years ending December, 1851, of whom 1,950 had on admission marks of smallpox, and 3,824 either had marks of Vaccination or were on admission vaccinated:[1] of the former 6·15 per thousand, and of the latter 7·06 per thousand, contracted smallpox subsequently during their residence in the asylum. Dr. Balfour, the able and accurate statistician to whom we are indebted for these facts, tells us also that in a long series of years antecedent to the publication of his Memoir,[1] the annual number of *cases* of smallpox in the British army had not been more than 6·6 per 10,000 men, one-fifth of the men, it was calculated, being protected by previous smallpox, and the other four-fifths by Vaccination. That portion of the troops which was serving in the United Kingdom, and was quartered chiefly in large towns (from which, to the disgrace of this country, smallpox is seldom absent), suffered in greater proportion, but still the average annual admissions among these were for a period of ten years (1837–46) only 22 out of every 10,000 men. The returns for the four years 1859–62 show on the average a considerable diminution even in this ratio.[2]

Year.	Number of Troops in United Kingdom.	Cases of Smallpox.	Deaths.	Per 10,000.	
				Cases.	Deaths.
1859	71,715	175	7	24·3	0·97
1860	85,443	140	9	16·8	1·05
1861	88,955	51	4	5·9	0·45
1862	78,173	64	4	8·1	0·51

In civil practice, numerical inquiries have sometimes been made as to the protection enjoyed by individuals under the highest degree of exposure to smallpox—members of families in which that disease existed, who were living and sleeping at home, in the same house, even generally in the same room, and sometimes in the same bed, with the infected case. The result of such an inquiry, made by Mr. Cross, of Norwich, was that of 215 unprotected members of families so circumstanced, 200 contracted the disease and 46 died; while of 91 vaccinated, only 2 took the disease, and these both had it in its modified form. A rather more extensive inquiry was made some years later by Mr. Marshall, of Chelsea,[3] into the facts connected with 757 individuals in infected families. He found that of 231 who had been protected by Vaccination, 27 had contracted smallpox during an epidemic that had just then prevailed; that of the unprotected every one had been attacked except 7; and that 14 cases had occurred in persons who had previously had variola. The only inquiry of this kind, so far as I know, in which regard has been had, not merely to the fact of Vaccina-

[1] This period extends back as far as 1803: it will be noticed that one out of every three children admitted in these forty-eight years showed marks of smallpox. No better illustration of the progress Vaccination has made and of its wonderful influence in protecting against smallpox can be given than is afforded by contrasting this proportion with the proportion we *now* find of children of our poorer classes who are marked with smallpox. When, five years ago, an examination was made among school children of this class in London, less than one in forty was found to have any traces of smallpox.

[1] In 1852; in Medico-Chirurg. Trans., vol. xxxv.

[2] The variation in the annual number of cases depends on the presence or absence of epidemic influence: two epidemic and two non-epidemic years are included in the table. It will be seen that the *recent ratio of cases in epidemic years* has not exceeded *the average of ten years*, 1837–1846. This improvement is no doubt mainly due to the closer scrutiny now given on the admission of recruits to the evidences of their protection against smallpox, and to the performance of revaccination. As regards the relative protective value of variola and vaccinia the improvement is significant, for the relative number of soldiers depending for their protection on vaccinia is much greater now than before.

[3] *Lancet*, vol. xxxvi.

tion, but to its amount and quality, has been the one made some years ago by the Epidemiological Society, as to the extent to which medical men—who, from their profession, would be unusually exposed to smallpox—had suffered from that disease after Vaccination or after previous variola. The results of this inquiry were, that of 347 who had been vaccinated, 44, or 12·6 per cent., had subsequently contracted variola, generally in the most modified form. Some of these had no cicatrix of their Vaccination, and in others the cicatrices were indifferent, or were only one or two; but of 57 who had three or more good cicatrices, only 2, or 3·5 per cent., had had smallpox. Of 82 medical men who had had smallpox, most of them by inoculation, in infancy—3, or 3·6 per cent., had contracted variola in later life.[1] These observations are strongly confirmatory of Jenner's anticipations as to the relative protective value of vaccinia and smallpox.

2. The facts showing the power of vaccinia in modifying smallpox, if it should happen to be subsequently contracted, and of disarming it of its terrors, are so ample that it is difficult to know whence to select examples. No epidemic of smallpox has occurred in any climate since the introduction of Vaccination without affording the most abundant evidence of it. While the mortality of natural smallpox is seldom below 20 per cent. and often amounts to 30 and 40 per cent. of the attacks, the death-rate among the vaccinated (taken indiscriminately and without regard to the quality of their vaccination) is rarely known to exceed 7 per cent. and is more frequently 3, 4, and 5 per cent. It will have been noticed, in the table above given of the cases of smallpox among the soldiers in the United Kingdom in the four years 1859–62, that of 430 cases of the disease only 24 were fatal, or 5½ per cent. In observations which, on account of the large scale on which they were made, are of great value, viz. those made for twenty-one years in Bohemia on four millions of people, it was found that the death-rate among vaccinated persons who happened to contract smallpox was $5\frac{1}{16}$ per cent., while the death-rate among non-vaccinated persons when they contracted smallpox was 29⅓ per cent.[1]

But the observations which outweigh all others in value, on account of the extreme accuracy and precision with which they have been made, are those which Mr. Marson has collected by thirty years' labor at the Smallpox Hospital. In this hospital above 15,000 cases of smallpox have during that time been under his personal care, and all particulars respecting them have been carefully recorded: and it has been found that while the unvaccinated have died at the rate of above 35 per cent., the vaccinated have died at the rate of only 6½ per cent.

But Mr. Marson's observations do far more than establish in a general way, in concurrence with others, the modifying power of Vaccination. They have a merit peculiarly their own. They show conclusively that *the degree of modifying power is in the exact ratio of the excellence and completeness of the Vaccination as shown by the cicatrices:* in other words, that it is directly as the amount of vaccine-marking and as the character of the marks. The subjoined table will show this at a glance, better than any detailed statement:[2]—

Classification of Patients affected with Smallpox.	Number of Deaths per cent. in each class respectively.
1. Unvaccinated	35
2. Stated to have been vaccinated, but having no cicatrix	23·57
3. Vaccinated—	
a. Having one vaccine cicatrix[3]	7·73
b. Having two vaccine cicatrices[3]	4·70
c. Having three vaccine cicatrices	1·95
d. Having four or more vaccine cicatrices	0·55
a. Having well-marked cicatrices	2·52
b. Having badly-marked cicatrices	8·82
4. Having previously had smallpox	19

So that, while the average of vaccinated persons, if they should ever contract smallpox, have about one-sixth of the chance of having it fatally which is run by those who have not been vaccinated at all; some of them, from bad Vaccination, incur in fact one-third of that risk, while on the other hand others, thoroughly well vaccinated, incur less than one-seventieth part of it.

[1] Seaton, on the Protective and Modifying Powers of Vaccination.

[1] Simon, op. cit. p. xxvii.

[2] See also the article on Smallpox. The deaths from superadded disease have been deducted both from the unvaccinated and the vaccinated.

[3] In these classes the influence of quality of cicatrix was remarkably seen. In class *a*

In regard therefore to the expectation of any case of smallpox turning out badly, the question is not merely whether the patient has been vaccinated or not, but also *how* he has been vaccinated.

These invaluable observations form the basis on which Vaccination should always be conducted. Nor, relying as we must do upon them, is it too much to affirm that no practitioner will have done his duty in any case in which he is called upon to vaccinate, unless, besides all requisite precautions with regard to the genuineness of the lymph employed and the means of insuring success, he has also taken care to vaccinate sufficiently, *i. e.*, to produce, so far as in him lies, four or five genuine good-sized vesicles, such as result from separate punctures, or if vaccinating otherwise than by separate puncture, to produce equivalent local results.

Observations made by Dr. Buchanan and myself, during the epidemic of smallpox in London in 1863, on upwards of 50,000 children in various national and parochial schools, workhouses, &c., showed from another point of view the necessity of having regard to the quality and amount of Vaccination in estimating its protective value against smallpox. Some of the children examined had never been vaccinated: the large majority had been vaccinated in various manners and degrees. Of every 1000 children without any mark of Vaccination we found that no fewer than 360 had scars of smallpox; while of every 1000 children who had evidence of Vaccination, only 1·78 had any such traces. And, on further classification with reference to the *kind* of Vaccination, we obtained the following results:—

Classification of Children examined.	Proportion marked with Smallpox per 1000 Children in each class respectively.
1. Having no vaccine marks	360
2. Vaccinated—	
a. Having one vaccine cicatrix	6·80
b. Having two vaccine cicatrices	2·49
c. Having three vaccine cicatrices	1·42
d. Having four or more vaccine cicatrices	0·67
α. Having cicatrix or cicatrices of bad quality.	7·60
β. Having cicatrix or cicatrices of tolerable quality.	2·35
γ. Having cicatrix or cicatrices of excellent quality	1·22

On taking the extremes, it appeared that of children having four or more perfect vaccine marks, only 0·62 per thousand had any trace of smallpox, while of those who had a single bad mark of Vaccination 19 per thousand were scarred by smallpox. As against smallpox therefore of such extent as to leave any traces, the best Vaccination had been upwards of thirty times as protective as the worst.[1] But this numerical statement is far from expressing the whole difference; for the marks smallpox had left on the vaccinated, and particularly on the well-vaccinated, were for the most part very slight—the cases being quite exceptional in which there was anything approaching to disfigurement; while of the unvaccinated a very large proportion were seriously marked and disfigured. Many of them were really hideous to look at, and in several the smallpox had left permanent blindness or deafness.[1]

The protective power of Vaccination against smallpox extends to every race of mankind, and is seen in every climate and in every part of the habitable globe. Wherever smallpox has been known to occur exemption from attack has been the rule among the vaccinated, the exception among the unvaccinated. Abundant illustrations of this protective value of Vaccination in hot climates will be found in Dr. Kinnis's Report on Smallpox in Ceylon in 1833–34,[2] in information collected by my-

(with one vaccine cicatrix), among cases in which this was well marked, the death-rate per cent. was 3·83; among cases in which it was badly marked the death-rate was 11·91. In class *b* (two vaccine cicatrices), among cases in which these were well marked, the death-rate was 2·32; among cases in which they were badly marked it was 8·34.

[1] Sixth Report of Medical Officer of the Privy Council, pp. 91–2.

[1] One of the most remarkable illustrations of the protective power of Vaccination I have ever met with was in a ragged industrial school at Hull—a really ragged school, in which the children were of the very lowest class. Of 170 children in the school 33 had no mark of Vaccination, and 30 of these had marks of smallpox, most of them being greatly disfigured by it. Of the 137 having marks of Vaccination, only one had marks of smallpox, and these were very slight. Before detecting them I had already called the master's attention to the unsatisfactory character of the vaccine marks on the boy's arm.

[2] Report on Smallpox as it appeared in Ceylon in 1833–34, by J. Kinnis, M.D. 8vo. Colombo, 1835.

self from the East and West Indies,[1] in the Annual Reports on Vaccination in the Bombay Presidency, &c. &c. And whenever in these climates vaccinated persons have contracted smallpox, it has usually been in the mild and modified form observed in this country. Thus, in an epidemic in the Mauritius, in which the mortality among unvaccinated patients was 42·7 per cent., it was found that of such vaccinated persons as contracted the disease, only 7 per cent. died, the greater part of these having unsatisfactory marks: and in the Ceylon epidemics reported by Dr. Kinnis, who, in his observations, noted in all instances the quality of the Vaccination, it was found that, while those who were known for certain never to have been vaccinated died at the rate of 41·5 per cent., and those who could give no account of themselves, or professing to have been vaccinated, had no marks, or bad marks, died at the rate of 26·1 per cent., persons having satisfactory marks of Vaccination died at the rate of only 1·6 per cent. Of 203 fatal cases, in fact, which occurred during these epidemics, only three were in persons having satisfactory marks of Vaccination, and two were in persons who had before had smallpox. In like manner in an epidemic in Jamaica in 1851, Drs. Bowerbank and Turner lost of 477 unprotected cases 75, but of 120 vaccinated patients only 4. Both the protective and modifying power was observed in the negroes and half-castes, as completely as in the white population.[2]

In consequence of the remarkable power of Vaccination in protecting against smallpox, and the adoption of the practice universally by educated people and in annually increasing proportion by the population at large, the present average death-rate from smallpox is scarcely in any European country one-tenth part, and in those countries in which Vaccination has been most carefully carried out it is much less than one-tenth part, of what it was at the end of the last century. Thus in Sweden, where before Vaccination was discovered the average annual death-rate from smallpox was 2050, out of every million of population, during the forty years 1810–50, it was but 158; in Westphalia, where the smallpox death-rate used to be 2643, it was from 1816–50 only 114; in Bohemia, Moravia, and Austrian Silesia, it has been reduced in like manner from 4000 to 200; in Copenhagen, from 3128 to 286; and in Berlin from 3422 to 176.[1] And although our own country has been, at all events until lately, behind most others in Europe, as to the extent to which Vaccination has been adopted,[2] and behind many as to the completeness with which it has been performed,[3] yet the smallpox death-rate of England and Wales, which at the close of last century was estimated by Dr. Lettsom and Sir Gilbert Blane at not less than 3000 per million of population, was for the average of the years 1841–53 only 304, and on the average of the succeeding twelve years 1854–65 has, notwithstanding three very severe epidemics, fallen as low as 202. The following table, exhibiting the smallpox mortality in England at various periods, not only shows the influence of Vaccination in the prevention of smallpox, but is of interest also as illustrating the value of legislative and administrative action in diffusing the blessings of that practice:—

Periods compared.	Annual deaths by Smallpox in England and Wales.	Annual rate per million of the Population.
1. Average of thirty years previous to introduction of Vaccination estimated by Dr. Lettsom and Sir Gilbert Blane. .	. . . . .	3,000
2. Average of three years (1838–40[4]) when Vaccination had become to a great extent diffused, but before any public provision was made for its gratuitous performance . . .	11,944	770
3. Average of nine[5] of the years (1841–53) when public Vaccination was gratuitously provided, but Vaccination was not obligatory	5,221	304
4. Average of the twelve years (1854–65) during which Vaccination has been to a certain extent obligatory	3,967	202

[1] Board of Health, Papers relating to the History and Practice of Vaccination, 1857, p. 139.

[2] Ibid.

[1] Simon, op. cit. p. xxiii.

[2] Report of Smallpox and Vaccination Committee of Epidemiological Society, 1853.

[3] Marson, Medico-Chir. Trans., vol. xxxvi.

[4] The present system of registering deaths commenced only in 1837.

[5] During the years 1843–46 causes of death were not distinguished in the Reports of the Registrar-General.

With such proof of the protective value of Vaccination, it might well indeed be a matter of astonishment that smallpox should still annually make such ravages amongst us, if we had not ample evidence of the extent to which, through ignorance and apathy, and to a certain degree through prejudice not yet eradicated, the practice of Vaccination has been neglected, as well as of the imperfect and insufficient way in which the operation has not unfrequently been performed. The inquiries made, under the direction of the Government, from 1860 to 1864, by Drs. Stevens, Buchanan, Sanderson, and myself, into the state of Vaccination throughout England, showed that the universal performance of Vaccination in early infancy which is indispensable for the effectual protection of the community from smallpox was so far from being attained, that the proportion unvaccinated, even among children old enough to be in attendance at public infant schools, exceeded 13 per cent. Now, it is in the young unvaccinated portion of the population that the smallpox mortality chiefly occurs. Of the nearly 4000 deaths from this disease which are on an average still recorded every year in England, 56 per cent. are in children under five years of age, and as much as 70 per cent. in children under ten years of age.[1] We can have no hesitation in saying that in all the fatal cases at this early age there must, with very rare exceptions, have been neglect of Vaccination; for when that operation has been performed, even with the effect of raising a single vesicle only, subsequent death from smallpox in *childhood* very seldom indeed occurs.[2] We know further that of the mortality above ten years of age a very large proportion takes place in persons in whom Vaccination had never been performed. So that an estimate which should ascribe four-fifths of the present mortality from smallpox to the omission of Vaccination would most certainly be very much below the mark. Many, however, of the deaths from smallpox which take place after puberty—and there are on an average above 1000 deaths annually from this cause in England in persons over fifteen years of age—are (especially at epidemic periods) in individuals who had been vaccinated, and who believed themselves protected against smallpox. But as regards these, there arises the further question, *how* had they been vaccinated? Now, though we cannot, of course, answer this question as regards the individuals, we may fairly apply to the group the observations made in the Smallpox Hospital on fatal cases of smallpox in persons believed to have been successfully vaccinated. Of 402 such fatal cases, occurring in 20 years, 101 exhibited on their arms no evidence whatever of having ever had effective Vaccination; 277 had but one or two vaccine marks, and these, in 191 of them, were of imperfect character; 16 were in persons who had three cicatrices; and only 5 in persons who had been vaccinated in the way which has been shown to be the most protective—of which 5, two did not die of smallpox, but of concurrent or superadded disease.[1] We may be sure then that in only a few of those who die from smallpox after Vaccination in England, the Vaccination had been done in the best way.

In the official inquiries above referred to, in the course of which the arms of nearly half a million vaccinated children were examined, evidence was obtained of the great extent to which imperfect or insufficient Vaccination had heretofore prevailed in England; taking the country throughout, not more than one child in eight was found to be so vaccinated as to have the highest degree of protection that Vaccination is capable of affording; not more than one in three could, on the most indulgent estimate, be considered as well protected; while in more than one in four the Vaccination had been of a very inferior kind indeed, resulting in marks of imperfect character, or in only one or two marks of merely passable character.[2] These imperfections were mainly traceable to the following causes: (1) the frequency with which practitioners, in-

[1] Deaths in England from Smallpox, at different ages, for the nine years 1855–63:—

All ages.	Under 1 Year.	1—2 Years.	2—3 Years.	3—4 Years.	4—5 Years.
30,707	7,334	3,370	2,666	2,152	1,732

Under 5 Years.	5—10 Years.	10—15 Years.	15—25 Years.	25—35 Years.	35 Years and upwards.
17,254	4,078	1,169	3,552	2,422	2,232

[2] Gregory, Marson, &c., as quoted in "Handbook of Vaccination," pp. 224–5.

[1] See also the article on Smallpox.

[2] These observations were made on children most of whom had been vaccinated by public vaccinators, but a large number of whom had been operated on by private practitioners, and, without affording statistical evidence of the fact, they left a strong impression that, as a rule, the latter were less well vaccinated than the former. My own experience has satisfied me in other ways that many in the upper and middle classes in England have been very imperfectly vaccinated—the chief reason why smallpox is so much less met with among them than it is among the lower classes being that they are so very much less exposed to it.

stead of attempting fully to infect the system, had been satisfied with insertions of lymph, sufficient to produce only one, two, or three ordinary vesicles; (2) the want of due attention to the *selection* of the lymph used in vaccinating; (3) carelessness and clumsiness in the performance of the Vaccination, so that, if the operation did not wholly fail, it very frequently resulted in a less degree of effect than it had been the aim of the operator to produce; and (4) the great and unnecessary extent to which the use of preserved and conveyed lymph was substituted for the Vaccination direct from the arm, which should be the rule of all vaccinators.[1]

It is satisfactory that these, the chief causes of imperfection, are of a kind for which we have in future an obvious remedy (and which, it may be added, are now rapidly being remedied), in the proper practical instruction of vaccinators in the employment of more care and attention in vaccinating, and in better arrangements for transferring lymph. Other causes of imperfect Vaccination which are quite independent of the vaccinator, though far less widely operative than those just enumerated, must not, however, be overlooked: as, something particular in the child's constitution or condition at the time of Vaccination, which even the most experienced vaccinator may have been unable to detect; aberrations of lymph—rarely indeed, but still occasionally, met with—occurring under undefinable conditions, of which the first manifestation to the vaccinator has been the unsatisfactory result; the carelessness of parents in allowing the vaccine vesicles to be disturbed in their course and the crusts to be prematurely removed.

It has been contended that, apart from imperfections in the mode of conducting Vaccination, the vaccine lymph itself necessarily degenerates by repeated transmissions through the human body, and loses something of its infective and protective power.

The hypothesis that lymph would deteriorate by mere transmission through human bodies was started in the very earliest days of Vaccination. Jenner thought the notion a very improbable one, but could only then say of it that time was necessary to determine the question. But after many years' experience he felt himself quite justified in pronouncing the hypothesis as groundless. Drawing, as he never failed to draw, the broad and most important distinction between such deterioration of lymph as may result if in continuous vaccinations due care be not taken in selection of the lymph employed, and the doctrine of inevitable deterioration by mere transmission: he remarked, writing in 1816, that lymph in passing even from one individual to another might undergo a change which rendered it unfit for further use, but that the notion of necessary degeneration was a conjecture "he could destroy by facts:" and he referred, in proof, to the vesicles he was then producing, which, he says, "are in every respect as perfect and correct in size, shape, color, state of the lymph, the period of the appearance and disappearance of the areola, its tint, and finally the compact texture of the scab, as they were in the first year of Vaccination; and to the best of my knowledge the matter from which they are derived was that taken from a cow about sixteen years ago."[1] Numerous trustworthy observers, who had watched the vaccine disease at the introduction of Vaccination, on comparing what they had then seen with the effects produced by lymph of the earliest stocks, after a lapse of thirty or forty years, were unable to detect the slightest difference either in the character or course of the vesicles. And the same appears certainly to be the case at the present day. Having for the last thirty years been a close observer of the vaccine vesicle, and having during the last nine years enjoyed such opportunities of witnessing the practice of Vaccination in the hands of various vaccinators, and of noticing the results of Vaccination in the cicatrices left on the arms of individuals vaccinated, as have scarcely perhaps fallen to the lot of any other person, I can confidently affirm that the vaccinators of the present day who are masters of their craft do their work as surely with ordinary long-humanized lymph and infect their patients as completely as the earlier vaccinators did, producing vesicles which in character and course differ in nothing from the description that Jenner has given us, and which leave cicatrices as perfect as those which I have seen on the arms of persons who had been vaccinated by Jenner himself or by his well-known contemporary, Dr. Walker.[2] What-

[1] For evidence in detail see Reports of Medical Officer of Privy Council, iii.—vii. It is beyond the limits permissible for the present article to enter into an inquiry into the subject of the best arrangements for the performance of Vaccination and for maintaining stocks of active lymph: but it is a subject of the utmost importance, and one which I have endeavored to treat fully in my "Handbook of Vaccination," chap. vii.

[1] Letter in Baron's Life, vol. ii. p. 398. M. Bousquet appears to have overlooked this passage. See his "Nouveau Traité de la Vaccine," p. 399.

[2] See Fourth Report of Medical Officer of Privy Council, p. 64.

ever has been found unsatisfactory in the hands of other vaccinators, whether as regards the course of the vesicles or the character of the cicatrix, was fairly traceable to the causes which have been already stated, and especially to looseness and carelessness in the selection of lymph: and though I have sometimes found it desirable to recommend a vaccinator to change the stock of lymph he was employing, I have always felt it enough to take care that the stock substituted was good active lymph without troubling myself as to the time that had elapsed since it had come from the cow. It is quite true that the earliest transmissions of lymph from the cow to the human subject have usually a peculiar intensity of local irritative effect. But this result (which is so far from being an advantage that it is often found to need controlling)[1] is generally lost in a few transmissions, often indeed in one or two transmissions; besides that different primary stocks are found to differ much as to the local effect they thus produce.[2] Ceely, whose testimony on this subject is of the utmost importance, because he, more than any other living inquirer, has studied the natural disease in the cow, and has experimented on its transference to the human subject, entirely disclaims belief in the superior protective efficacy of lymph thus recently transferred over active humanized lymph.[3] Several practitioners residing near the Bridgewater Level, and in the Vale of Gloucester (in which districts the natural cow-pox is still not unfrequently seen in the dairy farms), stated a few years ago to Dr. Sanderson, that they had inoculated lymph direct from the cow with success; but all agreed that, after the first or second transmission, the results did not differ from those of ordinary Vaccination, either in the character or progress of the vesicles. It is in truth not to the cow, but to adequate care and skill on the part of vaccinators in the selection of the children and vesicles from which lymph is taken, that we must look for maintaining stocks of active lymph.[4]

Facts are equally at variance with the supposition that transmission through human bodies causes vaccine lymph to lose anything of its *protective* power. Persons vaccinated with lymph direct, or only a few removes from the cow, and others vaccinated with long humanized lymph, have been submitted to the test of variolous inoculation; but the result in each class of cases has been the same. This experiment in fact is made to hand on a large scale in every epidemic of smallpox. In each such epidemic—no matter at what period since the introduction of Vaccination—it has not been the persons vaccinated with the then current lymph, the lymph furthest from the cow, but those vaccinated some sixteen, twenty, or twenty-five years before, with lymph so much nearer to the parent source, who have been found to be the chief sufferers from post-vaccinal smallpox.

Another subject for consideration in reference to the protective power of vaccination is its permanency in the individual. It has been often said that it is undoubtedly a protection, but for a limited time only; that it wears out by age, and requires successive renewals. Although this theory is sometimes thus broadly advanced, the permanency of the protection which a single efficient Vaccination gives against smallpox is so completely established as the law of the human economy, that we need only deal with it as offering an explanation of *that proportion of cases* in which smallpox is met with in those who have been vaccinated. As regards these, we may observe in the first place that at times when epidemic smallpox prevails, and especially under circumstances of great exposure, as where children are living and sleeping in the same room, perhaps sleeping in the same bed, with a case of smallpox, the disease is sometimes met with in very young children, and occasionally very shortly after Vaccination. Even so early as the year 1806, Willan was able to record numerous cases in which a very trivial eruption, but of true variolous character, appeared in children at intervals which varied from five months to seven years after Vaccination had been performed. Similar examples have been met with in every epidemic down to the present time. But this eruption is generally so slight (except where the Vaccination has been

[1] To one unpleasant result frequently met with in early vaccinations from the cow, Mr. Ceely has especially called attention in a recent communication; it is the occurrence of a special vesicular vaccine eruption called by the Germans "Nachpocken," which causes a good deal of temporary disfigurement and annoyance, and sometimes, when copious, severe and even dangerous symptoms. (See Brit. Med. Journ., Jan. 7, 1865.)

[2] Handbook of Vaccination, chap. x.

[3] Observations on Var. Vacc. in Trans. of Prov. Med. Assoc., vol. viii.

[4] The important point, it seems to me, is that vaccinators should not be induced to look to anything extrinsic and inevitable for an explanation of deteriorations which, if they occur at all, may be more properly ascribed to want of skill and care somewhere. So long as this is kept in mind, there can of course be no objection to the introduction at any time, when opportunity offers, of fresh stocks; but those who are about to undertake the transplantation of lymph may meet with some difficulties, and would do well to study Mr. Ceely's admirable "Observations on the Variolæ Vaccinæ," in Trans. of the Prov. Med. Assoc., vols. viii. and x.

spurious, or ineffective) as to attract little attention, or only to attract observation on account of its amazing contrast with the variola of unprotected children. After puberty smallpox is met with in vaccinated persons more frequently, and though no doubt generally modified, is, if there have been any imperfection in the original Vaccination, in not a few cases severe and even fatal; even after the most complete Vaccination, cases are met with, some of which are severe, and a very few —not much more than the half of 1 per cent.—fatal. It has been found, however, in the experience of the army, in that of the Smallpox Hospital, and in practice generally, that cases of smallpox after Vaccination occur chiefly between the ages of fifteen and twenty-five, and that after the age of twenty-five they sensibly diminish. Thus, while in the army the deaths from smallpox among soldiers under twenty years of age were 3·4 per 10,000 of aggregate strength, and those among soldiers from twenty to twenty-five years old 3·1 per 10,000; the deaths from this cause among the soldiers above twenty-five years old have been but 1 per 10,000. And from the observations of Professor Heim, on 1055 cases of smallpox in vaccinated persons, examined not in relation to the age but specially as to the number of years that had elapsed since Vaccination, we find that while the average annual number of cases that occurred in the first twelve years after Vaccination was twelve, and in the next thirteen years was over fifty-one, the average for the following ten years was under twenty-five. The real explanation appears to lie not in the weakening influence of time, which would be a progressively increasing influence, but rather in certain disturbing influences of which, no doubt, puberty is the chief.

The liability of persons to renew their susceptibility to smallpox is, unquestionably, very much a matter of diathesis. It is frequently met with in various members of the same family; and I know of many instances in which, under the same circumstances, other members of these families have suffered from second attacks of smallpox.

Revaccination.—The numerous instances in which, from whatever cause, the protection of Vaccination has proved insufficient, have led to the very frequent adoption of late years of revaccination. By many this practice is looked upon as only called for or useful where there has been some defect in the primary Vaccination; but there is reason to believe that it has a use beyond this—that it extinguishes that renewed susceptibility to smallpox which, it has been already pointed out, occurs in an indeterminate proportion of persons after even the most perfect Vaccination.

When a child has unfortunately been imperfectly vaccinated, no doubt the best thing that can be done is to vaccinate it again. But the chance revaccination may afford of correcting original imperfection must never be held for a moment to absolve any practitioner from the pains he is bound to bestow to make his original Vaccination full and complete. For what daily happens is this:—A child is vaccinated and takes badly; either at once, or at some no distant period, it is vaccinated again, and perhaps a third time, and cannot be made to take; it grows up, gets smallpox, and very likely dies. Or this happens: The parents are directed to bring it again at some specified time, and fail to do so, and the same fate as in the other case befalls it. Take it at the best, an originally imperfect or incomplete Vaccination is a very great misfortune.

But supposing it to have occurred, how is the practitioner to act? Is he to revaccinate at once, or is he to wait till puberty, when the chief danger of insufficient Vaccination manifests itself? He must be guided in determining partly by the degree of imperfection, and partly by the liability to exposure to smallpox. If the Vaccination has been spurious, irregular, or disturbed in such a way as to divest it of protective power, he would, unless there were something in the child's then state of health which might have been the cause of these imperfections or irregularities, at the earliest opportunity vaccinate it again; if anything were found amiss with the child, he would of course wait till that was corrected. Very likely the Vaccination would not take, for quite spurious Vaccination will often prevent real Vaccination from taking effect afterwards. But the chance must be given, and given with every care to produce effect. If such should result, even though it be only the ordinary spurious effect of a revaccination, he will have done all he can do, and need recommend no further proceedings till puberty—at all events unless there should arise some immediate danger of smallpox. But if there be no local evidence that the lymph applied on the revaccination had been absorbed, the operation should be repeated at intervals until he is satisfied that the child is, for the time at all events, insusceptible. Supposing, however, the result of the primary Vaccination have been one of insufficiency rather than of imperfection, —that is, if a single vesicle have risen instead of the four or five it was desired to produce, but that vesicle has run its course perfectly,—I do not usually recommend, except under circumstances of danger, any further Vaccination till the child grows up.

The same rule guides me in judging from the cicatrices left on the arms of young children whether revaccination is called for. If these be decidedly imperfect in character, I advise the parents to seek revaccination with as little delay as possible; but if the cicatrices be only deficient in number, or if the character, though less strikingly good than it might be, is yet genuine, I hold the child to be pretty safe up to the age of puberty, but strictly enjoin its revaccination then.

The occurrence of a case of smallpox in a house leads to a stricter rule as regards all the inmates. Those who are past or approaching puberty should, except such as have already since puberty been successfully vaccinated, be revaccinated at once; and those under puberty who have two or more thoroughly characteristic marks should not be meddled with; the children whose marks are not thoroughly good, or who have but a single good mark, should be revaccinated. In a crowded court this course should not be limited to the house in which the smallpox appeared, but extended to each house. If, on the outbreak of an epidemic of smallpox, this plan were uniformly adopted, together, of course, with the immediate Vaccination of all who in the house or court were found unvaccinated, there can be very little doubt the epidemic might be cut short, and very certainly indeed the occurrence of fatal or severe cases of smallpox would be all but entirely prevented.[1]

Revaccination about, or after, puberty is of extreme importance when the original Vaccination has been anything short of Mr. Marson's highest class, and is necessary in proportion as it falls short of it: but it seems also certain that those whose original Vaccination has been complete may derive additional security from a revaccination at, or after, this period of their lives. We have already seen that a certain proportion of the most thoroughly vaccinated contract smallpox, though no doubt with comparatively little danger, after growing up: as many as 367 such cases were admitted to the Smallpox Hospital in the great epidemic of 1863. But after effectual *revaccination*, smallpox, even in its most modified form, is found very rarely, or scarcely ever, to occur. Thus, Heim found that in five years there occurred among 14,384 revaccinated soldiers in Wirtemberg, only one instance of varioloid, and among 30,000 revaccinated persons in civil practice only two cases of varioloid, though during these years smallpox had prevailed in 344 localities, producing 1,674 cases of modified or unmodified smallpox among the not revaccinated, and in part not vaccinated, population of 363,298 persons in those places in which it had prevailed. In the Prussian army, since the introduction of systematic revaccination of all, the annual deaths from smallpox (which at one time were 104) have not averaged more than 2; and on analysis of 40 fatal cases that occurred in twenty years, it appeared that only 4 were in persons who were said to have been successfully revaccinated.[1] Other national experience might be referred to, but it will be better to have recourse once more to Mr. Marson's very precise statements. In the thirty-two years and upwards that he has been connected with the Smallpox Hospital, no nurse or servant has taken smallpox, he having taken care always to revaccinate them on their coming to live in the hospital; and at a time when a large number of workpeople were employed for several months about the hospital, most of whom consented to be revaccinated, two only were attacked by smallpox, and these two were amongst the few who were not revaccinated.

These broad facts, while they show the great importance of the practice of revaccination, attest at the same time the utter uselessness and folly of repeating this operation again and again in the same individual, as seems to have become a recent fashion, whenever epidemics of smallpox arise. One thoroughly primary Vaccination to start with, and one careful revaccination after puberty, so conducted as to give evidence that the lymph was absorbed, are all that is necessary for the complete protection of the population against smallpox. No doubt by the first Vaccination, nine out of ten are perfectly well and permanently protected; but who can predicate of any individual whether he is one of the nine or the exceptional one?

The revaccination, however, must be done with all the care that should be employed to secure the success of a primary Vaccination; and the practitioner should always endeavor to get some evidence that the lymph is absorbed. In many cases, however, even after repetition of the Vaccination, this will not happen; and in these cases a further attempt may very properly be made at some future time. Revaccination should always be done by preference when it can be done leisurely, and as part of the ordinary work of Vaccination, and not under the alarm and influence of panic.[2] The wholesale

[1] See a remarkable illustration of this in Third Report of Medical Officer of Privy Council, p. 50; and see also illustrations in Report of Smallpox and Vaccination Committee of Epidemiological Society, 1853.

[1] Simon, op. cit., pp. xxxv. xxxvi.

[2] Under these circumstances the demand for revaccination often becomes so great that it is difficult to find lymph to meet it. I have known revaccination-lymph used under these circumstances for performing other revaccinations, and very bad arms resulting.

and sham revaccinations, which would under such circumstances appear to have been practised of late, will tend much, it is to be feared, to bring the practice into disrepute and contempt.[1]

The following table, showing the results of revaccination in each 1000 individuals revaccinated in the Wirtemberg army in 1831–5, and in our own army in 1861, will give some indication of the local results that may be expected from the performance of revaccination in adults :—

Persons in whom the Revaccinations were performed.	Degree of success of Revaccination.	In those who bore marks of previous Smallpox.	In those who bore good marks of previous Vaccination.	In those who bore doubtful or imperfect marks of previous Vaccination.	In those who bore no marks of previous Vaccination or Smallpox.
Wirtemberg Army, 1831–5 (13,861 cases)	Perfect	319·5	310·4	280·7	337·3
	Modified	248·1	280·5	259·	191·1
	None	432·3	409·2	460·4	471·6
		1000	1000	1000	1000
Soldiers in Brit. Army, not recruits, in 1861 (2,053 cases)	Perfect	451·4	484·6	236·8	326·
	Modified	159·6	157·4	505·3	277·5
	None	389·0	358·0	257·9	396·5
		1000	1000	1000	1000
Recruits in Brit. Army in 1861 (4,395 cases)	Perfect	345·5	407·3	461·3	527·3
	Modified	266·8	240·8	301·3	202·6
	None	387·7	351·9	237·4	270·1
		1000	1000	1000	1000

A perfect local result following a revaccination is constantly appealed to by practitioners as evidence that the person in whom it was developed was liable to take smallpox, or, at all events, more liable than those in whom imperfect or no results followed. But this conclusion appears to me by no means warranted. If it were, these curious results would follow, that (taking as our guide the observations in the Wirtemberg army) 319 out of 1000 persons having had smallpox, 310 out of 1000 who had been well-vaccinated, and only 281 out of 1000 who had been ill-vaccinated, were in present danger of taking smallpox ; and of the soldiers (not recruits) in our own army, 451, 485, and 237 would represent the ratio in the three classes respectively, which is clearly a *reductio ad absurdum*. We cannot, indeed, that I can see, draw from the local phenomena of revaccination any inferences whatever as to the state in which the revaccinated person was as to liability to smallpox. Jenner himself, indeed, pointed this out in his first treatise, and showed that the natural cow-pox might be induced again and again in persons who, being protected against variola by their first attack of cow-pox, could not be variolated either by inoculation or by exposure, and also that cow-pox might be made to take on those who had had smallpox.[2]

The utility and necessity of revaccination stand not upon any speculative reasoning from the local effects it produces, but upon the broad grounds of observation and experience.

IV. Relations of Variola and Vaccinia.—Jenner believed the cow-pox of the cow and the smallpox of the human subject to be essentially the same disease, as he implied when he denominated the former Variolæ Vaccinæ : he further believed they had a common origin—the grease of the horse. He did not himself perform any inoculation of cattle with the lymph of human variola. But, as early as 1801, Gassner, of Günsburg, by inoculating eleven cows with smallpox matter, produced on one of them vesicles having all the character of vaccine vesicles, and from which a stock of genuine vaccine lymph was obtained. Another successful variolous inoculation of cows, at the Veterinary College at Berlin, is referred to by M. Viborg of Copenhagen so early as 1802, but no details are given respecting it. In 1830, Dr. Sonderland, of Barmen, stated that he had infected cows with the variolous contagion by enveloping them in blankets taken from the bed of a patient who had died of smallpox, and also hanging the blankets up round the head of the animal that it might breathe the effluvia arising from them. The cows, he says, in a few days manifested the symptoms of cow-pox, and lymph taken from them produced genuine vaccine vesicles in the human subject. Dr. Sonderland's experi-

[1] See Sixth Report of Medical Officer of Privy Council, p. 113.

[2] Obs. on the Variolæ Vaccinæ, pp. 21, 22, and p. 51 ; Continuation, &c. p. 25.

ments were repeated in India by Mr. Macpherson, in this country by Mr. Ceely of Aylesbury, and in various places abroad, without success. But Mr. Ceely was able, by the much more satisfactory process of direct inoculation with smallpox virus (in February, 1839), to induce vesicles in two out of three sturks operated on, and with lymph taken from these to vaccinate many children, from whom a regular lymph-stock was continued.[1] In 1840, Mr. Badcock of Brighton, without previous knowledge of Mr. Ceely's experiments, succeeded also in variolating the cow, and deriving thence a stock of genuine vaccine lymph: and since then he has, by inoculating cows with the lymph of human variola, raised stocks of vaccine lymph for use on no fewer than thirty-seven separate occasions. The common origin, then, of smallpox and cow-pox may thus be considered as established.

The case, as regards the grease, appears to stand thus: the disease really known as grease has nothing to do with cow-pox or smallpox; but the horse is subject at times to a true equine pox, which is precisely of the same kind as the smallpox in man and the cow-pox in the cow. This disease is met with as an epizootic, particularly when cow-pox is epizootic among cows and smallpox is epidemic. The equine matter used by Jenner himself,[2] and that used by Sacco and others, for the purposes of Vaccination, were derived from this equine pox; and on various other occasions equine lymph has been employed.[3]

V. Alleged Dangers of Vaccination.—Space renders it quite impossible for me to enter into the consideration of certain objections that have been urged from time to time to the general utility of Vaccination, or have been thought to prove that it was injurious: such as the displacement-of-mortality theory of M. Carnot, the allegations that scrofula and typhoid fever have become more frequent in consequence of the introduction of the practice (being in fact, it has been said, "vaccinational varieties or introversions of smallpox"), and other like absurdities.

The practitioner who desires to acquaint himself with all that has been brought forward on these subjects will find the fallacies of the various statements thoroughly exposed, and the questions themselves finally settled, in the admirable memoir which Mr. Simon has prefixed to the Papers relating to the History and Practice of Vaccination, to which I have already so often referred. What is before us now to consider is of more limited scope, but of much greater real importance, and relates not to whether Vaccination is a proceeding we ought still all to adopt, but to the special care and precaution which should be brought to its practice. Is it possible in vaccinating to communicate accidentally other diseases—as cutaneous diseases, scrofula, or syphilis?

Reserving for separate consideration what has to be said regarding syphilis, I may state that the invaccination of cutaneous and scrofulous diseases, though a popular, has never been a professional belief. These diseases are met with constantly in infancy and childhood, as well in the unvaccinated as in the vaccinated, from the influence of various exciting causes acting on constitutional predisposition. I am not aware of any facts which prove, or even render probable, their greater frequency among vaccinated than among unvaccinated children of the same ages respectively. When eczema and other eruptions manifest themselves, as they may do, shortly after Vaccination has been performed,[1] this is held by some to be—and no doubt very generally is—a mere coincidence, and due in reality to one of the various eruption-producing influences to which children at the usual age for Vaccination are subject, such as teething, &c. But there are many medical men who hold, and with great probability, that in a part at least of these cases the Vaccination may itself have been, by the febrile action it set up, the *exciting* cause. No medical authorities believe in the transference of scrofulous and cutaneous diseases from one child to another by Vaccination. Parents, however, as Mr. Marson observes, "are unwilling to believe that there is anything constitutionally wrong in their offspring; and, when other diseases follow, Vaccination gets blamed for what is really and truly due to other causes." Hence, parental complaints that disease has been set up in this way are not unfrequent; but, as showing the prejudice under which such complaints are preferred, it may be worth while to state that, though I have carefully investigated a great number of them, I have never yet in a single instance found that the child from whom the lymph was taken was suffering from the disease it was said to have imparted.

[1] Obs. on Var. Vacc. in Transactions of Prov. Medical Assoc., vol. viii. Three years before, Dr. Thiele, of Kasan, in Russia, had made similar successful experiments (Henke's Zeitschrift, t. xxxvii. h. 1), which were not known to Mr. Ceely, and, in fact, were not published till 1839.

[2] Baron's Life, vol. i. p. 254.

[3] The limits of this article do not allow me to enter further into this interesting subject, which is fully discussed in chaps. ii. and iv. of my "Handbook of Vaccination."

[1] Such eruptions as are part and parcel of the constitutional symptoms of Vaccination have been already treated of in Sect. i.

Those who have had most to do with the performance of Vaccination, on the one hand, and those who have been most concerned in the treatment of infantile disease, on the other, concur in the belief of the non-communicability of disease by Vaccination. Mr. Marson, in the performance of 50,000 vaccinations and more, "has never seen other diseases communicated with the vaccine disease, nor does he believe in the popular report that they are so communicated."[1] Such also was the experience of the late Mr. Leese, whose opportunities of observation were scarcely, if any, less.[2] Sir W. Jenner stated some years ago that at University College Hospital and at the Hospital for Sick Children he had had, in six years, more than 13,000 sick adults and children under observation, and that in no case had he reason to believe, or even to suspect, that any constitutional taint had been conveyed from one person to another by Vaccination.[3] Dr. West's experience on 26,000 infants and children under his care in seventeen years is to the like effect; and in stating that he had seen nothing in that time to make him believe that Vaccination excites cutaneous eruptions in any but very exceptional cases, he referred such exceptional cases to a disposition in the children themselves, brought out by the Vaccination as it might have been by teething.[4] And Professor Paget, speaking from his large experience among children in the out-patients' room at St. Bartholomew's, and enumerating some of the causes which develop cutaneous diseases in young children, says, "Now, Vaccination may do, though I believe it very rarely does, what these several accidents may do; namely, by disturbing for a time the general health, it may give opportunity for the external manifestation and complete evolution of some constitutional affection, which, but for it, might have remained rather longer latent." "This is," he adds, "the worst thing that can with any show of reason be charged against Vaccination; even this can very seldom be charged with truth."[5]

Although the direct inoculability of the syphilitic poison from one human being to another distinguishes it remarkably from cutaneous diseases generally and from scrofula, I should still, but for certain recent occurrences which have excited much attention, and to which I shall immediately advert, not have thought it necessary to speak separately of the communication of that disease by Vaccination. It was indeed not only included with other diseases, but was specially so included, in the opinions of the distinguished practitioners whom I have just cited; Dr. West informing us that there had never come under his notice "any instance in which there seemed the slightest pretext for supposing that syphilis had been communicated to infants through the medium of the vaccine lymph," and Professor Paget that he does not remember "to have heard infantile syphilis ascribed to Vaccination, frequent as the instances of it (inf. syphilis) are among the out-patients." In the experience of Mr. Marson, Mr. Leese, or the National Vaccine Establishment of England, such a case has never been met with. And referring generally to the experience of practitioners at home and abroad, it may safely be said that there is scarcely a subject in medicine in which there has been, till within the last few years, a more general concurrence of opinion.[1] One broad general fact seemed to be, and still probably is, conclusive on the matter. In the seventy years that have passed since Vaccination was introduced, it seems certain that, "if syphilis could be diffused by the vaccine lymph of children with an hereditary taint of that disease, this possibility must long ago have been made evident on a scale far too considerable for question."[2]

Scientific authority unites with general medical experience to negative the possibility of the vaccinal communication of syphilis, — it being implied always of course that the vaccination is true vaccination, *i. e.*, with vaccine lymph taken from a true Jennerian vesicle. Professor Paget states the pathological grounds for disbelieving the possibility of any such communication to be, (1) because *infantile syphilis* (which alone need be here considered), though conveyable in some instances by its own peculiar morbid products, does not render the blood of the patient capable of directly conveying the disease; and (2) because, if the blood of a syphilitic child could so modify the vaccine disease within it as that the vaccine lymph should be capable of conveying any other disease, there is every reason to believe that the vaccine vesicle formed in the diseased child would be modified in correspondence with the modified lymph. "All pathological researches," he observes, "accumulate the evidences of the constant correspondence between the material in the blood, on which each specific disease depends, and the morbid structure, by which each is manifested. Thus the

[1] Papers relating to the History and Practice of Vaccination, p. 25.
[2] Seaton on Protective and Modifying Powers of Vaccination, p. 23.
[3] Papers relating to, &c., p. 75.
[4] Ibid., p. 146.
[5] Ibid.

[1] See replies of eminent members of the profession to the queries addressed to them (in 1856) by Mr. Simon.
[2] Simon, in Papers, &c., p. lxvi.

transformations of the syphilitic poison are indicated in the successive external characters of the primary, secondary, and tertiary affections; the transformation of the scarlatina poison by its regular symptoms and its sequelæ. And so, if the vaccine virus were capable of any transformations besides those which mark its regular influence in each patient, such transformations, we may be sure, would be indicated by corresponding and evident changes in the vaccine vesicle. In other words, if the vaccine were changed into any other virus, there would be no vaccine vesicle."[1] The opinions of Hebra and other distinguished pathologists are to the like effect.

Direct experiments made on a large scale, at many times, and by many individuals, have led in every single instance to the same conclusion. M. Cullerier and other experimenters in France, especially M. Taupin, have taken lymph on purpose from syphilitic children, have vaccinated healthy children with it, and watched the result. In no instance has syphilis been communicated. Heim made similar experiments in Germany with the same result. It was found no more possible to produce syphilis by vaccine lymph taken from a syphilitic child, than it is to produce smallpox by lymph taken from vaccine vesicles on the arms of patients who are incubating, or suffering from that disease. This, it is well known, has been done hundreds of times, but never has smallpox been thus communicated or anything but a vaccine vesicle resulted.[2]

Cases had indeed been from time to time recorded, in which it was believed or suspected that syphilis had been communicated form one person to another along with Vaccinia. But, in searching the literature of sixty years, notice can scarcely be found of a dozen occasions in which events of this kind were supposed to have happened, where the circumstances are stated with sufficient detail to enable us to form a judgment of the value of the evidence; and, in all of these, the details, when they were examined, were found so wanting in scientific precision, and so open to sources of fallacy, that the cases had been rejected as worthless for proof. So that about eight years ago the mind of the profession generally, never till then, so far as I am aware, very seriously disturbed, may be said to have been at rest on the subject. This quietude, however, was soon afterwards broken by a very circumstantial account of a singular outbreak of endemic syphilis at Rivalta, in 1861, traceable, it was said, to a vaccinal origin, and by one or two cases—especially by one which occurred in the wards of M. Trousseau, at the Hôtel Dieu in Paris, in 1861—which afforded, it was alleged, direct proof of vaccino-syphilitic inoculation. To the events then announced a new and adventitious interest was imparted by the bearing which proof of the occurrence of vaccino-syphilitic inoculation, if it could be afforded, would have on the doctrines then being sharply contended for by rival schools of syphilographers.[1] All the cases detailed from the earliest days of Vaccination were eagerly hunted up; the discussions which ensued caused a few fresh (alleged) cases to be recorded; and the evidence thus collected has been held by certain authorities in syphilis to establish that the inoculation of syphilis in vaccinating from a genuine vaccine vesicle, though of excessively rare occurrence, is yet possible, supposing that the child from whom the lymph was taken had, or was incubating, syphilis, and that some of the blood of the syphilitic child was inoculated along with the vaccine lymph. The allegation is not, it will be observed, of carelessly syphilizing instead of vaccinating, as by taking syphilitic matter instead of, or along with, vaccine lymph, in which cases we might expect, of course, syphilis only, or syphilis with Vaccinia, to result. It is that in the ordinary performance of Vaccination (the absence of syphilitic matter on the lancet being presupposed), syphilis may be accidentally implanted along with the vaccine. It is not contended, in the face of the accumulated evidence to the contrary from pathological science, general experience, and direct experiment, that vaccine lymph would impart syphilis or any other than its own specific contagion. But it is said that there may be twofold inoculation, and the communication may take place through the blood.[2] Lymph and syphilitic

[1] Papers relating to, &c., p. 139.

[2] Simon, op. cit. p. 43.

[1] A reader of the recent discussion in the Académie de Médecine will be surprised at the sort of jubilant tone with which the vaccinal inoculation of syphilis is hailed as giving the "dernier coop" to a certain "école syphilographique," &c. &c., as well as at the mere hearsay evidence on which facts are pressed into the controversy.

[2] In experiments with the mixed viruses made by Sigmund, by Friedinger, and by Boeck, syphilis *only* was produced: there was no Vaccinia. In one of Friedinger's experiments, however, and in one out of many experiments by Baumès and Sperino with the matter of *soft* chancre mixed with vaccine lymph, an *irregular* vesicle resulted, which in its development had some considerable points of resemblance with a vaccine vesicle, but the matter from which, taken on the eighth day, produced only chancre (De la Syph. Vacc., Paris, 1865, p. 280). As regards true syphilis, however, one cannot see any reason why, if its inoculable products were mixed with vaccine lymph and inserted on the arm of a

blood being inoculated together, each within its own period of incubation will produce its own specific results. The vaccine disease will first run its course, and this being over or approaching its end, the effects of the blood inoculation will manifest themselves. But even of the possibility of accidental communication in this way it appears to me that the very strictest proof, and a complete absence of every possible fallacy,[1] are demanded: (1) because we have hitherto been without any evidence whatever of the direct communication of *infantile* syphilis by inoculation of blood; (2) because inoculation of syphilis by blood of the adult is a matter of very great difficulty and very frequent failure, requiring always the exposure of a very large absorbing surface, while in the cases now brought forward the inoculation seems to be effected wholesale and by the minutest drop of blood; and (3) because in the numerous vaccinations which in the course of seventy years must have been done accidentally from syphilitic children, blood must often have been invaccinated, and in many of the experimental vaccinations from syphilitic children blood was purposely mixed, and yet no syphilis had resulted.

Now the cases which have been brought forward, whatever ground they may give for caution (and in a matter of such extreme consequence there can never be too much caution), do not appear to me to afford the strict proof requisite, or indeed anything like it; on the contrary, each one of them is wanting in some essential point, or is open to some source of fallacy. Either there was no evidence that the child said to have originated the syphilis was at any time syphilitic; or it was not shown that the alleged syphilitic vaccination was not in fact a syphilitic inoculation *instead* of a vaccination; or there was a possibility, and indeed a probability, that the lymph used had been mixed with the inoculable products of syphilis; or there was reason to believe that the syphilis which developed itself after the Vaccination had an independent origin; or the facts were inquired into at too great a distance of time, and depended too much on the statements of ignorant persons to be wholly relied on. Thus, in the occurrences at Rivalta the circumstances were not inquired into till four months after their origin.

It is not intended in this article to consider the alleged cases of vaccino-syphilitic inoculation in detail. In none of them is the proof stronger than in the two to which I have already referred—the case of M. Trousseau at the Hôtel Dieu, and the syphilitic endemic at Rivalta; it will be sufficient, therefore, to examine these. In M. Trousseau's case a young woman was revaccinated from a child, healthy at the time, and, so far as was known, continuing healthy, from which child four children received their primary Vaccination, went through it perfectly, and (certainly) had no subsequent syphilitic affection. Only some small papules arose on the arm of the young woman, and the revaccination was considered to have failed. She remained in the hospital a month after the Vaccination, and *after being out another month* came back with two undoubtedly syphilitic sores on the arm on which she had been vaccinated. It was afterwards known, but not at the time, that she was a young woman of loose character. Now, assuredly no one who knows the extraordinary situations in which chancres have been met with, such as the cheek, the corner of the eye, all sorts of situations in which there was no suspicion of Vaccination, can say that there was no fallacy in this case, especially when we consider, first, that there was not a shadow of proof adduced that the child from whom the lymph was taken ever had syphilis, and there was every presumption that it had not; and secondly, the unusual length of the syphilitic incubation, supposing it dated from the time when the Vaccination was done.

The syphilitic endemic at Rivalta was a very remarkable occurrence, and under any point of view is of the deepest interest. Four other such endemics, said to have occurred in 1814, 1821, 1841, and 1856 respectively, have been ascribed also to a vaccinal origin; and not the least curious part of the matter is that all these should have occurred in Italy, and that none like them should have been met with in any other country. In their mode of communication, apart from the Vaccination—for many persons were affected who were not at the time the subjects of Vaccination—they bear a close resemblance to what we read of endemics of sibbens and other syphiloid diseases, recorded before Vaccination was known; not only was there communication of disease from babies to their nurses and from wives to their husbands, but children infected one another by the act of kissing, and we are even told that when poor people were crowded together in confined and neglected dwellings, whole families were affected.

child, each infection should not within its own period of incubation produce its own specific results; and this is, in fact, by far the most probable explanation of one or two occurrences in which there seems authentic evidence of vaccinia and syphilis having been received at the same operation. (See Handbook of Vaccination, pp. 317–18.)

[1] See particularly the observations made as regards some of these by Simon, op. cit. p. lxvi.

In the Rivalta endemic, the alleged vaccinal origin was made the subject of careful inquiry by a scientific commission. But unfortunately this inquiry did not take place till four months after the outbreak; and, as in no single case of the children said to have been syphilized by Vaccination had any application been made to a medical man on account of the condition of the arm, facts and dates, requiring the closest and most accurate observation, with careful record from day to day, had to be taken at that distance of time on the testimony of the parents and villagers. Under these circumstances we are not surprised that different conclusions were arrived at; and that though Dr. Pachiotti and the other commissioners, after a most careful and painstaking inquiry, reported themselves satisfied of the vaccinal origin of the disease, Sperino, who also went to Rivalta and saw the cases, and treated some of them afterwards at Turin, was equally satisfied that this origin was altogether independent of the Vaccination. The story, as regards the vaccinal origin, is this:—

A child (Chiabrera) apparently in good health, but really incubating syphilis, was vaccinated with some lymph obtained in a tube; this child's arm was used *on the tenth day* for the vaccination of 46 children, and one of these 46 children, named Manzone, *again on the tenth day* furnished lymph for vaccinating 17 children; of these 63 children 46 had, within two months, a disease considered by the commission to have been syphilis,—the syphilitic symptoms having manifested themselves in some cases *within ten days*, and *as a mean*, at twenty days from the Vaccination. It need scarcely be pointed out how irreconcilable these dates are with all that we have been taught as to the incubation period of constitutional syphilis. But supposing this teaching to have been erroneous (and, unless it is, the Rivalta cases as connected with Vaccination fall of themselves), and supposing that it is possible to produce the primary symptoms of constitutional syphilis within ten days of the inoculation of the poison, and that from Chiabrera's arm the whole mischief arose, there is still the question *from what sort of vesicle* on his arm was the lymph taken? We have not only the higher authority of Ricord that the chancrous pustule, "initial lesion of the primary syphilic ulcer when it develops itself on the skin," has characters which may cause it to be confounded by careless persons with the vaccine pustule: but we know also that such a vesicle as may be produced by mixture of the matter of soft chancre with vaccine virus, *though according to the description given it would be impossible for any careful person to confound it with the regular vaccine vesicle*, has yet much of the vaccine character about it. Now of the vesicles on Chiabrera's arm we have no reliable account: nor of the vesicles on the arms of any of the other children, at the period of their so-called Vaccination. So far as we have details of the Vaccination, they are not such as to give us any confidence in the operation, and it is quite an open question whether the children were not carelessly syphilized instead of being vaccinated.[1] When the cases were seen by Dr. Catt, or by the Commission of Inquiry, they presented either so many syphilitic sores, or cicatrices which, according to the description given, had no vaccine character about them: and the revaccination of five of the children afterwards without effect (a revaccination which it is not stated was repeated) is far from being conclusive.

In the years which have passed since these occurrences became the subject of discussion, attention in this country has been wide awake to find any that should be like them. None have been met with. On one occasion one of our ablest, as he is

[1] Mr. Simon informs us that in a child having latent syphilis he has known a clean incision, made in performing a trifling surgical operation, develop in a few days a syphilitic sore. I do not say that Chiabrera's arm had such a sore on it; the state of his arm is the very thing regarding which we want evidence. Knowing the careless and miscellaneous way in which Vaccination has sometimes been carried on, and the ignorant hands into which its performance has sometimes fallen, I am rather surprised that accidents have not more frequently occurred. It is only a few years ago that I was the means of stopping a druggist, who was rather an extensive vaccinator, from proceeding to vaccinate some children from *an open sore* upon an arm which had nothing of vaccine character whatever about it, but which he assumed to be a vaccine sore, because he had vaccinated the child a week before: and the details of some of the Continental vaccinations in which accidents have occurred are truly astonishing. Further, the importance and necessity, in alleged cases of vaccino-syphilitic inoculation, of having precise details, and of using care and reserve in the admission of statements, are well illustrated by the particulars of a case often referred to in the discussions in the Académie to show that syphilis had been implanted by a properly performed Vaccination. It is the case well known as that of "Le Vétérinaire B."—in which 19 out of 24 persons revaccinated by him had afterwards signs of syphilis, the stuff with which all were vaccinated being, it was said, vaccine taken "sur un infant qui était fort, et qui paraissait complètement sain;" but concerning whom, further inquiry elicited this important information, "*On sut depuis* que l'éruption vaccinale ne s'était pas fait régulièrement chez lui, que le huitième jour il n'y avait pas encore trace de boutons!"

one of our most candid workers in syphilis, met with a case which he stated to the Medico-Chirurgical Society was one of syphilitic infection communicated in vaccinating, but it turned out that even his experienced judgment had been deceived, and that, as he allowed subsequently, he had mistaken for a syphilitic sore a merely degenerated vaccine vesicle. Nor with all his vast experience of syphilis has he to this day met with any case in which there was ground for believing that that disease had been communicated in vaccinating.

The real lesson which, as it appears to me, is derivable from the alleged cases of vaccino-syphilitic inoculation, is the extreme care with which Vaccination should be conducted. We must not only be on our guard against any possible admixture of syphilitic matter with our vaccine lymph, either through the lancet or otherwise; but we must be careful that our lymph itself is taken only from the healthiest children, from the most perfect and regular vesicles at the proper period of their course, and that it is pure unmixed vaccine lymph, free from the slightest stain of blood. We have no right to run a *merely possible* risk that can be avoided.

GLANDERS—EQUINIA.

By Arthur Gamgee, M.D., and John Gamgee.

Definition.—A febrile disease, due to the introduction into the system of a specific poison, originating in the horse, ass, or mule, and communicated directly or indirectly from them to man.

It is usually ushered in by rigors, which are followed by articular pains, and great prostration. There is more or less affection of the lymphatic vessels and glands, which inflame and suppurate. Ulcerations appear on the pituitary mucous membrane, from which there flows an aqueous or purulent discharge. A pustular eruption often occurs on the surface of the skin, which in parts becomes affected with inflammation of erysipelatous character. Abscesses form in the subcutaneous cellular tissue. The disease is usually fatal.

Nomenclature and History.—Unthe name of Μύλις, Aristotle[1] described a disease affecting the ass, which was probably identical with the "malleus" or "morbus humidus" which an early writer on veterinary medicine, Vegetius, subsequently described as affecting the horse, and with the disease to which, from an early period, the name of Glanders has been applied by English writers. The term Glanders includes several affections, which, undoubtedly due to the same specific virus, must be looked upon as mere varieties of one disease, but which, differing very remarkably in character, were for a long period of time considered to be altogether distinct.

Vegetius, and the authors who followed him, described under the term Malleus humidus, Morbus humidus,[1] Cymoira,[2] Cimoria,[3] Capitis morbus, that variety to which the term Glanders has been restricted by English writers—to an affection of horses which is characterized by ulcerations of the Schneiderian mucous membrane, accompaaied by a discharge from its surface, and by enlargement and induration of the submaxillary glands; which may run a long or short course, and which may be, but often is not, accompanied by marked constitutional symptoms. They described in addition, under the terms Morbus farciminosus,[4] Vermis equi,[5] Vermis volativus,[6] Farcina equi,[7] Turtae, cutis equorum,[8] Glandulæ et scrophulæ equi,[9] an affection which has now been proved to be merely a variety of the one described, and of which the striking and characteristic feature is the formation of abscesses and swellings in the course of

[1] Aristotle, De Hist. Anim. lib. viii. cap. xxv.

[1] Vegetii Renati Artis Veterinariæ, sive Mulomedicinæ, libri quatuor, &c., lib. i. c. vii.

[2] Laurentius Rusius, c. lxxi. p. 72, quoted by Heusinger.

[3] Jordanus Ruffus, c. xvi. p. 48, quoted by Heusinger.

[4] Vegetius, op. cit. lib. i. c. iii.

[5] Jordanus Ruffus, c. i. p. 23.

[6] Ibid. c. ii. p. 27.

[7] Albertus Magnus, p. 92.

[8] Ibid. p. 589.

[9] De Crescentiis, lib. x. c. xii. p. 275.

the lymphatic vessels and glands, and of small tumors beneath the skin, and which is unaccompanied by any affection of the Schneiderian membrane. This, which for a long period of time was looked upon as altogether a distinct disease from Glanders, received, in English, the name of Farcy, and will in the following pages be described as one of the important forms or varieties of Glanders.

As Glanders is a disease which always originates in the horse and ass, never occurring in man except when communicated, directly or indirectly from them, and the nomenclature of the disease in man having been borrowed, to a great extent, from that previously in use among veterinary writers, its complete history, in the first place, necessitates an account of the mode in which it originates in the lower animals, and of the forms which it presents in them.

Glanders and its Varieties in the Horse, Ass, and Mule.—This disease appears to affect the horse in all parts of the world, although perhaps it is modified to a certain extent by climatic and other agencies. In the deserts of Arabia it is said not to possess the dreadful characters which distinguish it elsewhere, and is a comparatively rare disease. It may occur under four forms, as, 1st, Chronic Glanders; 2d, Acute Glanders; 3d, Chronic Farcy; 4th, Acute Farcy.

Chronic Glanders is the most common form affecting the horse. It is propagated by contagion and infection (?). It never occurs as a termination of acute Glanders. Its period of incubation is uncertain, and has been stated to vary from a few days to a year (?).

Synonyms.—Morve chronique, French; Chronischer Rotz, German; Ciamorro cronico, Morva cronica, Italian.

Semeiology.—The general health is little, if at all, affected. There is swelling and hardening of the submaxillary lymphatic glands. A discharge occurs from one or both nostrils, generally from one only (usually the left), which is at first of watery consistence, becoming more gluey, purulent, sanious, and fetid. Elevations and ulcerations occur on the Schneiderian mucous membrane. A horse thus affected often appears to be in perfect health. When placed under unfavorable circumstances, especially if fed scantily, symptoms of acute Glanders rapidly make their appearance, and death then soon inevitably follows.

Morbid Anatomy. — The mucous membrane lining the cavities of the nose and sinuses, presents small white elevations, varying in size from a small to a large pin's head, and larger patches of a yellowish-white color, having a smooth surface. These elevations and patches soften in the centre, and then present the appearance of excavated ulcers. The ulcerations sometimes, though not usually, implicate the whole thickness of the mucous membrane, and affect the bones; perforation of the septum narium, and of the nasal bones, sometimes occurring.

The mucous membrane of the larynx, trachea, and bronchi, presents at an early stage of the disease, little white elevations, resembling the cicatrices of leech bites; these afterwards coalesce, become injected, and ulcerate, giving rise to excavated ulcers, of a deep-red color.[1]

The lungs contain small fibrinous deposits, varying in size from a pin's head to that of a grain of flax, around which the pulmonary texture presents quite a healthy appearance. Larger masses, of a bluish-white, lardaceous, or gelatiniform appearance, which sometimes are of the size of a hen's egg, are also observed. A condition of lobular pneumonia has been described to exist in certain cases. Although veterinarians have spoken of these alterations in the lung as tubercular, there is no ground for the opinion, as the researches of Rayer,[2] Tardieu,[3] and Trousseau[4] have proved.

Acute Glanders occurs more rarely in the horse than the chronic form, of which, as of acute and chronic Farcy, it is a frequent termination. In the ass and mule it is the common form of the disease.

Synonyms. — Morve aigue, French; Acuter Rotz, German; Ciamorro acuto, Morva acuta, Italian.

Like the chronic, the acute form of Glanders is an intensely contagious disease. The period of incubation is uncertain. It has been stated to be only from three to five days,[5] although it is undoubtedly longer in certain cases.

Semeiology.—The disease sets in suddenly, a short time (a few days) after exposure to contagion, with symptoms of inflammatory fever. The respirations are hurried. A copious yellow, purulent, or sanious discharge flows from the nostrils. There is watering of the eyes. Sometimes an eruption of small cutaneous, or subcutaneous, tumors occurs. Then supervenes violent inflammation of the pitui-

---

[1] Tardieu, De la Morve et du Farcin chroniques chez l'Homme et les Solipèdes. Paris, 1843. P. 36.

[2] Rayer, De la Morve et du Farcin chez l'Homme. Mém. de l'Acad. R. de Méd. Tome sixième, pp. 828–833.

[3] Tardieu, op. cit. p. 41.

[4] Trousseau, Recherches Anat. et Pathol. faites à Montfaucon.

[5] Hering, Specielle Pathologie und Therapie für Thierärzte. Stuttgart, 1858. P. 98.

tary membrane, which becomes deeply and extremely ulcerated. Cough and shortness of breathing occur. Death invariably follows.

Morbid Anatomy. — The mucous membrane, lining the nares and frontal sinuses, is found acutely inflamed, and generally presents a pustular eruption, which is bathed in a purulent fluid. If the disease lasts for some time, ulcers form, which are either small and round, or large and irregular; in the latter case having been formed by the ulceration of several confluent pustules. The ulcers are excavated, often appearing as if cut with a punch. The lungs are almost constantly the seat of limited pneumonia, and frequently purulent deposits are formed in them. Petechiæ occur on the surface of the pleura, pericardium, and peritoneum. In certain cases (morve aiguë, hémorrhagique et gangréneuse, Rayer) petechiæ and large ecchymoses are noticed, after death, on the pituitary membrane. In these cases the mucous membrane is generally, over some part of its extent, destroyed, softened, and exhales a gangrenous odor.

Chronic Farcy is, like the other forms of Glanders, highly contagious. It may be produced by the inoculation of the altered secretions of farcied or glandered horses.

Synomyms.—Farcin chronique, French; Ch. Hautwurm, Wurm, German; Farcino, Italian.

Semeiology. — The disease usually commences by an indolent inflammation of the lymphatic vessels and glands, which become red, tender, and acquire a large size; the swellings occurring chiefly in the situation of the valves of the lymphatics (farcy-buds).

An eruption of small subcutaneous and cutaneous tumors occurs. The large glands and cutaneous swellings have a tendency to suppurate, and indolent ulcers result from the opening of the abscesses; these secrete an ichorous discharge, capable of producing Farcy, or Glanders, or both. The general health may continue good for a long period of time, the disease remaining stationary. If the animal be not destroyed, symptoms of general constitutional disturbance supervene. The animal loses flesh, has a staring coat, coughs, and usually falls a victim to acute or chronic Glanders.

Morbid Anatomy.—Structural alterations of lungs and other organs, as in Glanders. Induration and swelling of the lymphatics.

Acute Farcy is distinguished from chronic Farcy by the rapidity of its course, the urgency of the constitutional symptoms, and by its being almost invariably associated with acute or chronic Glanders. The chief anatomical difference between the two forms is the occurrence, in the acute, of truly cutaneous abscesses or boils. Acute Farcy proves invariably fatal.

Morbid Anatomy.—The same as that of Glanders and chronic Farcy.

Having described briefly the essential characters of the different varieties of Glanders, as they occur in the horse, ass, and mule, before proceeding to the consideration of the history of the disease as it affects man, it will be well to state shortly what appears to have been definitely made out, by veterinarians, as to the nature of the disease, the relations which exist between its various forms, and the mode in which it is transmitted. It has been satisfactorily proved—

1st. That the different forms of Glanders are due to, or are associated with, the formation of a specific virus, which exists in the blood and secretions of animals affected with it. The virus is readily absorbed by an excoriated or wounded surface, or when matters containing it are injected into the blood. It is likewise probably absorbed by the unbroken surface of mucous membranes.

In one or other of its forms the disease may almost certainly be induced by introducing beneath the skin of a healthy animal, the nasal mucus of a glandered horse, or the purulent or ichorous discharge which flows from the ulcerated swellings of Farcy. It may be induced by injecting into the circulation of a healthy animal, the blood of one suffering from Glanders.[1] The mucous membrane of the alimentary canal appears to be capable of absorbing the virus under certain circumstances.[2] There is reason to believe, however, either that gastric digestion is capable of destroying the virus, or that it may pass through the alimentary canal without injurious consequences resulting, provided the mucous surface be not abraded.[3]

2d. The virus of glanders and farcy is identical. The same pus may, if introduced into the system of one horse, produce acute glanders; into that of a second, farcy; into that of a third, chronic glanders.

On certain points the opinions of veterinary writers have been much divided. The chief subjects of dispute are, (1) the possibility of the disease originating spon-

[1] Travers, An Inquiry concerning that disturbed State of the Vital Functions usually denominated Constitutional Irritation. London, 1826. Vol. i. p. 355.

[2] Sainbel, Vial de St., Experiments and Observations on Glandered Horses, p. 109.

[3] Parent Duchâtelet, Hygiène Publique, tome xi. p. 194.

taneously; (2) the mode of its transmission; whether always by contagion, or by contagion and infection.

The intensely contagious nature of the disease is admitted at the present day by all writers; nearly all, however, admitting the possibility of the disease originating spontaneously. An impure and confined atmosphere, excessive work, and insufficient food, are the chief causes which have been alleged to induce it. That these circumstances favor its spread, cannot be doubted; that they act as most powerful predisposing causes, to this as to other diseases, has been unequivocally proved, by the ravages which Glanders has so often caused amongst the horses of armies subjected to famine and other injurious influences. French writers have all very strenuously maintained the possibility of the spontaneous origin of Glanders, a position which was strongly held by our countryman, Professor Coleman, who certainly has been one of the chief contributors to our knowledge of this disease. It was alleged, in an important discussion on this matter in the French Academy of Medicine, in 1861,[1] that at a period when the fortifications of Paris were being constructed, the horses of the poor and small contractors were found to suffer to a great extent from Glanders, whilst those belonging to the wealthier contractors escaped the disease. Those who adduced these facts attributed the occurrence of the disease, in the former case, to the insufficient nourishment and the harder work which the animals belonging to the poorer contractors received, whilst they appear to be susceptible of a very different and much more likely explanation. The laws which exist in France for preventing the use of glandered horses have been inefficiently carried into execution, glandered horses being by some people systematically employed; these horses are necessarily very much cheaper than sound ones, and would be more likely to be bought by the poor, than by the rich contractor. In our own country Glanders was, at one time, a most prevalent disease, which created the greatest ravages. Glandered horses were systematically sold and worked. The practice caused the greatest pecuniary loss to the country at large, for one case of Glanders having been introduced into a stable, the chances were considerable that a majority of the horses in it would sooner or later die of the disease. To remedy this state of matters a most stringent law was passed, which prevented the working of glandered horses. Carried into execution with great rigor, this law has had the effect of rendering Glanders, in all its forms, a very rare disease in this country. Thousands of horses are exposed to those causes which are supposed to give rise to Glanders; are hard worked, ill-fed, and kept in badly-ventilated stables, in the same way as horses used to be a century ago, but yet Glanders does not originate. Glanders has now almost ceased to affect the horses of our army, and the sporadic cases which very rarely occur need cause no astonishment when we remember that the disease is one which still prevails to a certain extent, especially in Ireland, whence most of our cavalry horses are obtained, and that the disease may have a long period of incubation. The history of Glanders amongst the lower animals in this country leads us to form the opinion that it never originates spontaneously in our climate. We must fully admit, however, as we have already done, that bad feeding, bad ventilation, and excessive work are powerful predisposing causes of this disease, and to a great extent explain the ravages which it has often caused amongst the horses of armies, which in time of war, from the large and indiscriminate purchases of horses that must necessarily be made, contain abundantly the germs of the disease.

Whether actual contact of some article, containing the virus, with a mucous membrane, or a broken cutaneous surface, be required to induce the disease, or whether its germs may be communicated through the atmosphere—whether, in short, Glanders is always communicated by contagion, using the term in its more limited sense, or by contagion and infection, has been warmly argued by various writers. Probably in nearly every case actual contact of glandered matter with an absorbing surface does take place. The mucus flowing from the nose of the glandered horse becomes attached to the stable and the stable utensils, and comes almost necessarily in contact with the water, hay, and straw, which horses in the same stable employ, so that they are frequently exposed to conditions *positively* known to be capable of inducing the disease.

Some cases have, however, been recorded which cannot well be explained on the hypothesis of actual contact, and we therefore do not deny the *possibility* of Glanders being transmitted from one lower animal to another through the medium of the atmosphere, although we believe this to occur, if at all, only very rarely.

History of the Disease in Man.—No connection had been traced between the terrible diseases in the lower animals which have been briefly described, and an affection which then as now must have occasionally affected those who had charge of horses suffering from Glanders and Farcy, until the year 1810 when Waldin-

[1] Recueil de Médecine Vétér., Août 1861, p. 645.

ger[1] drew attention to the fact that special precautions ought to be adopted in the dissection of horses affected with Glanders and Farcy, inasmuch as the direst consequences, even death, might result from the inoculation of the purulent matter. The accuracy of the statements of Wadlinger was supported by the publication, in 1812, of a paper by a French military surgeon, Lorin, who, under the title "Observations sur la Communication du Farcin aux Hommes," described the case of a veterinary surgeon who, having accidentally pricked himself whilst operating upon a glandered horse, suffered in consequence from inflammation of the hand.[2] The statements of Waldinger do not, however, entitle him to be considered the first person who pointed out that Glanders is communicable from the lower animals to man; for although he stated that dangerous consequences might result from the inoculation of the purulent matter of Glanders and Farcy, he did not state that the affection induced in man in any way resembled that of the horse. Again, the observation of Lorin was of the most unsatisfactory description, for the case which he describes does not differ materially from many cases of dissection wounds, and possessed, in no respects, the peculiarities of Farcy. Schilling, however, published in 1821,[3] under the title "Merkwürdige Krankheit und Sections Geschichte einer wahrscheinlich durch uebertragung eines thierischen Giftes erzeugten Brandrose," the case of a man who, having washed out the nares of a glandered horse, became affected with a pustular eruption on the skin, an offensive discharge from the nostrils and erysipelatous inflammation of the face, and who died after an illness of eight days. Rust looked upon this as a case of Glanders in man, and in support of this view gave the notes of another case of a similar nature. Both these were indeed most typical cases, and must be considered to be the first well-marked cases of Glanders occurring in man which were published. In the same year that Schilling's and Rusts cases were published, there appeared in the *Edinburgh Medical Journal*[4] a short notice copied from a subscription paper in the Hotel Coffee-house, Leeds. This short notice (of nine lines) was signed by T. Muscroft, surgeon, Pontefract, and consisted in an appeal on behalf of the family of the dog-feeder of the hounds belonging to the Badsworth Hunt, who, in cutting up the carcase of a horse which had died of Glanders, had accidentally wounded his hand. "In a few days he betrayed all the symptoms which are at first shown in the horses beginning in the above disease. He gradually became worse, and at the end of the week he died raving mad, laboring under a confirmed complaint of Glanders."

Feeling considerably interested in reading this notice, I wrote to Henry Muscroft, Esq., surgeon, of Pontefract, asking if the notice which had appeared in the Edinburgh Journal in 1821 had been written by a relative of his, and requesting to be put in possession of any information he might possess on the matter. Mr. Muscroft informed me in reply that the notice had been signed by his father, who at that time was practising in Pontefract, and added that he had never heard anything about the case. Stimulated by my questions, Mr. Muscroft, however, instituted inquiries, which ended in his discovery of the widow of the unfortunate man. The old woman is now eighty-six years of age; she was, however, able to give a very complete account of her husband's illness. This account was written down verbatim by Mr. Muscroft, who has sent it to me. Information obtained from other sources proves that the dates mentioned in this statement are perfectly correct. Mr. Muscroft's pay-book, on being referred to, shows that he attended John Turpin from March 18, 1821, to March 27; and the parish register of deaths contains an entry to the effect that John Turpin died on the 29th March. The statement of the old woman is interesting, as referring to the first case of Glanders, which appears to have been recognized as such, in this country.

Statement of Sarah Hazelgrave (formerly Turpin), aged eighty-six years.

My first husband, John Turpin, whilst skinning a dead horse, in the month of March, 1821, cut the third finger of the left hand, and, heeding but little this accident, on the following day he left me for a week, to fetch home to the Badsworth's kennels some young hounds that had been reared in the neighborhood of York.

On his arrival at home, he was very weak and scarcely able to walk, and he at once said that "he should never go out again, he was so ill." He complained of severe pain in the head, and there was profuse discharge from the nostrils; and on different parts of his body there were a number of blisters of different sizes, which, after a time, became blue.

Mr. Muscroft was sent for, and after he had been at my husband's bedside for some time, he said, he was "afraid Turpin was inoculated by the blood of the

[1] Waldinger, Wahrnehmungen in Pferden. 2te Aufl. Wien, 1810.

[2] Journ. de Méd. Chir. et Pharm., Fév. 1812.

[3] Rust's Magazine, vol. i. p. 480.

[4] Edin. Med. and Surg. Journ., vol. xviii. p. 321.

dead horse he had cut up, and that there was no remedy, the disease being horse-farcy, or glanders, but that, if the finger had been taken off at first, this might not have happened."

Mr. Muscroft gave him medicines, and saw him daily until his death, which took place ten days after he returned from York. He retained consciousness to the last. Large lumps appeared on his forehead and face, and his throat was swelled. His head and face became very large. The wound of his finger was very bad, and the finger looked as if it would rot off, and from the wound there were hard cords, like the stem of a pipe, up the arm, and the armpit also swelled. The smell was very bad.

May 18, 1865.

Before the publication of the cases of Schilling and Rust, and before the notice referred to appeared in the *Edinburgh Medical Journal*, certain cases of transmission of Glanders from horses to man had been observed in London, which, although not published until some years later, received at the time a thorough investigation—a study which first and satisfactorily proved that the virus of Glanders is transmissible from horses to man, and generates in him affections which, although slightly different in some of their characters, are identical with the disease as it is observed in the horse. These observations were first published by Mr. Travers, at page 350 of the first volume of his work on "constitutional irritation."[1] The first and most interesting of these cases occurred in the year 1817, and was that of Mr. William Turner, a veterinary student, who injured his finger in examining the head of a horse which had died of Glanders. An ulcer followed with inflammation of the absorbents and cellular tissue of the hand, and symptomatic fever. After some days an abscess formed in the opposite arm, and another in the lower part of the back. Matter taken from the abscess of the arm was sent to Mr. Coleman, who inoculated an ass with it and produced fatal Glanders. Mr. Turner's health was seriously affected. He became hectic, and was sent to Brighton for the benefit of sailing and tepid bathing. Subsequently an abscess formed in his lungs, another in his kidney, and after his return to his residence at Croydon, at the expiration of several months, abscesses formed successively upon each knee-joint. From the last, matter was taken for the purpose of inoculating an ass, which in eleven days died glandered. Shortly after this, Mr. Turner's long and painful illness terminated in death. The third case, recorded by Mr. Travers, that of Nimrod Lambert, is however of greatest importance. N. L., a healthy hackney coachman, æt. 32, in January 1822, infected a chap on the inside of the right thumb, by inserting it into the nostril of a glandered horse, to pull off a scab. He was taken suddenly ill three days after, and suffered subsequently from inflammation of the lymphatic vessels and glands of the arm, which suppurated and ulcerated. "The glands at either angle of the lower jaw, and in the groin, became swollen, and he was much afflicted with pain between the eyes and down the nose, and ulcerations of the membrana narium, attended with discharge." After a long and painful illness the man recovered, although his constitution appears to have been violently affected and permanently injured by it. An ass, inoculated with the matter from this man's sores by Mr. Sewell, died of Glanders. The great interest attaching to Lambert's case rests in the fact that it is the first *well-marked* case of Glanders occurring in man in which the diagnosis was rendered perfectly certain by the inoculation of animals with the purulent matter. Mr. Turner's case, on the other hand, was by no means a well-marked instance of chronic Farcy, but is valuable as being the first case which proved that in its passage through the human body, the virus of Glanders loses none of its properties, and is again able to generate the disease in lower animals. With these cases Mr. Travers published a letter from Professor Coleman, in which that gentleman described the experiments which he had made with the pus obtained from Mr. Turner's abscess. In this letter Mr. Coleman stated the result of a most interesting experiment, in which he succeeded in inducing Glanders, "by removing the healthy blood from an ass, until the animal was nearly exhausted, and then transferring from a glandered horse blood from the carotid artery into the jugular vein." (Op. cit. page 355.) Although Mr. Travers published these cases, he does not appear to have considered the disease induced in Mr. Turner and Lambert, to have been *glanders*. "The reader," says Mr. Travers, "cannot fail to perceive some points of analogy between the effects produced by the absorption of glandered matter, and of the fluids of the human body after death. I think there can be no difficulty in admitting in either of these cases that a poison was imbibed, but the evidence gives no ground for the conclusion that it was a morbid poison, as regards its operation in the human subject." (Op. cit. p. 365.)

[1] An Inquiry concerning that disordered State of the Vital Functions, usually denominated Constitutional Irritation, by Benj. Travers. London, 1826.

Grubb,[1] Krieg,[2] and Brown[3] described after this several interesting cases of Glanders transmitted from the horse to man. It was, however, by the publication of a memoir entitled "On the Glanders in the Human Subject,"[4] that Dr. Elliotson attracted the attention of the profession to this subject. In this paper he gave the history of two interesting cases of Acute Glanders, which had been observed by Dr. Roots and himself, and of a third which had been communicated to him by Mr. Parrott, of Clapham, and he collected nearly all the observations which had, up to that date, been placed on record. In a second memoir,[5] entitled "Additional Facts respecting Glanders in the Human Subject," Dr. Elliotson gave an account of another case of acute Glanders which had fallen under his notice. Subsequently to the date of Dr. Elliotson's papers, several cases of Glanders were published in various journals. No very valuable addition to the knowledge of the subject was however made until the year 1837, when Rayer, in a splendid memoir, entitled "De la Morve et du Farcin chez l'Homme,"[6] gave an account of all the cases of Glanders which had been observed up to that date, and gave an original and complete description of the different forms of Glanders both in the horse and in man. The subject of Chronic Glanders and Farcy was afterwards made the subject of special investigation by Tardieu, who, in a memoir entitled "De la Morve et du Farcin chroniques,"[7] supplemented the knowledge which Rayer had already collected on these subjects. It is to the researches of these distinguished physicians that we owe almost the whole of our knowledge of the morbid anatomy of the various forms of Glanders.

Since the date of the publication of these memoirs, many cases of Glanders possessed of great interest have been recorded: no very important addition to the existing knowledge of the disease has, however, been made.

Etiology of the Disease in Man.—This section of our subject has been already so fully treated of in discussing the subject of Glanders affecting the horse, that it here needs but a very brief consideration.

Always communicated from the horse, the ass, or the mule to man, those are specially predisposed to contract the disease whose avocations lead them to come in contact with these animals. The great majority of the cases recorded have occurred amongst veterinary surgeons, veterinary students, grooms, coachmen, cavalry soldiers, horse-slaughterers, &c. Writers on Glanders in man have admitted that, although the disease is often or generally due to inoculation of morbid matters from glandered horses, it may be occasionally communicated to man by infection. It has been stated, in support of this view, that a frequent cause of Glanders and Farcy is the custom of causing men to sleep in stables. Those who hold the above view seem to forget that these men, besides sleeping in the stables, are, from the very nature of their duties, exposed to the greatest danger of contracting, *by inoculation*, Glanders from any affected horse which may be placed under their charge. If facts are very scanty to support the belief that Glanders is propagated from horse to horse by infection, they assuredly are still more so in the case of man. In the immense majority of cases of Glanders in man which have occurred, there is the most conclusive evidence that the sufferers were in constant habit of *handling* glandered horses, whilst in a considerable number of cases there has been actual evidence of the disease having been transmitted by inoculation. To explain, as due to infection (using the term in its more limited sense), the cases which have occurred in which no history of inoculation through a wound is to be obtained, appears to the writer altogether unjustifiable, when regard is had to the facts that the virus of Glanders, besides being capable of being absorbed by the most trifling cutaneous abrasion, is probably absorbed by unbroken mucous membrane, and that of the very large number of persons who formerly used to be brought in contact with glandered horses, only an excessively small fraction contracted the disease, although a large number of these people were most careless and imprudent in their treatment of glandered horses. It may, we think, be safely stated that Glanders is only communicable from the lower animals to man by inoculation.

The virus of Glanders may adhere to stable utensils, clothes, and other articles, for a long period of time, and then when brought in contact with an absorbing surface may give rise to the disease.

Not only can the disease be transmitted from the horse, ass, or mule to man, but likewise from one human being to another. Several facts have placed this beyond dispute. A distinguished young French veterinarian, M. Gerard, died of Acute Glanders induced by a wound which he

[1] Diss. sistens casum singularem morbi contagio mallei humidi in hominem translato orti. Berolin, 1829.

[2] De Typho malloide. Berolin, 1829.

[3] London Medical Gazette, vol. iv. p. 134.

[4] Med.-Chir. Trans., vol. xvi., 1830.

[5] Ibid., vol. xviii., 1833.

[6] Mém. de l'Académie de Médecine, vol. vi. p. 625.

[7] Paris, 1843.

inflicted on himself whilst making the post-mortem examination of a veterinary student who had died of Glanders.[1] Elliotson,[2] in describing a case of Glanders, mentions that the laundress who washed the clothes of the patient, contracted Glanders. An externe of a Paris hospital died from Acute Glanders contracted from a stableman suffering from Glanders.[3] In this case, besides dressing the wounds of the patient, the externe had taken an active part in the examination of the body after death.

Although usually contact of the virus of Glanders with an absorbing surface gives rise to the disease, this does not invariably follow.

It has been erroneously stated that a weak state of the constitution, intemperate and irregular habits, predispose individuals to attacks of Glanders. Whatever influence they may have on its progress and issue, there is absolutely no proof that they exert any influence in the way mentioned. It has, indeed, been correctly remarked, that a large majority of the recorded cases of Glanders have occurred in men of robust constitutions, and in the prime of life.

SEMEIOLOGY.—We shall consider separately the semeiology of the four varieties of Glanders which are observed in man as in the horse.

Acute Glanders occurs in man more frequently than the other forms of the disease, and presents in him characters resembling those of cases in the horse in which Acute Glanders and Farcy are combined. The period of incubation of Acute Glanders varies probably from twenty-four hours to a fortnight; its limits, in the majority of cases, being from three to eight days.

The mode of invasion of the disease varies considerably. In those cases where a distinct wound exists, through which the virus has been introduced, redness, pain, and tension, accompanied by an erysipelatous appearance around the wound, and swelling of the lymphatics, are often noticed before the supervention of any constitutional symptoms. Occasionally no wound can be discovered, or the wound through which the virus was introduced may remain in a passive condition. Whether local symptoms have been developed or not, the disease is usually ushered in by feelings of lassitude, headache, and rigors, frequency of the pulse, and often by vomiting and diarrhœa. Articular and muscular pains occur from an early period of the disease, and increase during its progress. The limbs and body become the seat of subcutaneous abscesses, which are specially found on the face, and near the articulations. Over the abscesses the skin becomes of a red and violet color, and sometimes limited gangrene sets in. The pus which forms in the abscesses is serous and fetid. A remarkable pustular eruption generally appears on the surface of the body, being specially found on the cheeks, arms, and thighs. The pustules commence as little red spots, which afterwards present the appearance of white papules, and become full of pus; they are often surrounded by a red areola. The pustules vary greatly in size, presenting a flattened or pointed surface, and either occur singly or so closely aggregated as to be almost confluent. The pustules are often accompanied by bullæ of a dark color, on the face, trunk, and organs of generation. An erysipelatous inflammation of the nose, eyes, and neighboring parts of the face, sometimes extending to the scalp, is far from infrequent. The cutaneous eruption, which has been described, has been compared to that of varicella, variola, ecthyma, and yaws, but is considered by all the best writers on Glanders to be quite distinct and characteristic. (Rayer.) Amongst the most constant and pathognomonic of all the symptoms of Glanders, is however one which has been noticed in the large majority of the recorded cases. A yellow, viscous, purulent discharge, often mixed with blood, and not infrequently possessed of great fetor, exudes from the nares. This proceeds from the Schneiderian membrane, which is *invariably* the seat of a pustular eruption, or of ulcerations.

The mucous membrane of the mouth is sometimes likewise the seat of ulcerations or pustules, and from it, in certain cases, a purulent fluid has been observed to exude. The submaxillary lymphatic glands are usually not enlarged.

The prostration, which has been stated to usher in the disease, increases during its progress. The pulse becomes excessively frequent, small, and compressible. The voice is feeble, and the breath fetid. The constipation, which frequently exists at the commencement of the disease, is replaced by diarrhœa; the stools are extremely fetid. A hard, dry cough, accompanied by a little expectoration, and often by difficulty of breathing, testify to the existence in man, as in the horse, of pulmonary complications. With the increasing prostration, delirium sets in, which is followed by coma, and ultimately by death. The duration of the disease has varied from three to fifty-nine days. As a rule, death has occurred about the end of the second or commencement of

[1] Breschet, Revue Méd., tome ii. p. 96, 1826.
[2] Elliotson, Lancet, 1838.
[3] Tardieu, op. cit. pp. 140, 141; and Bérard, Bull. de l'Acad. de Méd., Nov. 1841, tome vii. p. 182.

the third week. Acute Glanders runs an unusually rapid course when it occurs as a sequela of the other and more chronic forms of the disease.

Chronic Glanders is the rarest form of the disease in man. When it occurs, it is usually as a sequela of Farcy. Uncomplicated by Farcy, the disease has but seldom been observed. When Chronic Glanders occurs primarily (*i. e.* not as a sequela of Farcy), it commences with lassitude and very severe articular pains, which readily pass away, and are followed by a cough, sore throat, and a disagreeable feeling in the nose.

When Farcy has existed before the appearance of Chronic Glanders, no lassitude or pains may be noticed. In these cases, the first symptoms are the sore throat, cough, and affection of the nose, which have been alluded to. A pain is then felt in the trachea, the voice becomes altered in character, or true aphonia may occur. The cough is accompanied by dyspnœa, and often by expectoration. Capillary bronchitis and pneumonia sometimes supervene. The disagreeable sensations in the nose increase—the patient complaining of a feeling of its being stopped up; sometimes, although by no means always, there is a dull aching pain at the root of the nose. On blowing the nose, a puriform mucus, mixed with little bloodclots, becomes detached, or there may be a regular discharge (*jetage*) from the nose. On examining the nares at this stage, ulcerations may not infrequently be perceived on the Schneiderian membrane. By the introduction of a probe, inequalities of the surface, and even perforation, of the septum narium, may be detected.

Ulcerations may sometimes be likewise observed in the pharynx. There is very rarely any induration of the submaxillary glands.

The skin is free from eruption.

In addition to the special phenomena which have been described, the patient is usually prostrated, suffers from articular and muscular pains, diarrhœa, and nausea, loses flesh, and falls into a state of marasmus. He may die of simple exhaustion, or Acute Glanders may set in and rapidly close the scene. This form of Glanders has been once observed to terminate in recovery (case of Nimrod Lambert). Its duration is excessively various; those cases which are complicated with Farcy proving more rapidly fatal than others. As a general rule, the course of the disease extends over several months, and cases have been recorded where it appears to have lasted for some years.

Acute Farcy presents, almost constantly, all the characters which have been described as those of Acute Glanders, with the single exception that there is a total absence of affection of, or discharge from, the nares. So great is the resemblance between the two affections, that only certain special points in connection with Acute Farcy need be alluded to. A subdivision of all cases of Acute Farcy may be made, into cases accompanied by a cutaneous eruption, and cases unaccompanied by eruption. In some cases of Acute Farcy, there is merely an inflammation of the lymphatic vessels and glands, accompanied with the formation of soft subcutaneous tumors in various parts of the body. In other, and by far the greater number of cases, an eruption, exactly similar to that of Acute Glanders, occurs, such cases being distinguished from instances of the latter disease only by the absence of affection of the nose. These cases are very much more serious than those without eruption, and follow exactly the same course as Acute Glanders, proving fatal usually between the thirteenth and nineteenth day. (Rayer, op. cit. p. 787.) In the cases where there is no eruption, and which have received the special name of cases of acute farcinous angeioleucitis, the disease very frequently terminates in recovery, or passes into Chronic Farcy.

Chronic Farcy may exist alone, or associated with Chronic Glanders. It is ushered in by lassitude, wandering pains, uneasiness, anorexia, accompanied by fever, which often assumes a tertain type. An abscess usually forms on the forehead, the calves of the legs, or some other part of the body, which is followed by other indolent and fluctuating tumors. Some of these open spontaneously, and give exit to blood, or ill-conditioned pus. The lymphatic glands only secondarily, and to a slight extent, become affected. With the successive appearance of abscesses, the general health and strength of the patient decline; he loses flesh; the skin becomes dry, and earthy; the countenance yellow and livid. Frequent rigors occur. The open abscesses become converted into horrible ulcers, which have no tendency to heal. A dry cough harasses the patient, who, with his body covered with foul ulcers, falls into a condition of most miserable marasmus. The course of the disease is tedious and uncertain. The abscesses commence to form from the third to the fifteenth day of the disease. The subsequent stage is, however, most protracted and uncertain in its issue. It may terminate by an attack of Acute Glanders, or of Chronic Glanders, or the patient may die of exhaustion, or from an attack of pyæmia. Lastly, recovery may occur. The duration of the recorded cases of Chronic Farcy has varied from four months to three years; it usually termi-

nates in from ten to fifteen months. Of twenty-two cases of the disease, cited by Tardieu, six recovered.[1]

Diagnosis.—Having described the characters of the different varieties of Glanders, there remains for consideration their differential diagnosis.

Acute Glanders is distinguished by such remarkable characters, which are always associated together, that when fully developed it would be impossible to confound it with any known disease. In its early stage, when the articular and muscular pains are very urgent, it might be mistaken for acute rheumatism. The occupation of the patient, the existence of a wound in a state of irritation, the much greater degree of prostration than is common in acute rheumatism, the absence (usually) of redness and swelling around the painful joints, would arouse the suspicions of the physician. At a later stage, the association of the peculiar and characteristic eruption of pustules and bullæ, with ulceration of, and discharge from the nose, taken in connection with the history of the affection, distinguish in the clearest manner Acute Glanders, from erysipelas of the face, typhoid fever, malignant pustule, pyæmia, and some other diseases, to which it has been supposed to present points of resemblance.

Chronic Glanders offers much greater difficulties in diagnosis than the acute form of the disease. When uncomplicated by Farcy, it might be readily mistaken at first sight for one of the different forms of ozæna. The occupation of the patient, the mode of invasion of the affection, the absence of other evidences of strumous or syphilitic affections, would furnish the data for forming a diagnosis. Cases of uncomplicated Chronic Glanders are, however, as has been already stated, of great variety. In the cases where Chronic Glanders and Farcy are combined, the diagnosis is more simple, for an association of symptoms then exists which is not presented by any other disease. Cases of Chronic Glanders must, however, occasionally of necessity occur, in which the physician must experience great difficulty in forming a correct diagnosis. In these cases an ass, or a horse, might be inoculated with some of the nasal mucus, if the case were one of uncomplicated Chronic Glanders; or with pus from an abscess, if the affection were Chronic Glanders and Farcy.

Acute Farcy, when occurring in a characteristic form, could scarcely be mistaken for any other disease. The history of the case, the great rapidity of its course, and the extraordinary pustular eruption, together with the numerous subcutaneous abscesses and tumors, would distinguish the case from cases where multiple abscesses occur from the introduction of other septic matters into the system. In cases of Acute Farcy without eruption, and of uncomplicated Chronic Farcy, only an accurate acquaintance with the early history of the case, and an accurate study of its progress, could enable the physician to arrive at a correct opinion. The supervention of Acute or Chronic Glanders would, in a large proportion of such cases, remove the difficulties which they at first present.

Morbid Anatomy.—Having described, in the preceding pages, the morbid anatomy of the various forms of Glanders in the horse, there remains little to be said of the morbid anatomy of the disease as it affects man, for the structural alterations of the nasal fossæ, of the larynx, trachea, and lungs, are as characteristic of the disease in man as they are of it in the horse. The chief point of difference in the morbid anatomy appears to be that, in all the different forms of Glanders seen in the horse, the lymphatic system is very much more involved than in man.

1. *Acute Glanders.*—In all cases where the nasal fossæ have been examined, they have been found the seat of disease. Sometimes the Schneiderian membrane is ecchymosed and gangrenous, whilst often it presents numerous little pustules of the size of millet seeds. The frontal sinuses are frequently found filled with puriform mucus. The larynx has been the seat of eruption and ulceration in several cases. The lungs frequently present patches of pneumonia, or purulent formations are formed in them.

2. In *Chronic Glanders*, the affection of the nose differs considerably in character. At an early stage, the mucous membrane is often found injected and much swollen. Small submucous abscesses form. These open and become ulcerated; the ulceration spreads to the bones and cartilages, and necrosis generally follows. These ulcerations generally occur on the septum, which, in almost every case of Chronic Glanders becomes ultimately perforated. Ulcerations of the larynx, trachea, and bronchi are very commonly found. The epiglottis is sometimes ulcerated, but the vocal cords are usually unaffected. The ulcerations of the trachea are often very extensive, and affect the whole thickness of the mucous membrane. They exhibit a remarkable tendency to heal spontaneously. The lungs are, by no means so constantly as in Acute Glanders, the seat of the disease. The pleura is often covered with small yellow elevations, which, on being incised, are found full of pus.

[1] See Tardieu, op. cit. p. 75, from whom the above description of Chronic Farcy has been abridged.

Beneath the pleura, deposits of a purely fibrinous nature are found. Occasionally a portion of lung substance is indurated, in consequence of fibrinous exudation; in the centre of the indurated portion, pus is formed. The lymphatic glands are often somewhat enlarged, reddish, and of soft consistency. The bronchial glands have, in cases where ulcerations of the air-passages existed, been found enlarged, softened, and in state of suppuration. The alimentary canal, the liver, and spleen present no peculiar appearance.

The morbid anatomy of Acute Farcy is identical with that of Acute Glanders, except that the Schneiderian membrane is found perfectly healthy.

In Chronic Farcy, the morbid appearances resemble those of Chronic Glanders. There is, however, no affection of the nose.

Prognosis.—In all the forms of Glanders, the prognosis is of the most unfavorable description. Acute Glanders and Acute Farcy, when accompanied by the characteristic eruption, are almost necessarily fatal. One case of Acute Glanders, and several of Acute Farcy, have been recorded, in which recovery took place. In Farcy, unaccompanied by eruption, the prognosis is much more favorable, recovery being the rule, and not the exception. Chronic Glanders, especially when complicated by Farcy, is almost invariably fatal. The case of Lambert, which has been referred to, proves, however, that even under these desperate circumstances recovery may take place.

Chronic Farcy, although a most dangerous disease, usually terminates in recovery.

In forming a prognosis, in apparently slight cases of Chronic Glanders or Farcy, it must not be forgotten that Acute Glanders often suddenly supervenes, in the course of these affections, and then invariably proves fatal.

Therapeutics.—This section of the subject need unfortunately be treated of very briefly, for no treatment which has been adopted, in the cases which have hitherto occurred, has appeared to exercise the slightest influence in checking or modifying the progress of the disease.

Characterized as it is by symptoms of the greatest constitutional prostration, a stimulant and supporting plan of treatment appears to be indicated, and has been recommended by all writers on Glanders.

All attempts which have been made to cure Glanders in the horse have proved futile. There is considerable evidence, however, that arsenic, especially when administered with nux vomica or strychnia, has in some cases of Chronic Glanders excited a remarkable influence in checking the progress of the local affections; and we therefore think a trial of these remedies might with propriety be made, in cases of Chronic Glanders occurring in man.

HYDROPHOBIA.

By John Gamgee, and Arthur Gamgee, M.D.

Definition.—A disease due to a specific animal poison which resides in the saliva of animals affected with it. It never originates spontaneously, and is communicated directly or indirectly, and usually by biting, from carnivorous or omnivorous animals, and especially dogs, to man. It is alone recognized by its physiological effects on man or animals. It induces pain and stiffness in the inoculated part, exalted sensitiveness and irritability, feverishness, mental anxiety, flitting pains, spasm of the throat on the sight of liquids, tremors, headache and delirium, vomiting, eructations and tympanites, great prostration, and death. Its period of incubation varies from a few days to many months.

Synonyms.—Λύσσα, κυνόλυσσα, ὑδροφοβία; Phobodypson, Pheugydron, Rabies, Rabies canina, Rabies contagiosa, Entasia Lyssa, Canine Madness; La Rage, Hydrophobie (French); Die Hundswuth, Wasserscheu, Tollwuth (German); Rabbia, Idrofobia (Italian); Hidrophobia (Spanish); Watervrees (Dutch).

History.—Few diseases have, at different periods of the history of medicine, excited greater attention, curiosity, and study than Hydrophobia, and few have

been described in a more graphic or more accurate manner by ancient writers. Celsus,[1] Dioscorides,[2] Cælius Aurelianus,[3] and Galen, have left us accounts of the disease which equal those of the best writers who have followed them.

It has been argued and indeed generally admitted, that the disease was not unknown to Homer, and the word λύσσα, which is so often employed in the Iliad to describe a wild and reckless fury, is supposed primarily to have been used to designate the madness of dogs.

This view has been held since the days of Cælius Aurelianus, and is supported by the best authorities.[4] Thus Dr. Bardsley, in a very learned memoir on Hydrophobia,[5] says :—

"We have already said that the disease was well known to Homer, and applied by him, with his usual critical exactness of similitude, to the indiscriminate havoc with which Hector sweeps through the battle-field of his enemies;" and after quoting several passages[6] in which the word λύσσα or one of its derivatives is used, he adds : "The poet with much propriety puts these words into the mouth of Ajax his enemy, for dog was already a term of reproach among the Greeks as well as the Jews." The passages alluded to do not appear to us to prove satisfactorily that their author was acquainted with Hydrophobia. The word λύσσα, and its derivatives, is used by Homer on all occasions, as it is by many other Greek writers,[7] to denote martial rage ; and in spite of the occurrence of the expression κύνα λυσσητῆρα, we feel inclined to believe that this was the primary meaning of the word, which was only secondarily applied to designate the disease of the dog, in the same manner as madness, rage (Fr.) and rabbia have in modern European languages been secondarily used in a limited and specific sense to denote Hydrophobia. Excepting that the word was subsequently used to designate the madness of dogs, there is no evidence that it had that meaning at the period when the Homeric poems were written ; whilst in using the expression 'κύνα λυσσητῆρα,' already referred to, the author of the Iliad much more probably referred to dogs infuriated with anger than to dogs suffering from Hydrophobia. All who are practically acquainted with the disease will admit that whilst on the one hand it is most improbable that the snappish ill-temper of rabid dogs should have been referred to, as a point of comparison, by Homer in the passages quoted by authors, there is on the other hand nothing more natural than that he should compare an angry man to a furious dog, inasmuch as there is nothing more suggestive of ungovernable passion than the frenzy of an infuriated (but not *rabid*) dog.

Whatever may have been the primary meaning of the word, there can be no doubt that, in course of time, the word λύσσα and its very numerous derivatives —λυσσάω, λυσσαίνω, λυσσηδόν, λυσσήεις, λυσσητήρ, λυσσώδης, λυσσόδηκτος, λυσσητικός—were employed to express Hydrophobia, and in this sense the word λύσσα is employed by Plutarch and Dioscorides.

Hippocrates only alluded in the faintest manner, if at all, to Hydrophobia, and we agree with Dr. Bardsley in the opinion that when speaking of the "φρενέτικοι βραχυπόται,"[1] the father of medicine probably only means to treat of a "variety of phrenitis or mania."

Democritus, however, who was the contemporary of Hippocrates, if we are to believe the statements of Cælius Aurelianus,[2] was acquainted with the disease, and wrote upon its nature and treatment. "Etenim Democritus qui Hippocrati convixit, non solum hanc memoravit passionem, sed etiam ejus causam tradidit, cum de opisthotonicis scriberet." In two other passages in his interesting account of Hydrophobia, Cælius alludes to the opinions of Democritus as to the affection being one of the nervous system, and mentions the remedies which that author recommended for its treatment.

Aristotle was undoubtedly acquainted with the Hydrophobia of dogs. In his "Historia Animalium,"[3] besides describing it, he mentions that the disease is communicable to all animals but man. The opinion, although erroneous, points to the fact of his having directed attention not only to the characters but also to the mode of propagation of the disease. Both Asclepiades and Themison, according to Cælius Aurelianus and Dioscorides, were acquainted with Hydrophobia, and entertained peculiar views as to its nature. Themison is indeed said to have been himself subject to an attack of Hydrophobia, but as he recovered from the

[1] A. C. Celsi Med. lib. v. cap. xxvii.

[2] P. Dioscoridis Op. omnia. Vienna edition, 1598. Σημεῖα κυνὸς λυσσῶντος, καὶ τῶν ὑπ' αὐτοῦ δεδηγμένων. κεφ. α.

[3] C. Aurelianus, De Morbis acutis et chronicis, lib. iii. cap. ix.–xvi.

[4] See Thesaurus Grecæ Linguæ ab. H. Stephano construct. vol. iv. fasc. 2 ; also Damm. Nov. Lex. Græcum etymol. et real. ; cui pro basi substratæ sunt concordantiæ et elucidationes Homericæ et Pindaricæ. This author derives λύσσα from λύκος,, wolf, this animal being very often subject to Hydrophobia.

[5] Cyclop. of Pract. Med., vol. ii. art. Hydrophobia.

[6] Iliad, lib. viii. 299 ; ix. 237 ; xiii. 53.

[7] Plato, Herodotus, Sophocles.

[1] Prædict., lib. i. p. 69. Prænot. Lect. ii. p. 131.

[2] Op. cit., lib. iii. cap. xv.

[3] Hist. Animalium, lib. viii. c. 22.

affection we may with probability conjecture that its nature was very different from that of true Hydrophobia.

Of the ancient writers, Celsus and Dioscorides, Cælius Aurelianus, Galen, and Avicenna, are those to whom we are chiefly indebted for our knowledge of Hydrophobia, and to their opinions we shall, on more than one occasion, revert. Since the period when they wrote, all systematic writers on medicine have treated of Hydrophobia. Foremost amongst those who have contributed to our knowledge on the subject are Van Swieten, Sauvages, Cullen, Fothergill, Mead, Trollicts, and Bardsley.

The recent history of Hydrophobia refers principally to Europe, not only because greater attention has been paid to this as to every other disease of men and animals in the Old World, but because the malady has been to a very great extent confined to Europe. We shall afterwards attempt an explanation of this fact, but in the mean time we may state that the oldest report of scientific interest that we can trace, refers to wolves afflicted with rabies, in Fraconia, in 1271. Contrary to their usual habits, these animals spared the herds and flocks, and attacked shepherds in the fields, or followed human beings into the towns and villages. Upwards of thirty men fell victims to these attacks.[1] Canine madness prevailed considerably in Spain in 1500.[2] In 1590, we are told by Bauhin,[3] rabies raged in an epizootic form amongst the wolves in the province of Monthelliard. In 1604 it prevailed in Paris, and although it was doubtless common there, and after in various parts of Europe, we do not hear of it again until 1691,[4] when the heat of summer proved insufferable, the crops withered for want of rain, animals died in great numbers in Italy, and dogs went mad. This reference to the dependence of rabies on the hot season must simply be regarded as pointing to a coincidence. Severe outbreaks of rabies occurred in France and Germany from 1719 to 1721, in Hungary in 1722 and 1723, and atmospheric influences were blamed for similar attacks in various parts of the continent, from 1725 to 1726.[5] We learn, however, that wolves were affected as well as dogs at this period, and special mention is made of wild animals being seized with the disease in Silesia and Lusatia (now Bautzen, in Saxony). The malady was rife in London in 1760, in Philadelphia in 1779, in the West Indian Islands in 1783,[1] and in various parts of Europe from 1785 to 1789.[2] At this period special notice is again taken of rabid wolves which communicated the disease and terrified the people far more than even rabid dogs.

It was not till the commencement of the present century that foxes were discovered to suffer much and frequently from canine madness. In 1803 these animals were seen rabid in large numbers in the Pays de Vaud, in various parts of the Aubonne, Cossenay, Orbe, and Yserden districts at the foot of the Jura, and in 1804 similar outbreaks were witnessed on the northern shore of the Lake of Constance, and thence extending throughout the whole of Germany. Blaine tells us that in 1806 rabies in the dog became so prevalent in the vicinity of London that a day seldom passed without his being consulted on one or more cases of it, and sometimes he would see three, four, or five a day for weeks together. The kingdom of Wirtemberg and Grand Duchy of Baden were sadly ravaged by it in 1808 and 1809. Foxes continued to be affected, and these usually shy animals faced or followed men, cattle, or dogs in open daylight, bit them, and communicated the disease. The Wirtemberg Government ordered a report to be published on this subject in 1829, which contains particulars of these outbreaks.

From 1803 to 1820 is a memorable period in the history of Hydrophobia. In 1810 it spread rapidly through Southern Russia, and the same year it appeared in America, in the State of Ohio, where it destroyed dogs, wolves, and foxes, besides other domestic animals, and no small number of human beings. In 1815 the malady was raging in Denmark. Blaine tells us that in 1820 it was again on the increase in England, and for three or four years "continued alarmingly common" but moderated again for a few seasons. During the years 1819 and 1829, rabies was rife in Italy, and Brera speaks of a wolf which bit thirteen persons, of whom nine died hydrophobic. Wirth specially notices the period from 1819 to 1826 as remarkable for rabies amongst the foxes of Switzerland and Germany,[3] and the foxes communicated the malady to dogs, cats, horned cattle, horses, pigs, goats, and sheep. In 1824 rabies prevailed ex-

[1] Addit. ad Lambert, Schafnaburg.
[2] Blaine's Canine Pathology.
[3] J. Bauhin, Memorab. Historia Luporum, 1591.
[4] Ramazzini, pp. 157–186, and Baglivi (1828), vol. ii. p. 331.
[5] Breslau, Sammlungen, xxxiii. p. 90.

[1] Moseley's Treatise on Tropical Diseases.
[2] Schnurrer, ii. p. 389; and Fehr, Ueber die Hundswuth, Münster, 1739.
[3] Franque, Die Seuche unter den Füchsen und die ursprüngliche Wuth Krankheit der Hunde; Frankfort, 1827. Köchlin, Ueber die in unsern Zeiten herrschende Krankheit unter den Füchsen; Zürich, 1835. Wirth, Lehrbuch der Seuchen und ansteckenden Krankheiten der Hausthiere; Zurich, 1846.

tensively amongst foxes, wolves, cats, and reindeer in Sweden, Norway, and even Russia. In the Rhine Provinces, various cantons of Switzerland, and in the kingdom of Wirtemberg, many men were bitten by mad foxes in the year 1827, but owing to the prompt measures usually adopted the individuals escaped; dogs and cats thus bitten, however, became rabid.

In 1830 the subject of rabies attracted great attention in England, and evidence on the subject was laid before a select committee of the House of Commons. Sir Benjamin C. Brodie, Professor Coleman, Messrs. William Youatt, Morgan of Guy's Hospital, Earle of St. Bartholomew's, Benjamin Travers, and others were examined. Some idea of the state of popular terror, if not of the number of bites by rabid dogs, may be gleaned from Mr. Youatt's statement in evidence, to the effect that he had applied lunar caustic to about 400 bitten people, and a surgeon at St. George's declared that within a limited time he had similarly operated on 4,000 without an accident. Mr. Youatt particularly referred to a great increase in the prevalence of the disease as contrasted with eighteen months previously. Mr. Henry Earle, of St. Bartholomew's, furnished unmistakable evidence of the increased frequency of the disease in England, and stated that he had seen twenty-five cases of Hydrophobia in man in twenty-five years, whereas his father had only seen one in fifty years, before his time. It is certain from the statements of Blaine, Coleman, and others, that prior to 1830 foxhounds were affected in this country to an extent never witnessed before nor since.

That there has been a decided abatement in the frequency of the malady in England during the second quarter of the present century, as compared with the first, cannot be doubted, and it is believed that the law abolishing the use of dogs as beasts of burthen has operated greatly in favor of the change. That law not only led to a diminution in the number of dogs owned by a class of persons who could not keep these animals in a way calculated to prevent the spread of contagious diseases amongst them, but indirectly it led to the destruction of a lot of roving curs, kept by the poorer classes. The dog tax, too, which might have been beneficially enforced to a greater extent than it had been, no doubt tended to limit the number of valueless animals kept. Whatever may be the cause, it is certain that there is far less canine rabies, hence much less human Hydrophobia, in the British isles than on the Continent, and during recent severe outbreaks of this disease abroad little has been heard or seen of it amongst us.

Some remarkable statements have been published regarding Hydrophobia in France and the German States during the thirty-five years of comparative immunity which we have enjoyed. In 1830, when attention was attracted by the prevalence of the malady in London, there was occasion for alarm in Vienna, where thirty-nine cases of rabies in the dog were reported in rapid succession. Few instances of the disease were seen there for the succeeding seven years, but in 1838, 117 cases occurred; in 1839, sixty-three; in 1840, 317; and in 1841, no less than 141. Of the last number only thirty-one animals were affected with the dumb rabies. Only fifteen of the 141 were bitches. Two cases occurred in animals under one year, and only one in dogs between seven and fourteen years of age. The disease was specially rife in the months of February and May, in each of which twenty-one cases were observed; in January there were sixteen, seventeen in April, and eighteen in June. The fewest attacks were in the months of September, November, and December. From 1839 to 1842, rabies in the dog was very prevalent in Wirtemburg, and this was attributed by Professor Remy to communication from foxes. From the 1st of January, 1840, to the last of February, 1842, no less than 230 cases of rabies occurred in dogs, and twenty-one in bitches in the small Grand Duchy of Baden. In 1840-41-42, the malady was very destructive in Lyons and other parts of France. In the space of thirty years, no less than 779 dogs have died of rabies in the Lyons Veterinary School, giving a yearly average of nearly twenty-six cases. The largest number of these cases occurred in the months of June and of April. Only so late as 1864, the city of Lyons was in great commotion owing to the terrible frequency of rabies canina.

Some idea may be formed of the great difference between Great Britain and the Continent in relation to the frequency of this disease, from the fact that having been more or less connected with veterinary colleges in this country since 1849, not a single case of rabies has come under our own observation here, whereas many instances have been seen by us abroad, on almost every occasion that we have visited foreign schools. When in Lyons and Paris in 1854, many were the cases we saw, and often did we see ladies unconsciously carrying their rabid pets to be visited by the professors.

CAUSES.—A glance at the history of Hydrophobia suffices to prove that, so far as man is concerned, the specific disease is due to only one cause, contagion. The poison is a fixed one, and flows from the sick animal's body with the abundant

saliva which is secreted. It must penetrate the system to take effect, and is usually deposited by the teeth of rabid animals in and beneath the skin. It is not volatile, and the disease is therefore not infectious. Many circumstances affect its transmission, and on this we shall say more, after having spoken of the malady as it attacks the lower animals.

It is generally supposed that rabies originates spontaneously in the dog and other carnivorous animals. So deeply-rooted is this belief, that even men of eminence have attempted various explanations of the supposed fact. One of the oldest views refers canine rabies to the heat of the "dog-days" and the tortures of thirst. Another, recently revived, is that the malady is developed in dogs kept under restraint, and from indulging at proper seasons in sexual intercourse. These and other theories are so absurd, and so opposed to many of the facts which we must incidentally allude to in these pages, that we may state dogmatically they have no foundation in truth. Without entering into a discussion on matters of theory, we consider it more profitable to refer to the peculiar features of rabies in those of the lower animals which undoubtedly are capable of inoculating human beings with the rabid virus. We shall, therefore, speak of the disease as it occurs in the dog, wolf, fox, badger, marten, cat, horse, or sheep, pig, and goat.

Dog.—One of the facts of primary importance, in the history of canine rabies, is that it is far more prevalent in dogs than bitches. Professor Coleman stated in evidence in 1830, that on the occasion of rabies entering fox-kennels, the mad dogs bit dogs, but spared the bitches. We well know that, as a rule, rabid animals retain a certain affection for animals and people they know and care for. A dog will not bite its master at first, but rather avoid the presence of all he likes. There is some method in their madness, and we have no better proof of this than in the way in which bitches are spared by the rabid dog. Leblanc has published statistics, confirming the extraordinary prevalence of the disease in the males of the canine species, but he tried to bend the facts to demonstrate his theory that the disease is connected in its development with the repression of the desire for sexual intercourse. There are no experiments, no reliable facts indicating any ground for the belief that canine rabies originates in anything else than contagion, even in the dog; and the best British authorities, such as Blaine and Youatt, long since advocated this, the undoubtedly correct view of the subject.

It is singular that, even in relation to so active and certain a virus as that of rabies. we find instances of remarkable constitutional resistance to its effects. Thus when I visited the Lyons Veterinary College, in 1853, a pointer dog was in the infirmary which had been bitten no less than seventeen times by dogs affected with rabies, with which he had been purposely confined by Professor Ray. Other cases of a like kind have been recorded, but they may be considered rare and exceptional.

The period of incubation of the disease in the dog has been set down as between three and seven weeks. Blaine has met with a case in which rabid symptoms succeeded a bite in a week, though Youatt never saw a case with less than seventeen intervening days. In 1862 M. Renault made a communication to the Academy of Sciences at Paris, and stated that during twenty-four years he had made numerous experiments with a view to ascertain the period of incubation of Hydrophobia in the dog. During that period, 131 dogs have, under conditions which he describes, been either bitten by mad dogs, or inoculated with the foam as immediately collected from the mad animals. Of this number, 63 having presented no signs of disease during the four subsequent months, were not further observed. Of the 68 others, the Hydrophobia was developed at various periods, as shown in the following table :—

In 1 dog	between	the 5th	and	10th	days.
4 dogs	"	10th	"	15th	"
6 "	"	15th	"	20th	"
5 "	"	20th	"	25th	"
9 "	"	25th	"	30th	"
10 "	"	30th	"	35th	"
2 "	"	35th	"	40th	"
8 "	"	40th	"	45th	"
7 "	"	45th	"	50th	"
2 "	"	50th	"	55th	"
2 "	"	55th	"	60th	"
4 "	"	60th	"	65th	"
1 dog	"	65th	"	70th	"
2 dogs	"	70th	"	80th	"

There are no premonitory signs of an attack of the disease in the dog. When the period of incubation is passed, the animal is restless, dull, watchful, and snaps at dogs, other animals, or men, which come in its way. It shuns the light, but with much slyness seeks an opportunity of escape, and roves about town or country, manifesting extraordinary powers of exertion, and marked insensibility to blows and ill-usage. The habits of an animal may not change completely at first, and the recognition of persons it has been daily in contact with is sometimes very remarkable. The dilated pupils, the manner in which the eyes follow any object moved before them, and the peculiar modification in the bark, which is more of the nature of a howl, are amongst the most characteristic symp-

toms. The appetite is lost, thirst often considerable, and the animals usually drink without difficulty. The coat is staring, skin tight on the ribs, abdomen tucked up, head depressed, and nose protruded, with a dirty mouth and tongue, and sometimes a discharge of mucus and saliva from the sides of the mouth. In a certain number of cases, the nervous symptoms are very prominent at this period, and the lower jaw drops from paralysis of the muscles connected with it. The howl is then lost, hence the name dumb rabies for the cases associated with this symptom. Emaciation, craving after filth, which is swallowed with some difficulty, constipation, and a scanty discharge of high-colored urine, are amongst the noticeable symptoms. Mad dogs have been known to eat portions of dead dogs, and mad bitches to devour their young. There is a singular absence of any marked acceleration of the pulse and breathing during the disease; the animal sinks, often paralyzed in the hind quarters, and dies somewhat tranquilly from the fourth to the eighth day.

Schrader says that, of 267 cases of rabies, 223 belonged to the barking or raging variety, and 44 to the dumb or paralytic form.

[There is good evidence to show that Hydrophobia may be produced by the bite of a healthy dog, much *enraged;* as in fighting with another animal.—H.]

Foxes show signs of the disease, as stated above, by losing all their shyness, following animals and men, biting them, losing their consciousness, and becoming paralytic, and otherwise presenting most of the characters of the disease in the dog.

Wolves are more to be feared than foxes, from their greater strength and ferocity. They attack human beings without fear, and usually bite them about the face, neck, and hands. As they approach death, they skulk away, and die in retired spots, blind, powerless, and in the quiet stupor seen in typhus.

Cats have scarcely less propensity to attack the uncovered parts of the bodies of individuals, and especially the face, than foxes. They scratch, whine in a hoarse manner, and die from the third to the fourth day.

Martens and *badgers* present most of the symptoms seen in cats and foxes.

[The *skunk*, in the United State, is liable to rabies, and has several times been known to communicate it, by its bite, to human beings.—H.]

In *the pig* there is a peculiar restlessness, squealing grunt, disposition to bite, gaping, salivation, marasmus, paralysis of the hind quarters, and death from the fourth to the fifth day.

Rabies canina has been often observed in herbivorous quadrupeds, horses, oxen, sheep, deer, and other animals. The symptoms are marked and unmistakable, presenting all the characters of the signs of rabies in the dog. From the imperfect powers of biting, communication of the disease from vegetable feeders to man is rare, and we need not, therefore, enter into further particulars on the subject here.

Hydrophobia in Man.—As actual contact of the saliva of a rabid animal with a wounded or abraded surface is required for the development of Hydrophobia, we should expect that, of the total number of persons bitten, only a certain proportion should fall victims to the disease, and this is really found to be the case. Whether some individuals are, from idiosyncrasy, less prone to contract Hydrophobia than others, is not known, and it does not appear that age, sex, or constitution materially modifies the transmission of the disease. The situation and character of the wound affect, in all probability, the result, for it would appear that where the injuries are situated on the uncovered portions of the body, as on the hands or face, there is greater danger of the supervention of Hydrophobia than when they have been inflicted on the covered portion of the body or limbs. The clothing, in the latter case, protects the wound from the action of the saliva. The bites of wolves appear to have been more frequently followed by Hydrophobia than those of dogs; probably owing to the fact that, from their natural ferocity, the former animals, when attacking men, very commonly inflict severe injuries on the face and neck. Such circumstances as prevent the adoption of an energetic prophylactic treatment of those bitten by rabid animals, obviously favor the development of the malady.

The actual ratio of cases of Hydrophobia to the total number of persons bitten by rabid animals, has been very variously estimated by different writers on this subject. Whilst some have stated that only 5 per cent. of all persons bitten by rabid dogs become affected with Hydrophobia, others have estimated that 55 per cent. of the bites inflicted by rabid dogs are effectual in transmitting the disease. The ratio varies very greatly; probably between the limits we have mentioned. In the case of the bites inflicted by rabid wolves, the ratio of cases of Hydrophobia would appear to be as high as 60 per cent.

Symptoms of Hydrophobia in Man.—As it affects man, Hydrophobia presents symptoms which differ from those which have been described as characteristic of the affection in the dog. The chief point of difference is the almost con-

stant occurrence in man of the peculiar spasms of the pharynx,[1] excited when the patient makes an effort to drink, and which gives rise to the dread of fluids, to which the disease owes its name. The cerebral symptoms appear to be less constant in man than in the dog; for—whereas amongst the most prominent of the symptoms in the dog is the thorough change in the natural instincts of the animal, and an uncontrollable tendency to attack and bite all around him—it would appear that in man delirium, although a frequent, is by no means a constant or necessary accompaniment of the affection.

Period of Incubation.—We are probably acquainted with no disease which possesses so long a period of incubation as Hydrophobia, or in which the period of incubation varies to so great an extent. This fact was well known to the earlier writers on Hydrophobia, one of which most accurately remarks that whereas the affection usually supervenes within forty days after the infliction of a bite by a rabid animal, some are only affected after a year or more has elapsed.[2]

The disease has been very rarely developed sooner than eight days after the bite of a rabid animal, and rarely after a longer period than two or three months. The large majority of cases has occurred within four or eight weeks. Many authentic cases have occurred in which the period of incubation extended to eight or nine months, and a few where it appeared to be much longer. A remarkable case has been placed on record, in which a man who had been in prison for a period of more than two years, became affected with Hydrophobia, although he had only been bitten by a mad dog seven years previously.

During the period of incubation there is nothing which specially distinguishes the bite of a rabid from one inflicted by a healthy animal. The wound may heal rapidly, or it may continue in an irritable condition, no peculiar train of symptoms occurring which can enable us to form a prognosis as to the probable issue of the case. The period of latency, or *delitescence*, as it has been termed, having elapsed, the following train of symptoms usually occurs. The wound becomes intensely painful, the pain often shooting from the extremities (if it be situated upon them), towards the trunk. If the wound have cicatrized, the cicatrix becomes red and irritable, or if, as is generally the case when the injuries inflicted by the rabid animal have been severe, it have not healed, it assumes an unhealthy appearance. With this local irritation, there is sometimes, though very rarely, some affection of the lymphatics of the part. In addition to the local phenomena, others of a constitutional character soon set in. The patient becomes peevish, irritable, and depressed; the skin is hot, the pulse rapid, and the appetite is lost. The peculiar depression of spirits just alluded to has been thought to be very characteristic of this, the first stage of Hydrophobia, and is indeed rarely absent. The unfortunate patient, who has been dreading the consequences of an accident of which the gravity is well known to him, often deludes himself with the hope, from the absence of all symptoms for a time, that all fear of danger has passed, and only awakens to a feeling of the real horror of his position when the local phenomena, which have been described, supervene. It is not strange, then, that they should be accompanied by a feeling of melancholy and impending evil.

The first stage of Hydrophobia has been termed that of "recrudescence." Its characters are not fixed and invariable, for it has been noticed, in some cases, that no local symptoms occurred before the development of the true hydrophobic phenomena, and in others that melancholy and irritability were absent. In these cases, a feeling of general "malaise" and pyrectic phenomena have alone been present. This stage of recrudescence, of irritation and melancholy, as it has been variously styled, is usually of very short duration. After a few hours, or at most a day or two, the patient begins to complain of stiffness about the head and neck; and then the most characteristic manifestation of the disease sets in—an affection of the pharyngeal muscles, which throws them into the most painful spasms when the unfortunate sufferer attempts to eat, but specially to drink. The patient suffers from the most agonizing thirst, whilst the knowledge of the spasms which will accompany any attempts at drinking cause him to dread even the sight of liquids. "Miserrimum genus morbi; in quo simul æger et siti et aquæ metu cruciatur."[1] The hot and parched mouth becomes full of a clammy and viscid saliva which the poor sufferer is continually attempting to spit out, giving rise to the sound which the older writers compared to the barking of a dog. The heat of skin, and the frequency of the pulse, become excessive; the countenance is intensely anxious; the eye is bright and restless, and shuns the sight of a bright or luminous object, which often causes a fit of the painful spasms which, at first, attempts at deglutition only pro-

[1] [The respiratory muscles are usually still more affected.—H.]

[2] Cæl. Aurel., lib. iii. cap. ix.

[1] Celsi Med., lib. v. cap. xxvii.

duced, but which afterwards the mere sight or even thought of fluids brings on. Delirium may be, and often is, absent; when present, it is often violent, and almost maniacal. In some cases a fit of general convulsions supervenes; in others, the spasm becomes weaker, and the patient, sinking into a state of comparative quiet, may regain, after a period of agitation and delirium, composure and quiet. Death may supervene early; it is often preceded by a stage of profound coma. A certain paralysis of the lower jaw, which drops, and allows the saliva to flow from the angles of the mouth, often marks the later stages of some cases of this sad disease, from which probably no one ever recovered.

The hydrophobic stage, which has just been described, is of short duration, death usually occurring before the end of the second day, life very rarely having been prolonged beyond the fourth day.

Diagnosis.—In its first stage, when the phenomena of *recrudescence*, as they have been termed, are first manifesting themselves, it is impossible to form an accurate diagnosis. The feverish symptoms, and the fresh accession of pain in the bitten part, are symptoms which may, and often do occur in persons who have been bitten by animals not in a rabid condition—bites being very liable to heal slowly, to become the seat of neuralgic pains, and to give rise to considerable constitutional disturbance.

When Hydrophobia is fully developed, a careful study of *all* the phenomena cannot fail to lead to a correct diagnosis. It has apparently been confounded with diseases whose only resemblance to it consisted in the occasional occurrence of pharyngeal spasms, as inflammation of the larynx, trachea, and œsophagus. Hydrophobia may, however, more readily be confounded with hysteria, acute mania, and tetanus. A perusal of many of the recorded cases, especially of those which are said to have terminated in recovery, will readily convince the inquirer, that acute mania has sometimes been mistaken for Hydrophobia. In certain cases of this disease, it would appear that a dread of fluids has been manifested, whilst the general appearance of the patient has singularly resembled that of patients affected with Hydrophobia. The absence of a history of a bite, the history of premonitory symptoms of mania having existed, the duration of the disease, and the rapidity of the pulse—which in acute mania is very rarely above 90, whilst in Hydrophobia it is usually much higher—are the data upon which a differential diagnosis should be based.

French writers have described a definite disease under the terms "hydrophobie non rabique," "hydrophobie rabiforme," "rage spontanée," which is said to resemble in the closest manner true Hydrophobia.

This affection, it is alleged, has generally been caused by fear, which has supervened after the bite of a rabid dog, or after communication with persons suffering from the disease. Its symptoms are, it is said, occasionally quite undistinguishable from those of true Hydrophobia; there is in the spurious form, however, very commonly a desire to bite. The patient may die in a few hours or days, or may remain affected for weeks or years. The diagnosis, it is stated, rests chiefly on the non-discovery of a part which has been bitten. We cannot see the propriety of admitting the existence of a non-rabid Hydrophobia, for the cases which have been described as examples of it may be more accurately classified as cases of either hysteria, acute mania, or true Hydrophobia, which had not been recognized.

Tetanus presents certain points of resemblance to Hydrophobia, tetanic spasms sometimes affecting the pharyngeal as well as the other muscles. The chief points of difference are, that Hydrophobia supervenes on a bite, whilst tetanus usually follows some other injuries; that the latter disease supervenes sooner after the injury than does Hydrophobia; that in tetanus the spasms are usually not intermitting; that trismus usually exists; that it is not marked by the distressing thirst, or the abundant discharge of saliva, which is so common in Hydrophobia; and that delirium very rarely occurs during its course.[1]

[The excess of excito-motor susceptibility is most extreme in Hydrophobia.—H.]

Morbid Anatomy.—There are no cadaveric lesions which can be said in any way to characterize Hydrophobia. The most constantly observed phenomena have been the following:—

Redness and swelling of the fauces and gullet, occasionally accompanied by enlargement of the salivary glands, and sometimes with redness of the stomach. The trachea and bronchi have often been found injected, and filled with frothy mucus. The lungs are congested, occasionally inflamed, sometimes empty and œdematous. (Trolliet.)

The brain has been found occasionally congested; sometimes there has been effusion into the arachnoid and lateral ventricles. The medulla oblongata, and the origins of the seventh, eighth, and ninth nerves have been noticed to be congested, thickened, and softened.

[1] Holmes' Surgery, vol. i. p. 313, art. "Tetanus," by Mr. Poland.

Prognosis.—When the disease is fully established, the prognosis is necessarily of the worst description, death being inevitable. Under this section a question of greater practical importance may be discussed, and the solution of which depends upon the facts which have already been referred to. A patient having been bitten by a rabid dog, when can the physician give a tolerably favorable opinion as to the issue of the case? when can he express a pretty confident opinion as to the safety of his patient? As the large majority of all those who are bitten by mad dogs escape Hydrophobia, even when no treatment is adopted, a favorable and encouraging, though a guarded, opinion can and should be given, even immediately after the accident. As the greater number of cases occur between the thirtieth and fortieth days, when the latter period is safely passed the prognosis becomes more and more favorable. After the end of the second month the large majority of patients may be considered safe.

Therapeutics.—Since Hydrophobia was first described, all authors have devoted considerable attention to the treatment of the disease, both prophylactic and curative. It has long been known that the evil effects of the bite of the mad dog are probably often prevented by the adoption of an active local treatment of the bitten part. Celsus accordingly recommended the application of the cupping glass to the bitten part, or even the employment of the actual cautery. Since his time, in addition to the supposed specific methods of prophylactic treatment, nearly all authors have recognized the use of local treatment. Excision of the bitten part, the application of the actual cautery, or a combination of excision and cauterization; the application of various caustics, amputation of a bitten limb above the seat of the injury, have all been suggested and employed by different surgeons, and there appears to be good reason for believing that good effects have followed all these various methods.

Complete and early excision of a bitten surface, as soon as possible after the infliction of the injury, is the method which has found most general favor in this country, and is perhaps the best which can be adopted. When, from the situation of the wound, excision is inadmissible, recourse should be had to the free use of caustics, and of these we should prefer the most powerful at our disposal—such as, presumably, would decompose such an animal virus as that of Hydrophobia; potassa fusa, or strong nitric acid, should have the preference. It must be mentioned, however, that, in the opinion of some, recourse need not be had to such severe methods. The late Mr. Youatt, who in the course of a long experience had treated a very large number of persons who had been bitten by dogs undoubtedly rabid, placed the greatest reliance upon cauterization with solid nitrate of silver, which in his experience had in every case prevented the development of Hydrophobia.

Besides the local modes of treatment, the older physicians believed in the efficiency of other remedies. Prolonged and sudden immersion in cold water was from the remotest times considered of great value in warding off a fit of Hydrophobia, and was practised to within a comparatively recent period. Celsus speaks of the plan with much favor, and Desault, in his well-known treatise on rabies, describes the mode in which the operation was performed in his day. It is in the memory of persons still living, that those bitten by mad dogs were, in England, taken to the sea-coast and dipped in the sea.

From the fact that so few of those bitten by rabid animals actually contract Hydrophobia, we should expect that a large number of specifics would have been proposed for its treatment. A credulous physician who happened to have administered some remedy to a few persons bitten by a mad dog, finding that no evil consequences followed, and forgetful that had nothing been administered his patients would, in all probability, have enjoyed equal immunity, was only too ready to believe that he had at last discovered a specific for a terrible disease. Thus, doubtless, it was that the Ormskirk medicine, a compound of chalk, alum, Armenian bole, elecampane root, and oil of anise; the Tonquin medicine, a mixture of musk and cinnabar; and the Tanjore pills, a combination of arsenic and mercury, acquired for a time a wide and fictitious reputation.

The use of the hot-air and vapor bath has lately been recommended as a prophylactic in Hydrophobia, and it has been stated even to have cured the disease when fully developed. In the absence of any well-authenticated cases, we cannot decide upon the value of the remedy. That it might help to eliminate a virus which lurks so long in the system before producing its specific effects, is, however, not impossible.

[The hypodermic injection of *curara*, and also that of chloral, are among the later experimental remedies employed.—H.]

Amongst the most important questions for discussion, in considering the treatment of Hydrophobia, is the following: In the event of the peculiar phenomena of recrudescence manifesting themselves after a very long interval in a person who has been bitten by a rabid animal, should any local treatment be adopted? Should

the bitten part or the cicatrix which has formed be excised or amputated? This question cannot at present be answered positively. As, however, cases have undoubtedly occurred in which such a local treatment of a bitten part has, even after the always-to-be-dreaded symptoms of recrudescence had set in, appeared to prevent the development of Hydrophobia, we are of opinion that the local treatment, severe though it may be, should be tried.

In the treatment of Hydrophobia, when fully developed, we must rely entirely upon general principles of medicine in attempting to relieve the sufferings of the patient. No specific method of treatment has been shown to have the slightest influence in checking or modifying this disease, from which, in all probability, no one ever recovered. We should try to soothe and comfort the unfortunate patient in every way in our power, to attempt to allay the great nervous excitement by means of sedatives, to support the failing strength by stimulants, and chiefly to prevent all noises, draughts, and other sources of excitement, which are so liable to bring on the painful pharyngeal spasms. Desault suggested, and apparently with good reason, that large fluid enemata might with advantage be administered. By checking the agonizing thirst they would, in all probability, greatly lessen the sufferings of the patient.

[While the production of mere *euthanasia* will not justify the unlimited employment of powerful narcotics, it would seem that the use of chloroform by inhalation is made suitable by its effect in quieting the respiratory spasms; and the system is found, in hydrophobia, to have a remarkable tolerance of chloroform. A child eight years old, under my care, in the height of the attack, inhaled several drachms of it, with the effect of producing tranquillity only, without deep narcotism. —H.]

ENTERIC OR TYPHOID FEVER.

By John Harley, M.D. Lond., F.L.S.

Definitive Description.—A continued febrile condition of uncertain duration, accompanied by marked intestinal derangement, and invariably associated with lesion of the solitary and agminated glands of the intestines. It commences in anorexia, with nausea or vomiting; its progress is marked by profuse diarrhœa of light ochre-colored watery stools, associated with abdominal pain, tenderness, and tympanitic swelling; and, if the issue be unfavorable, it terminates in exhaustion, intestinal hemorrhage, or perforation of the bowel. Death usually occurs in the fourth week. In the early period, the disease is attended by more or less pyrexia; as soon as it is fully developed, there is well-marked hectic fever. During the height of the disease a scattered papular rash appears in successive crops on the abdomen and chest. The rapidity with which the symptoms are manifested, and the degree to which they are developed, vary greatly in different cases. The intestinal disease is frequently obscured by the concurrence of pulmonary or cerebral complications.

Synonyms.—*German*—Abdominal Typhus, Darm-typhus, Typhus gangliaris vel entericus, Ileo-typhus. *French*—Fièvre Typhoïde, Entérite septicémique, Fièvre muqueuse, Fièvre entéro-mesentérique, Gastro-entérite, Dothiénentérite, Entérite-folliculeuse. *English*—Typhoid Fever, Autumnal or Fall Fever, Slow Nervous Fever, Common Continued Fever, Hectic Fever, Infantile Hectic Fever, Infantile Remittent Fever, Entero-mesenteric Fever, Gastric Fever, Enteric Fever, Intestinal Fever, Pythogenic Fever, Cesspool Fever. *Common*—Bilious Fever, Gastro-bilious Fever, Muco-enteritis.

The above are the principal synonyms in use. Many others could be given; but as they may be either recognized as modifications of the foregoing, or have no restricted application to the particular disease under consideration, they need not be mentioned here. All may be found in Dr. Murchison's valuable work on the "Continued Fevers of Great Britain," p. 385, *et seq.*

The appellations "Typhoid," "Abdominal Typhus," and the like, lead to an association of two diseases in the mind, which does not exist in reality; such terms therefore lead to confusion. "Gastric" has reference to an organ which, at most, only functionally sympa-

thizes with the principal lesion; the term "Pythogenic," introduced by Dr. Murchison, to imply the putrid source of the disease, is, on the one hand, too general, since it may be argued, with equal reason, that other acute diseases, besides the one under consideration, arise from this cause; and, on the other hand, it is not sufficiently comprehensive, since it would appear that Enteric Fever may arise from other causes than putrid or sewer emanations.

In adopting a term to distinguish the disease, we should select one which at once marks it out from all others, and points to a constant feature. Such a term is "Entero-mesenteric," employed by MM. Petit and Serres, in 1813. This appellation is a brief definition of the disease, and but for its inconvenient length we would employ it here. Acknowledging the direct sympathy which the mesenteric glands have with the intestinal lesion, we prefer, however, to use the shorter term, "Enteric Fever."

Preliminary Observations. — No disease presents, in the mode of the accession of its characteristic symptoms, in the gravity and sequence of these, and in its whole course and ending, so many variations, irregularities, complications, and accidents as Enteric Fever. A complete and consequent history of the disease will be best obtained by considering: 1st, The symptoms attending its development and progress, the condition of the secretions, and the modes of termination, the accidents, and sequences of the disease; 2dly, The morbid anatomy; 3dly, The pathology; 4thly, The associated pathology, meaning thereby a comparative view of the development of the particular intestinal lesion in other acute diseases—an investigation of great importance in the comprehension of the relation of acute diseases generally, but one of especial value in the elucidation of the nature of Enteric Fever; 5thly, The varieties; 6thly, The distribution; 7thly, The causes; and subsequently, there will remain to be considered the Diagnosis, Prognosis, Therapeutics, and Prophylactics of the disease.

Clinical History of the Disease.

Development and Progress.—In considering the clinical history of Enteric Fever, it will be convenient, with reference to the mode of access and development of the symptoms, to group the cases into three classes, viz. (1) Those in which the symptoms of gastro-intestinal irritation remain latent for days, or even weeks, after the patient has declined in health; (2) Those in which gastro-intestinal derangement is the chief feature of the disease from its outset to its termination; and (3) those which, in the suddenness of the invasion, the severity of the symptoms, and in the rapid course of the disease, closely resemble cases of narcotico-acrid poisoning.

Many of the cases of Enteric Fever belong to the *first class*. The disease indeed usually commences insidiously, and without premonitory indications of intestinal disorder. The decline of his health has been so slowly progressive and uniform, that the patient cannot state precisely when his illness commenced. For days or weeks past he has lost appetite, and felt weak, languid, and disinclined for bodily or mental occupation, complaining of a little headache, chilly sensations, chiefly referred to the spine, and of weariness and pains in the limbs. His increasing weakness sooner or later compels him to relinquish his ordinary occupations, and to apply for relief. We find the tongue moist, and tolerably clean; the skin cool, pallid, and free from rash; the pulse is rather small, and slightly accelerated; the mind is clear, and the expression natural; the bowels have responded to a purgative, but now they are regular, or perhaps again constipated; the abdomen is natural; the other functions of the body are regularly performed. The patient may remain in this condition for some time, but sooner or later the nature of the disease is manifested by the appearance of its characteristic symptoms. At first, there is increase of the early symptoms, anorexia is aggravated to nausea, and sometimes there is vomiting of green fluid; the skin becomes hot and dry; the pulse is increased in frequency; [bleeding at the nose is common; bronchial cough is rarely absent;—H.] the tongue is furred, and usually presents red prominent papillæ at the margins and tip; there are great restlessness and increased headache; the bowels become loose, and the abdomen is a little full, painful, and tender—the right iliac fossa especially so, and pressure upon this part usually produces gurgling. A few round rose-colored papules may now be observed upon the abdomen, chest, or back. They disappear on pressure, and closely resemble the papules of variola during the first few hours of their existence; but they are not quite so large, nor so hard. Their number varies much, and the quantity of rash bears no proportion to the severity of the disease. Usually we do not find more than three or four papules; occasionally the chest and abdomen are closely spotted with them. In one case we observed them profusely scattered over the thighs, legs, and feet. During the prevalence of the diarrhœa a few fresh spots appear every day, and after forty-eight hours the old ones begin to

fade away. Diarrhœa, frequently associated with bilious vomiting, now prevails, and the abdomen becomes distended, and in many cases more or less tympanitic; the alvine dejections are watery, and of a light ochre color, and putrid odor. At first they are acid, but they soon undergo change, and become ammoniacal and have an alkaline reaction.

With the supervention of diarrhœa, all the symptoms become greatly aggravated; the pulse ranges between 120 and 130; the skin is often pungently hot, and occasionally attains, toward night, a temperature of 107 or 108°. During this stage there is great irritability, and often considerable delirium, especially at night. In some cases there is no delirium and the mind remains clear to the last. Symptoms of active pulmonary congestion,—accelerated breathing, pain in the chest, mucous râles, and expectoration streaked with blood,—are also liable to arise. The aspect of the patient is usually indicative of suffering, but the countenance is clear, and the eyes bright, as in scarlatina; the cheeks are suffused with a hectic flush. The urine is clear and copious; it is frequently retained.

The patient may continue in this condition for several days, the body meantime undergoing rapid emaciation. The tongue may continue moist, in which case it becomes pale, large, and flabby, and is liable to ulceration about its margins, and the formation of deep fissures with everted margins across the dorsum. In many cases the tongue becomes dry, red, contracted, and fissured at this period. The mucous membranes become dry and inflamed; the gums are liable to bleed, epistaxis frequently appears, and sordes begin to form upon the dry teeth. Here is the turning point of the disease. If we can subdue the gastric irritation, and keep food in the stomach, and restrain the diarrhœa, the symptoms will usually take a favorable turn. The abdominal pain and hectic fever diminish; more nourishment is taken; the tongue begins to moisten at the edge, and the cracks to heal; the rough cuticle, especially that of the abdomen, to desquamate. Sometimes sweating is re-established suddenly, and with the appearance of a copious eruption of sudamina over the chest and abdomen; the bowels may continue loose, but the stools are of a darker, greener color. At this stage a relapse is very common—the diarrhœa, vomiting, and hectic returning with the former severity. The patient is not free from the danger of a relapse, until the stools have become solid. The improvement is slow, and, the diet being restricted, the emaciation persists for weeks. The desire for food is usually great; the digestive function is ultimately completely restored, and the patient regains his former weight and strength.

When the case tends to an unfavorable issue, the diarrhœa continues unchecked; the abdominal pain, and usually the tympanities also, increase; the exhausted patients lies motionless upon his side or back, drowsy or apathetic, and uttering feeble moans; the knees are drawn up, and his pinched, flushed countenance manifests pain on the slightest disturbance; the skin is pungently hot, the pulse very fast and thready, the teeth and tongue are blackened with sordes, [deafness, more or less marked, comes on.—H.] the continuous delirium lapses into coma —the typhous condition is complete. Watery stools are passed involuntarily, the patient hourly sinks, and at last quietly expires. As soon as the diarrhœa appears, and as long as it continues, the patient is liable to intestinal hemorrhage. The blood may appear repeatedly and in considerable quantity day after day in the stools, or the patient may become suddenly blanched and die of syncope, without any discharge of blood *per anum*. In such a case the intestines will be found distended with blood.

Hemorrhage, however, is not the only accident we have to anticipate. The patient is often cut off by perforation of the bowel. This dreaded event may be expected if, with a persistence of the diarrhœa, the tenderness and tympanites increase, and vomiting and hiccup supervene. Perforation is most commonly preceded by symptoms of general peritonitis accompanied by excessive tympanites, persistent hiccup, and vomiting. A paroxysm of more intense abdominal pain sometimes indicates the occurrence of this fatal result.

As an illustration of the insidiously progressive class of cases, I give the following outline of the history of a patient who came successively under the care of Dr. Murchison, Dr. Buchanan, and myself, in the London Fever Hospital.

Case 1.—C. Bushell, aged 24, a well-nourished, dark-complexioned woman, experienced a feeling of lassitude, accompanied by chilliness, pains in the limbs, and slight headaches, with loss of appetite, for about *four* days. She took to her bed on the *fifth* day, and was admitted on the *sixth*. A mild attack of typhus was suspected, and from day to day the tongue, pulse, surface of the chest and abdomen, and the nature of the secretions were examined. Still no positive disease declared itself, and no diagnosis was made. The pulse was 80–84, tongue natural, skin not hot and free from rash, bowels not acting every day, abdomen natural. During the time she remained in the hospital she exhibited no new symptom, complaining only of weakness, chilliness, general pains, and want of appetite. She ate fish, and subsequently meat, and was kept in bed

during a portion only of the time of her sojourn in the hospital. As she was apparently suffering from mere debility, and had improved a little, she was discharged on the *fifteenth* day. She was again admitted on the *twenty-ninth* day, and stated that she had not been well since she had left the hospital, having still suffered from excessive weariness and pains in the limbs, headache, and chilliness followed by a little feverishness. Lately she has had shivering, the bowels have been rather constipated, she has lost all appetite, and feels rather sick; pulse 120, feeble; tongue moist and white; no rash; no cerebral or pulmonary symptoms. *Thirty-first* day: tongue clean and red at edges, a moist thick fur on centre; bowels became rather loose, and six or seven rose-colored papules appeared on the abdomen; pain and gurgling in the right iliac fossa; pulse 120; skin hot; face flushed; sleeps badly. Day after day, to the *thirty-eighth* day, the purging increased, and fresh rose papules appeared; the abdominal pain increased; the tongue became dry, brown, and cracked; the pulse rose to 164. She died exhausted on the *thirty-ninth* day.

Autopsy.—Rotundity of the body preserved; lungs healthy, excepting engorgement of one lobe. Stomach, duodenum, and jejunum appeared healthy. The solitary and agminated glands of the lower part of the ileum swollen and inflamed; those near the valve were ulcerated and sloughy, and formed almost one continuous surface, raggedly disintegrated, and greatly swollen, extending around the whole of the last two inches of the bowel. A few of the solitary glands of the cæcum and ascending colon were inflamed and ulcerated. The corresponding mesenteric glands much swollen, congested, and softened. Spleen soft, twice its normal size; liver enlarged and fatty, weighing three pounds nine ounces (avoir.). Gall bladder distended with pale brown, watery bile, of excessively acid reaction and sulphuretted odor.

The *second class* of cases are perhaps the most frequent. In these the nature of the disease is manifest in the beginning. The patient may have felt a little indisposed previously; but he is in the midst of his usual occupations, or upon a journey, when he is overtaken with headache, shivering, and purging, followed by general pains and more or less pyrexia; there is complete anorexia, and nausea and vomiting are frequently amongst the earliest symptoms. There is pain in the abdomen, and great thirst. The prostration of the strength is very great, and the patient soon takes to his bed: the bowels continue to act two or three times a day, and the febrile symptoms and abdominal pain and tenderness persist; the tongue is moist, and usually coated with white fur; the edges and tip are red, and exhibit prominent fungiform papillæ. On the seventh day, or a little later, a few rose-colored papules appear upon the abdomen, chest, or arms; the belly is a little full; there is great tenderness and gurgling in the right iliac fossa; the patient is unable to take food, and is distressed by occasional vomiting of bilious fluid.[1] The fever runs high, there is great restlessness by day, and broken sleep and delirium at night. [Dulness of hearing is almost invariably present.—H.] Great pains are complained of in various parts of the trunk, the hepatic and splenic regions are tender, and there is increased dulness in the latter, indicating enlargement of the spleen. The breathing is often quick; there is some cough; and evidence of the presence of active congestion, or of acute inflammation of the lungs, is rarely wanting at this or a little later period. The bladder is very liable to become distended at this stage. These symptoms may persist with greater or less severity for the next week or ten days, the patient passing from two to six watery ochre-colored stools, containing a few shreddy flocculi, every day. The tongue becomes dry, with red irritable edges and tip, and elsewhere covered with a yellowish-brown cracked fur. If the patient have escaped the dangers of hemorrhage and perforation, he may, at the end of this time, begin to progress towards recovery, or, if the symptoms take an unfavorable turn, he will almost surely die. When the purging has persisted for weeks, the days are critical. The following is a common case of Enteric Fever, beginning apparently in ordinary diarrhœa:—

Case 2.—E. R., aged 19, a well-nourished healthy woman, taken while on a journey of pleasure with shivering and purging, followed by headache and general pains. The bowels had been regular previously. The purging and other symptoms continued, and she became slightly feverish, and lost appetite. She took to bed on the *fifth* day of her indisposition, and was admitted into the hospital on the *ninth*, presenting the following symptoms:—Pulse 104, full; skin hot; tongue moist, and coated with white fur, which is disposed to form cracks; bowels very loose; motions fluid, light, ochre-colored; abdomen tender, three distinct rounded and elevated rose-colored papules here and there; there is great thirst, and the patient is very feverish and fretful. *Tenth* day: tongue very thickly coated, cracked in the centre, clean and red at the tip and edges; bowels very loose; stools of greenish fluid; twelve or thirteen fresh papular spots on abdomen. *Eleventh* day:

[1] Vomiting is much more common in children than in adults.—H.]

pulse 96; tongue moist and superficially fissured; bowels still very loose; twenty-two fresh spots on abdomen; great pain across the abdomen and round the back. *Twelfth* day: tongue dry and brown at the tip and down the median line, aside of which it is covered with a thick crust of cracked yellowish-white fur; sides are moist and clean; stools frequent, copious, of light yellowish-brown fluid, possessing an acid reaction, and containing ragged yellowish flocculi; abdomen a little full; great tenderness and gurgling in the right iliac fossa; some fresh spots. *Sixteenth* day: the patient is emaciating very rapidly; pulse 90; tongue dry and red, devoid of fur except at base; purging a little diminished the last few days; yesterday's and this morning's stools together darkish-brown, fluid, alkaline; retention of urine, three pints drawn off; a few fresh spots, most of the old ones having faded and disappeared. *Eighteenth* day: no fresh spots; pulse 104, feeble; passes urine spontaneously. *Nineteenth* day: pulse 120; tongue dry, somewhat contracted, covered with a thin, dry, cracked, yellowish crust; much pain in the back and belly; cannot lie on the back "because it hurts her breath;" respirations 26; some fine crepitation at both bases behind; abdomen very tender; only two or three spots now visible; one copious ochre-colored stool this morning. *Twentieth* day: pulse 136, feeble; one copious light-brown watery stool; skin cooler; sleeps well; has been sick two or three times. *Twenty-third* day: is much better; pulse 100; tongue clean and moist, excepting a dry median streak; bowels not opened for two days; the rash has wholly disappeared; hunger. *Twenty-fifth* day: pulse 80; bowels act once in two days; stools light fawn-colored, semi-solid; abdomen natural, bears moderate pressure; tongue moist, but furred; hunger; to take solid food for the first time—fish and bread. *Thirtieth* day: slight relapse to-day; pulse 108; anorexia; thirst; pains in limbs; headache; skin hot; abdomen painful; no action of the bowels to-day; no fresh rose spots. *Thirty-first* day: increase of the feverish symptoms; pulse 126; a little diarrhœa; stools light yellow; a copious eruption of sudamina upon the abdomen. From this date she continued to improve, and was convalescent on the *fortieth* day.

In the *third class* of cases the symptoms are so sudden and severe that there may be suspicion of poisoning by some acrid narcotic, such as colchicum or poisonous mushrooms. We find the patient in a state of high fever; there is intense heat of the head; acute delirium; frequent vomiting and purging; the tongue is red and dry; the abdomen tense and painful. We learn that his illness commenced a few days ago, with vomiting, purging, and great headache. The patient lapses into a state of stupor; the diarrhœa persists, and he soon passes into the typhous condition, and dies on the eighth or fifth day, or even earlier. The following is a good example of this class of cases:—

Case 3.—Alfred S., aged 20, a powerful well-developed man, was admitted into the London Fever Hospital, October 7, 1865, in a state of stupor, pulse 156, very feeble, tongue dry and brown, conjunctivæ injected, head hot. He lay prostrate, passing liquid stools involuntarily, and died comatose twelve hours after admission; there was no rose spot or other rash upon the skin. His friends stated that he was suddenly taken ill with sickness and purging, followed by fever and delirium. The matters voided were of a bilious character.

Autopsy.—Body well nourished, skin clear. *Head*—meninges, and brain, quite healthy; the ventricles and theca vertebralis contained only one ounce and a half of serum. *Chest*—lungs engorged, weigh three pounds, everywhere crepitant. Heart healthy, small clot in right ventricle. *Abdomen*—liver weighs three pounds three ounces, softish and flabby, a little fatty; gall-bladder distended with pale watery faintly acid bile of the color of urine. Spleen enlarged, weighs fourteen ounces, natural in color and consistence. Stomach slightly injected at the great end; duodenum and jejunum healthy; ileum of a violet color externally; seven feet from the ileo-cæcal valve, a Peyer's gland, an inch long, was slightly swollen, and presented a prominent vascular elevation at one end. In the last six feet every Peyer's gland partially or wholly red, swollen and reticulated. In the last four feet the glands were much elevated and the villous surface abraded; the larger patches were raised a fourth of an inch above the level of the mucous membrane; all were very soft, and exceedingly vascular, and of a fiery red color. Between the Peyerian glands were innumerable solitary glands, forming elevations the size of a pea, surrounded by bright red areolæ, and presenting yellowish unbroken apices. The intervening mucous membrane highly inflamed. The valve much swollen and deeply wrinkled. Cæcum and first foot of colon thickly strewn with swollen solitary glands as large as peas, having sloughy centres. A biliary calculus, the size of a kidney bean, lay at the lower end of the dilated appendix, which was healthy, excepting where one solitary gland formed a vascular elevation. A few of the solitary glands in the transverse colon were enlarged; with this exception, the large intestine was quite healthy. The follicular glands, at the root of the tongue, were injected and

swollen. The mesenteric and mesocolic glands were enormously swollen, congested, and soft. The mucous membrane of the larynx, trachea, and bronchi was very red and covered with frothy mucus; the kidneys were congested; the bladder contained eight ounces of clear urine.

The two following cases illustrate the difference in the progress, termination, and effects of the disease in different individuals under the same general conditions:—

Two young men—J. Bennett and C. Beale—of the same age, and equally strong and well nourished, and resident together in a house in the immediate vicinity of King's College Hospital, were taken ill with febrile symptoms, the former on the 14th of August, 1865, the latter a week afterwards. Both patients died; Bennett on the 12th of September—the thirtieth day of the disease; Beale on the 13th of the same month—the twenty-third day of his illness. The rose spots were not developed in either case.

Case 4.—Bennett was admitted into King's College Hospital on the *fourteenth* day. He stated that he was attacked with headache and shivering, followed by sweating, general muscular pains, and sore throat. An aperient produced a loose state of the bowels for a day or two. Deglutition was very painful for three or four days. He got better, but remained very feeble, and did not recover his appetite. On admission he was pallid and weak, the throat had recovered; the tongue was moist, and the pulse but slightly accelerated; there was no diarrhœa, no rash, no abdominal pain or tenderness. He appeared to be suffering debility from a previous febrile attack. He continued to improve, regaining a little strength and appetite, and was discharged at the end of a week.

On leaving the hospital he went to his work, but soon felt too weak and ill to continue it, and after three days he again applied for advice, and was readmitted into King's College Hospital on the 8th of September, when he came under my care. He was dull, peevish, and prostrate; since he left the hospital the bowels had been loose. At this date there was moderate diarrhœa. The face and skin were pallid, the latter hot, perspiring, and free from rash; the abdomen slightly tympanitic and tender. Pulse 108. Tongue dry and brown, covered with a thick cracked crust. Respirations 42; slight dulness and fine crepitation over the back of the chest. During the next four days he lapsed into stupor, and lay on his back with the eyes closed, the knees a little drawn up, moaning occasionally, and picking with his fingers, the wrists and forearms being constantly twitched. The diarrhœa was soon checked by sulphate of copper and opium, but he resisted when attempts were made to open the jaws and administer drinks; the pulse and respirations increased, and he died comatose, four days after admission, on the *thirtieth* day of the disease.

Necroscopy nine hours and a half after death.—Body somewhat emaciated, viscera warm, blood fluid. *Chest*—lungs congested, bronchi much injected, two yellow masses of solid matter, the size of peas, like tubercle, in the lower lobe of the right lung near the border. Heart healthy, contained a pale soft clot. *Abdomen*—liver enlarged, weighed three pounds nine ounces; bile pale, watery, small in quantity. Spleen of natural consistence and color, weighed ten ounces and a half. Mucous membrane of the large end of stomach much congested. Peyer's glands of the upper portion of the ileum swollen, vascular, and reticulated; all those, and great numbers of the solitary glands in the lower four and a half feet of the ileum greatly swollen and superficially ulcerated, the larger glands forming fungous elevations, with margins raised a fourth of an inch above the level of the thin wall of the bowel, and resembled large indurated chancres. Fig. 9 (p. 211) represents one of these glands situated at a distance of 15 inches from the ileo-cæcal valve. The centres were slightly depressed, and stained of a dirty greenish-brown color. The swollen glands were firm and transversely wrinkled. The solitary glands formed smooth rounded elevations, the greater number corresponding in size to the tips of the fingers; each one presented a firmly adherent central slough. Cæcum healthy, but the solitary glands throughout the rest of the large intestine, including the upper part of the rectum, formed sloughy elevations like those of the ileum. In the sigmoid flexure there were fifty-four such elevations. In the transverse colon only six. In the ascending colon they were as thickly strewn as in the sigmoid flexure. The mesenteric and mesocolic glands were greatly enlarged, vascular and softish. The brain was not examined; all the other organs were healthy.

Case 5.—Beale was admitted under my care into the London Fever Hospital on the 1st of September. His illness commenced a week previously with anorexia, cold chills, headache, sickness, pain in the bowels, and diarrhœa. *Eighth* day: pulse 96; tongue moist and furred at the margins; skin pallid and hot, no rash, no headache; mind quite clear. Abdomen slightly distended; gurgling in the right iliac fossa. *Eleventh* day: bowels became very loose, and the abdomen tympanitic and tender. *Twelfth* day: pain in the abdomen; in the evening bowels very loose. *Eighteenth* day: febrile condition con-

tinues; pulse 108 to 120; tongue moist and furred; skin hot, free from rash; face very pale. The abdominal symptoms—diarrhœa, tympanites, and abdominal pain—have daily increased in severity since the twelfth day, and to-day there is evidence of general peritonitis; six leeches were applied to the right iliac fossa. *Nineteenth* day: leeches caused profuse bleeding, which was stopped with difficulty by the nitrate of silver; pulse 132, weak; tongue dry and brown; bowels quiet. He gradually sank, and died on the *twenty-third* day of his illness, retaining a clear intellect to the last.

Autopsy.—Body somewhat emaciated. *Chest*—lungs weighed fourteen ounces, floated in water, contained a dirty-brown fluid. Heart healthy, contained a colorless clot in the right ventricle. *Abdomen* displayed the effects of general peritonitis, the lower part of the cavity contained about a quart of turbid serum, and the coils of the small intestine were adherent to each other, and to the lower part of the abdominal wall, by layers of solid lymph. Liver weighed three pounds and a quarter, soft, friable, and fatty. Bile moderate in quantity, of light ochre color, watery, and very acid, instantly turning blue litmus paper bright red. Spleen weighed ten ounces, of natural color and consistence, but flabby. Intestines distended; on separating the purple adherent coils of the ileum, a perforation a fourth of an inch in diameter was discovered six inches from the cæcum; the opening in the intestinal wall was plugged with the solid lymph that adhered to the contiguous coils of the bowel, so that there was no escape of fecal matter into the peritoneal cavity. Stomach, duodenum, and jejunum healthy. Intestines contained some smooth, soft, formed feces, varying in color from light ochre to dirty white. Mucous membrane of the ileum uniformly red and inflamed, covered over with tenacious firmly adherent mucus of a bright ochre color. The solitary and agminated glands of the upper portion of the ileum quite healthy; lower down they were vascular and swollen; two feet from the cæcum the first signs of ulceration, and in this last portion of the ileum the solitary glands were swollen to the size of a pea, and presented ragged excavated centres. The last twelve inches contained several Peyer's glands in a ragged state of ulceration, the ulcers having raised, firm, very vascular, and angry-looking edges, and irregular depressed surfaces, formed apparently of yellow sloughs, adhering to a raw, almost bleeding surface, beneath. These sloughs could be readily separated with the finger-nail. Their lower surface had a yellowish color; they were friable, and some parts had an almost cartilaginous consistence and paler color. After washing and careful examination these sloughs were found to be composed of solid lymph, agreeing precisely in physical and microscopical characters with the solid lymph which adhered to the corresponding peritoneal surface of the bowel. The harder and whiter portions were composed of lymph contained in the meshes of the areolar tissue of the gland, and were, therefore, really sloughs. The more advanced ulcers were seated on the inflamed and thickened muscular layer. The perforation corresponded to the centre of one of the large ulcers. The cæcum, colon, and rectum, free from inflammation and perfectly healthy throughout, and the solitary glands inconspicuous. Mesenteric glands greatly congested and swollen; those lying in the angle of junction between the large and small intestine, the size of pigeons' eggs. Pancreas hardish, but apparently healthy. Bladder empty, healthy, as were the remaining viscera.

These two closely-associated cases are interesting, as illustrating the influence of constitution upon the progress of the disease. Bennett died in a typhous state from nervous complication, and with an amount of intestinal disease at least six times greater than that to which Beale succumbed a week earlier. Yet the intestinal disease in Bennett's case was latent to within five or six days of his death; the solid thickening of the affected glands (see Morbid Anatomy) forming, and promising to continue to do so, an effectual security against perforation. One of the parents of this young man died of consumption, and he himself had evidently been affected with syphilis.

Condition of the Alvine and Urinary Excretions in Enteric Fever.—(*a*) *The Stools* are remarkable for their fluidity and the absence of healthy bile; they have a pale ochre or drab color, and a sickly, offensive odor. On standing, a flaky matter is deposited, composed of epithelium, disintegrated sloughs from the intestinal ulcers, and undigested particles of food. According to Dr. Parkes (Med. Times, June, 1850, p. 396), the supernatant liquid has a specific gravity of 1015, and contains about 40 parts in 1000 of solid matter, consisting chiefly of albumen and soluble salts, particularly chloride of sodium. The stools are already in a state of decomposition, and after standing a short time are almost invariably alkaline. Immediately after they are passed they often have a neutral and sometimes an acid reaction. The offensive ammoniacal fluid contains much triple phosphate.

If salts of bismuth, lead, silver, or copper, have been administered, the dejections have a dark greenish-brown, or black color.

(*b*) *The Urine* in Enteric Fever does

not differ appreciably from that excreted in other inflammatory diseases. On the first accession of the febrile symptoms its quantity is usually diminished, but afterwards it becomes copious. As in all other febrile states, the chlorine is greatly diminished and the urea and uric acid increased. The chlorine is often reduced to a mere trace. The quantity of urea and uric acid excreted appears to be proportionate to the degree of fever; when the pyrexia is at its height, the quantity of these constituents excreted in twenty-four hours is usually doubled, sometimes trebled. As the fever declines the quantity of urea and uric acid diminish to the normal quantity or below it, while the chlorine reappears more slowly. In case 2, sixty ounces of darkish-colored, clear, acid urine were drawn from the bladder on the *sixteenth* day. After standing twenty-four hours it was quite bright and free from deposit; specific gravity 1024. One fluidounce contained a quantity of chlorine equivalent to 0·36 grain of chloride of sodium, 14·8 grains of urea, and ·3 grain of uric acid: or, in the sixty ounces, 22 grains of chloride, 889 grains of urea, and 18·9 grains of uric acid. On the *twenty-first* day, when the febrile symptoms began to subside, the urine was copious and neutral; specific gravity 1016·4. A fluidounce contained a quantity of chlorine equivalent to 3·9 grains of chloride of sodium, and 5·8 grains of urea. On the *twenty-third*, the urine was copious, of specific gravity 1010, clear, pale, and a fluidounce contained a quantity of chlorine equivalent to 3·2 grains of chloride of sodium, and 5·8 grains of urea. A small quantity of albumen often appears during the height of the fever.

Occasional Symptoms and Accidents.—*Peritonitis*, local or general, is liable to arise whenever the ulceration of the coats of the bowel extends deeply towards the peritoneum. This membrane becomes highly inflamed in places corresponding to the bases of the ulcers, and from these circumscribed patches of inflammation the increased vascular action may spread and involve the peritoneum more generally, and produce considerable serous effusion. Perforation is occasionally prevented by the adhesion of the inflamed patch to a neighboring coil or coils; and if it should occur after this adhesion has been effected, a circumscribed abscess, which may ultimately discharge itself into the bowel, is formed. Perforation frequently occurs, however, under less favorable circumstances, and the fecal matter is extravasated into the peritoneal cavity. Sudden increase of pain, accompanied by vomiting, and soon followed by cold, clammy sweats, and collapse, announce the nature of the accident. Sometimes sudden collapse alone is the only indication of this fatal issue. In other cases the perforation has taken place so gradually, the aperture formed is so small, and the extravasation so inconsiderable, that the symptoms of peritonitis come on and attain their maximum very gradually, and without any sudden increase in the severity of the symptoms.

Perforation of the Bowel usually occurs within six inches of the ileo-cæcal valve, and in almost every case it is the small intestine which is perforated Next to the lower end of the ileum, the cæcum is most liable to perforation. "Out of 435 autopsies recorded by Bretonneau, Chomel, Montault, Forget, Waters, Jenner, Bristowe, or made at the London Fever Hospital, perforation was observed in sixty cases, or 13·8 per cent." (Murchison, p. 511.)

Tympanites is present to some degree in almost every severe case. It usually comes on a week or nine days after the purging sets in. When excessive, it is a very grave symptom. It usually precedes perforation.

Intestinal Hemorrhage is a frequent accident in severe cases. It was observed in twenty-nine out of 139 cases observed by Murchison, Louis, and Jenner. It is a grave symptom, inasmuch as it generally indicates deeply-extended ulceration. The hemorrhage, however, frequently has its source in the congested capillaries of the common mucous surface, near the junction of the large and small intestines. The blood is never much changed. If the intestinal fluid be acid, it will be dark. The quantity of blood passed from the bowel does not always indicate the amount of the hemorrhage. In the case of a young girl which I witnessed, under Dr. Todd's care, in King's College Hospital, a trifling hemorrhage appeared, and shortly after, death occurred from syncope. The small intestines were found distended with red, clotted blood.

Retention of Urine is frequently present at the height of the early pyrexia. This condition cannot be overlooked for many hours unless there be considerable delirium.

Pregnancy.—Abortion is almost certain to occur if a pregnant woman be attacked with Enteric Fever. The only two pregnant women who have come under my care aborted, the one at the third month of gestation, the other at the fifth. Both recovered well. *Phlegmasia dolens* is apt to be a secondary complication in such cases.

Sequelæ.—*Marasmus* is the necessary attendant and consequent of extensive and prolonged disease of the mesenteric glands. After morbid action has ceased in these, they often become atrophied, and remain for a long time in a shrivelled, flaccid condition. In some cases the di-

gestive and assimilating functions remain so defective that the patient fails to regain appetite and flesh, and slowly starves to death.

Imbecility.—Patients who continue long in a state of extreme emaciation commonly manifest proportionate defect of mental power. They become forgetful and apathetic.

Tubercle of the Lung is considered by some physicians to be a common sequel of Enteric Fever. Many cases presenting such an apparent sequence may be regarded as instances of tuberculosis *ab initio*. See "Associated Pathology" of Enteric Fever.

Partial Anasarca, unassociated with albuminuria, is an occasional sequel of Enteric Fever in enfeebled constitutions. *General Anasarca* is rare. A scrofulous girl, E. Gain, aged 18, lately came under my care in the London Fever Hospital, with well-developed Enteric Fever. General anasarca suddenly appeared on the thirty-fourth and thirty-fifth day of the disease, when the stools were solid, and she was convalescing favorably; she had not, however, left her bed. Œdema appeared simultaneously in the lower extremities, the face, and hands; it was preceded by acceleration of the pulse and increased heat and dryness of the skin, which was pallid, and rough from fine desquamation of the cuticle. The tongue was red and glazy, with very prominent fungiform papillæ—a condition which had existed throughout. The anasarca increased from day to day, and was associated with considerable ascites. The integuments of the abdomen and chest were very œdematous. At one time the eyelids were closed by swelling, and the patient altogether presented the same appearance as one laboring under an attack of acute dropsy after scarlatina. Simultaneously with the development of the anasarca, albumen appeared in the urine, and became very abundant. The secretion, however, retained its natural color, and was normal in quantity, and, as long as the patient remained under my care, was free from renal casts or other deposits.

Morbid Anatomy.

Wherever the source of morbid action in Enteric Fever may lie, its effects are constantly manifested in the small intestine, and it is upon the solitary and agminated glands of the lower third of the ileum that the disease usually expends its virulence.

Without positive evidence of inflammatory action in these glands, the disease would not be Enteric Fever. How far the converse of this—the inflammatory lesion of Peyer's patches is always due to a specific Enteric Fever—is true, will appear upon consideration of the Associated Pathology of the disease.

Morbid changes, consequent upon Enteric Fever, are found (*a*) in the solitary and agminated glands of the intestine; (*b*) in the mesenteric glands; (*c*) in the spleen; (*d*) in the liver.

(*a*) *The Solitary and Agminated Glands.*—A Peyer's gland or "patch" presents in a state of health a variable number of rounded, shallow, concave depressions, averaging $\frac{1}{10}$ of an inch in diameter, and separated by narrow linear ridges of mucous membrane, running in from the general mucous surface and on a level with it, and forming a network, in the meshes of which—*i. e.*, in the depressions—the so-called "closed follicles" lie. In death, after the ninth day, from Enteric Fever, we shall rarely fail to find these and the solitary follicles in every stage of inflammation.

At the distance of four feet from the ileo-cæcal valve we shall generally find Peyer's glands in their normal condition. Six inches nearer the valve we may find one in the earliest stage of inflammation; it is slightly swollen, and raised above the general level of the surrounding mucous membrane, and it is a little more vascular than in health.[1] On careful examination the swelling is found to implicate the network of mucous membrane chiefly; the ridges between the closed follicles are more vascular, wider, and more prominent than in health; and the intervening depressions are thus contracted and deepened, and the patch is more distinctly reticulated. The follicles themselves appear to remain unaltered; minutely examined under water, they have a dark, semi-transparent, violet-gray appearance, while the intervening ridges are injected with minute divergent bloodvessels. Seen at a distance, the patch is clearly distinguishable from the common mucous surface. The general appearance is that of a fine pink or white swollen network, with dark rounded meshes. Passing downwards towards the ileo-cæcal valve, each succeeding gland presents the above-described characters in a more marked degree, and the patches consequently become more

[1] Roederer and Wagler call attention to a black dotted appearance of these glands, "resembling a freshly-shaven beard." This is the *forme pointille* of French writers. We have frequently seen this appearance, in persons dead of disease not affecting the intestines, produced by the exhibition of metallic salts. The cellular constituents of the intestinal glands become impregnated with the iron or copper salt, and on contact with the bile, a black sulphide of the metal is formed, dyeing these minute corpuscular masses black.

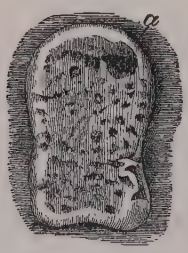
prominent and distinct. Fig. 1 represents an agminated gland in this early stage of inflammation. It was situated thirty inches from the ileo-cæcal valve.

Fig. 1.

Commencing inflammation in a Peyer's patch.

The ridges were wide, prominent, and very vascular, and the depression contracted and deep; at *a*, the swelling and contraction were greatest. Fig. 2 repre-

Fig. 2.



Inflamed agminated gland.

sents the next patch, nearer the cæcum. This gland was much swollen and soft, and formed a prominent, fungous-like projection of the mucous membrane. Its borders rose abruptly from the general mucous surface, and were smoothly rounded, devoid of reticulation, and slightly more elevated than the central parts of the patch. The ridges were greatly swollen, so as to convert the depressions into minute deep pits. The next stage consists in the breaking down of the swollen mucous membrane around the dark pits, and the formation of circular aphthous-like ulcers, each having for a centre a depression corresponding to a closed follicle. If this disintegration be general, the swollen gland soon presents a ragged, spongy appearance; examined under water, we find the irregular surface to be composed of a fine stroma of dirty, shreddy, fibrous tissue, containing a number of circular, rounded excavations: these are the follicles; they have not undergone further enlargement than slight thickening of their walls, which are thus rendered very distinct. In many places the follicles are seen to be dissected out, and only loosely connected with the surrounding shreddy tissue. The glands in the last foot of the ileum are always more or less implicated, and the innumerable and closely-placed solitary glands which form an almost continuous layer around the last two inches of the small intestine,—and which in some subjects are aggregated into one great terminal gland, the margin of which is coincident with the margin of the valve itself,—never altogether escape: and usually, indeed, the inflammation appears to have expended its whole force upon the glands of this part, and we find nearly the whole circumference of the last two inches of the mucous membrane greatly swollen, and in a ragged state of disintegration. The margin of the valve is not infrequently found as thick as the lips of the subject, and this part of the bowel usually presents a dirty ash-gray appearance, veined with blackish-purple ramifications. Some glands are merely swollen, and their turgid, everted margins overlap the contiguous mucous membrane; others are converted into ashy sloughs (*forme gangréneuse*, Cruveilhier), and often deeply stained with bile, sometimes dyed with blood. In some cases the ulcers are vascular and angry-looking; in others they are pale, anæmic, and have but slightly raised margins. Just as the inflammation does not always equally affect all parts of the Peyer's patch, so we very often find that the ulceration may be partial. A given gland may present one or several distinct ulcers. They rarely exceed $\frac{3}{8}$ths of an inch in diameter; they have rounded, elevated borders, and at first sloughy, ragged, broken-down centres; the most advanced ones have the bare, smooth layer or circular muscular fibres, or only a little intervening areolar tissue, for their bases. In the early stage the muscular tissue is pale and free from inflammation, but sooner or later it becomes red, thickened, and soft, and soon yields to the ulcerative process. The longitudinal layer yielding in like manner, the diminishing base of the ulcer comes to lie upon the peritoneal coat. In proportion as the base of the ulcer now nears the peritoneum, so does that membrane increase in inflammation: and if the ulcers be deep and numerous, the inflamed patches become confluent, and the outer surface presents the appearance of intense inflammation, and is occasionally covered with a layer of plastic lymph. Occasionally the ulcerative process extends through the peritoneal covering, and symptoms of perforation ensue immediately, or are retarded for a time by the adhesion of solid lymph exuded

upon its outer surface. The aperture formed in the peritoneum rarely exceeds three lines, and it is almost always formed within a distance of six inches from the ileo-cæcal valve. Sometimes the whole patch is converted by the confluence of smaller ulcers into a single deep ragged one, the sharp and perpendicular edges of which irregularly excavate the red, tumid mucous membrane immediately surrounding the diseased gland.

Occasionally the inflammatory process does not pass so soon into the gangrenous or ulcerative stage, and the glands become firmer and more prominent; the reticulations are completely effaced by the swelling, and the surface of these expanded, mushroom-like projections has a granular appearance (*forme granuleuse*, Cruveilhier). Glands in this condition may be restored to their natural state by resolution, or they may pass into the subsequent stages of gangrene or ulceration. The "*Plaques dures*" of Louis, which "à l'incision offrent une section ferme, lisse, et brillante," are very rarely observed in Enteric Fever distinguished from tuberculosis. In upwards of thirty fatal cases which I have examined, I have found this condition in only one (case 4), and in this I am inclined to attribute it to syphilitic taint. Fig. 3 represents one of the

Fig. 3.

Thickened and altered Peyer's gland. *a*. View from above. *b*. Vertical section.

chancre-like Peyer's patches from case 4: *b* will serve to convey an idea of the uniform thickening of the gland, overlying the unaltered muscular and peritoneal layers.

The solitary glands of the small intestine, and frequently also those of the cæcum and ascending colon, share more or less in the above-described changes. These minute glands occur in increased numbers towards the ileo-cæcal valve, where they become closely aggregated. Placed beneath the mucous membrane, and attached to its under surface, they lie loosely imbedded in the submucous areolar tissue, and in their healthy condition are hardly perceptible.

In many cases of Enteric Fever we find the last two feet of the ileum strewn with minute, round, semi-transparent elevations, varying in size from a mustard to a hemp seed. These are the solitary glands in a state of inflammation. In this early stage of the inflammatory process they have the appearance of a fine miliary eruption, and constitute the condition known as "*Psorenterie.*" When the solitary glands attain a larger size, and become a little harder and more opaque, the mucous membrane appears as if studded with pustules (*forme pustuleuse*, Cruveilhier). This appearance gave origin to the idea that Enteric Fever was "intestinal Variola." These swollen glands, however, are almost always solid: in only one case have I observed them to contain a yellow pultaceous matter, resembling inspissated pus.

If all the solitary glands be involved in the inflammatory process, the mucous membrane is thickly studded with them, and in the last two feet of the ileum the distance between them will average about $\frac{1}{8}$th of an inch. When an aggregation of a few solitary glands is swollen, a stud-shaped elevation is usually formed.

According to my own observations, the solitary glands are affected in proportion to the severity of the inflammation of the Peyerian glands.

In very rare cases the solitary glands alone are affected in Enteric Fever.

In many cases the disease is equally developed in the small intestine and cæcum; once I have seen death from perforation of the cæcum. Occasionally the large intestine is more extensively ulcerated than the small. In case 19, for example, the small intestine escaped, and the inflammation affected the solitary glands of the large intestine almost exclusively.

In proportion as the solitary glands are inflamed and swollen, they cause a projection and thinning of the mucous membrane. Attentively examined under water with a pocket-lens, they are seen to be of a delicate pink color, and exhibit a minute dark central point. Occasionally the swollen gland presents a yellowish summit surrounded by a minutely injected areola of converging bloodvessels. Ulceration commences by the softening and abrasion of the mucous membrane around the summit of the gland, the disintegration then becomes deeper, and spreading outwards, minute circular ulcers, with sloughy, shreddy centres, and purple, tumid margins, are formed. These ulcers rarely exceed $\frac{3}{8}$ths of an inch in diameter. Their further progress is identical with that of the ulcerated agminated glands, and they are equally liable to produce hemorrhage

and to perforate the bowel. In most cases we find a few of the solitary glands of the cæcum and large intestine thus inflamed and ulcerated. Occasionally the glands of the large intestine are more or less implicated along the chief part of its extent, and by the confluence of the small ulcers very large ones are sometimes formed in the cæcum and ascending colon. The direction of these ulcers is generally transverse. In Enteric Fever, ulceration always commences in the solitary or agminated glands; and if these were the only "follicular glands" in the intestinal canal, the term "Follicular Enteritis," by which Enteric Fever has been distinguished, would be a very suitable one.

We have now to consider the *nature* of that morbid process, the effects of which have been described. From the description just given, it is clear that the process is an inflammatory one. Usually there is evidence of very acute inflammation. It will be inferred from the foregoing description of the diseased glands that the inflammatory products are formed around the closed follicles, and not in their interior. Very careful observation leads me to speak positively on this point. If the new material were formed within the closed follicles, as Goodsir concludes, the follicles would indeed "become much distended," and, as a result, they would form projections upon the surface of the Peyer's patch, which I have never observed to be the case. On the contrary, I have always found them in the earliest stages of the inflammation to be placed far below the swollen ridges of mucous membrane and submucous tissue surrounding them; and in the latter stages, the follicles are completely buried beneath the inflamed surface of the patch, and concealed from view, and it is only when the excessively vascular and turgid ridges of the mucous membrane and subjacent tissue are disintegrated, that the follicles are again discovered, lying deeply in the abundant submucous tissue, and exhibiting little or no increase of size. The parts immediately surrounding them appear to have undergone considerable disorganization; for the follicles are often dissected from the surrounding parts, and remain attached to them by only a few tough fibres. In health, each follicle is surrounded by a close network of bloodvessels, which, as far as I have observed, chiefly constitutes the wall of the little gland; from this parietal network other branches, exceedingly fine and delicate, pass towards the centre of the parenchyma. If the vascular excitement be moderate, the central, as well as the circumferential parts of the gland, may increase in size; but usually the inflammation is acute. Cut off from all other parts of the circulation, and surrounded by inflamed vessels, congestion and stasis would very soon occur in the delicate vessels which pervade the parenchyma; and thus, whilst the parts external to the follicles would be increasing under the influence of the inflammation, the central parenchymatous parts would undergo no increase, but would tend to atrophy and disintegration. Hence the formation of the centrifugal ulcers and sloughs around the follicle; and such indeed must always be the results of inflammation in parts which have a similar arrangement of bloodvessels within them.

Structure and Characters of the Inflammatory Product.—This we find to be cellular. On examining vertical sections of Peyer's patches in the early stage of inflammation, represented in fig. 1, we find that the submucous tissue is composed of a very loose network of very elegantly waved and reticulated fibrous tissue, from which the so-called walls of the closed follicles are not defined. The meshes of this network are filled with finely granular corpuscles of various sizes, chiefly spherical, and averaging $\frac{1}{300}$th of an inch in diameter. (Fig. 4.) A few cells of adi-

Fig. 4.

Fibrous reticulum, with inflammatory corpuscles.

pose tissue, arranged in single rows, are occasionally seen. Sections through the more advanced and ulcerated patches present the same arrangement of the fibrous stroma; the cells are equally numerous, but they are a little larger, and of more uniform diameter, averaging $\frac{1}{200}$th of an

Fig. 5.

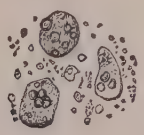
Enlarged granular corpuscles and reticulated structure.

inch, and a little more darkly granular. (Fig. 5.) Here and there a corpuscle is

observed containing one or more spherules of oil. Sections of the firmer swellings (*forme gaufrée*), and of those in a more advanced stage of ulceration, show that the corpuscles undergo fatty degeneration, and subsequent molecular disintegration. In these we observe multitudes of enlarged corpuscles containing spherules of oil, and much intercorpuscular molecular matter. (Fig. 6.) Rokitansky speaks of "the deposition of a typhous product" in the inflamed glands. The swelling, according to my own observation, is due to the rapid growth of the corpuscles forming the parenchyma of the glands, whether Peyerian or mesenteric.

Not unfrequently fibrinous exudation forms upon the surface of the ulcerated gland (case 5), or amongst its cellular constituents (case 4). Sections of the gland, which I have delineated in fig. 3, showed the elements represented in fig. 6, interspersed with minuter corpuscular matter and molecular fibres.

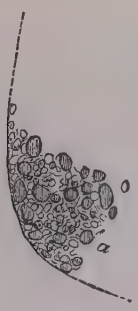
Fig. 6.



Corpuscles from ulcerated gland.

The *villi* upon the diseased patches and contiguous mucous membrane have a smooth outline and are denuded of their epithelium. They present a finely granular appearance, due to the presence of innumerable homogeneous, yellowish-tinted, refracted granules, which average $\frac{1}{500}$th of an inch in diameter. Some attain $\frac{1}{250}$th of an inch; others are mere molecules. Fig. 7 represents a minute portion of such villus highly magnified.

Fig. 7.



Portion of granulated villus, Peyer's gland.

Stages of the Local Disease.—Since the disease is usually developed so very insidiously, it will be difficult. and in the early stages impossible, to predicate with certainty the actual condition of the intestinal glands. The following generalizations, however, may prove useful (see also Diagnosis). For the first *nine* days the glands are undergoing inflammatory swelling, and at the end of this period they will be found projecting three or four lines from the mucous membrane, in the form of red, or purplish, fungous, soft excrescences, free from erosion. If death occur any day before this period, we shall find the glands more or less advanced towards this condition. About the *tenth* day the inflammation either subsides or increases. Resolution is effected in the usual way by diminution of the vascularity and swelling. If the inflammation increase, the swollen glands become a little firmer, and on the *eleventh* and *twelfth* days present softening, and erosion of the mucous membrane covering them. *Fourteenth* day: circular disintegrations around the follicles; a spongy sloughy appearance of the abraded patch, which is frequently stained of a deep ochre color by the bile—the formation and separation of ashy sloughs. *Sixteenth* day: complete separation of the sloughs, leaving ulcers limited below by muscular fibres or peritoneum and surrounded by red, swollen margins of mucous membrane; erosion of bloodvessels, and hemorrhage. *Twentieth* day: cicatrization begins. *Fortieth* day: cicatrization completed.

Reparation of the Intestinal and other Lesions.—In those who have died during a relapse of Enteric Fever, or at an advanced period, of pulmonary or other complication, we may often observe the process of reparation of the local disease. The following case exhibits the condition of the abdominal viscera during recovery from a severe attack of Enteric Fever, with pneumonia. The patient died of gangrene of the cheek (cancrum oris) and lungs.

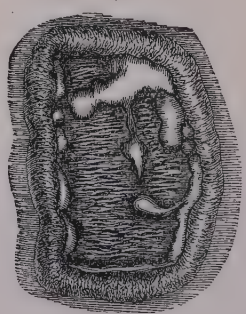
Case 6.—Joseph Taylor, aged 15, came under my care, August 15, 1865. He had been ill *three* days with headache, nausea, diarrhœa, and fever, and presented on admission all the symptoms of well-developed Enteric Fever (without rose rash, which never appeared), and pneumonia of the left lung. On the *sixteenth* day: pulse 144; respirations reduced to 28; diarrhœa and abdominal tenderness somewhat abated; dulness and fine crepitation over both bases of lungs behind. Three black sloughs, the size of peas, have formed in the mouth, two on the gums and the third on the centre of the left cheek. *Nineteenth* day: pulse 162, hardly perceptible; tongue dry and brown; bowels very loose; passed a considerable quantity of blood in the stools to-day; slough on the cheek spreading; cheek hard and swollen. *Twenty-second* day: pulse 144; moderate intestinal hemorrhage every day; diarrhœa restrained; cheek much swollen, duskily

flushed, hard, and shining; respirations less frequent. *Twenty-fourth* day: bowels quieter; no more hemorrhage; takes drink well and sleeps fairly; slough of cheek extending, those of the gums separated with the loss of two molar teeth. *Twenty-sixth* day: remains quite conscious and takes drink well. The left cheek is livid externally, and the eyelid closed by the swelling. From this date the pulmonary and abdominal symptoms declined, and the bowels acted naturally, the stools becoming solid. The gangrene, however, spread externally, and involved all the central parts of the cheek in a large circular slough, and the patient gradually sank, retaining a clear intellect throughout the disease, and died on the *thirty-second* day.

Autopsy.—Body much emaciated. *Chest*—lungs weighed together twenty-four ounces; apex of the left gangrenous, and partially broken down; lower lobes of both firm, slightly crepitant, pale-red, friable — recovering from pneumonia — here and there a small circular ashy slough; no trace of tubercle. Heart healthy; blood fluid; right internal iliac vein, at its junction with the cava, firmly plugged with a yellow, friable clot. *Abdomen*—liver weighed two pounds six ounces; firm; lobules indistinct, with a whitish speckling in the form of minute stellæ; the gland did not appear to me to be fatty, but microscopic examination showed the cells to be greatly enlarged, destitute of pigmentary matter, and replete with oil. Bile abundant, pale ochre-colored, watery, acid. Excepting a few patches of minute injection of the mucous membrane of the stomach, the alimentary canal was healthy to within four feet of the ileo-cæcal valve. This lower portion of the ileum was much injected and dark red. At four feet from the valve, a small Peyer's gland, the lower end of which presented a round, gently elevated swelling, with a central irregular excavation the size of a hemp seed, limited externally by the healing, granular margin of the pink mucous membrane. Three inches lower down, a larger gland, the lower half healthy, the upper with four cicatrizing ulcers—three so far healed as to be converted into minute stellate chinks, surrounded by pale red, wide, smooth borders, scarcely elevated above the surface of the healthy portion of the gland. Below this gland were nineteen minute cicatrizing ulcers, chiefly of the solitary glands, all with rounded, smooth, very soft vascular borders firmly attached to the less vascular transverse or longitudinal layers of muscular fibres, which formed clean, smooth bases to all the ulcers. Next occurred six large ulcers caused by the destruction of the whole of the large Peyer's glands of this part; they formed large, smooth, and soft, interrupted depressions, limited below by the very distinct reddish-gray muscular fibres, and surrounded by pale-red, raised, and rounded sinous borders reposing upon the muscular layer: two or three of these ulcers presented rounded islets, or projections of smooth red, mucous membrane running in from the raised border of the ulcer, and on a level with it. (Fig. 8.) One of these large patches presented a minute contracting ulcer at either end, the intervening space being occupied by a smooth, grayish-white, opaquish, slightly-depressed membrane. Nearer the ileo-cæcal valve were thirty-one other ulcers chiefly affecting the solitary glands, and varying in dimensions from mere linear chinks to three-fourths of an inch. All were in process of cicatrization. In the next portion of the ileum—the last four inches—there were a great many similar ulcers, all clean and healing, but not quite so far advanced in this process as those situated higher up. There were three small and distinct healing ulcers in the colon, the last one situated at the distance of a foot from the cæcum. The cæcum, and rest of the large intestine, including the rectum, were perfectly healthy. The solitary glands were all visible and marked by a central black dot just as they appear in the meconium-stained bowel of a newly-born infant.

The mesenteric glands were, for the most part, as large as almonds, and so flaccid that they could scarcely be distinguished, in the mesentery, between the thumb and finger: they were of a dusky-gray or ashy color, and of an almost leathery toughness. Entire sections of them could be readily made, and these were as tough as fibrous membrane, and presented an abundant, finely fibrous stroma, the ordinary corpuscles, and a considerable quantity of highly refractive granules.

Fig. 8.



Ulcer of Peyer's gland, healing.

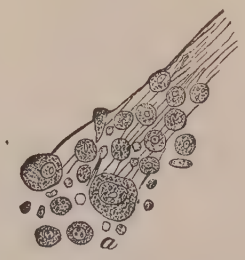
The recaptaculum chyli and thoracic duct were collapsed and empty. The spleen weighed five ounces and a drachm; it was of natural consistence, and pre-

sented a bright reddish-brown color on section. The remaining viscera were apparently healthy.

Floated under water, the rounded vascular borders of the healing ulcers present a double margin, the villi are seen to terminate in a wavy line, and from within and below the border so formed projects the paler and quite smooth soft border of advancing granulations. (Fig. 8.) Some of these spring up from the base of the ulcer, and form islands, which ultimately become confluent with each other and the margins of the ulcer, to form a smooth depressed membrane, which always remains destitute of villi and of closed follicles. In some of the cicatrized ulcers we occasionally observe a little cluster of closed follicles, but this simply points to the fact that a portion of the closed follicles of that particular gland escaped injury. After these follicles have been removed in the inflammatory process they are never regenerated. Years after an attack of Enteric Fever the ulcerated Peyer's patches will be found to be replaced by pale smooth, slightly depressed, but unwrinkled membranes, which are more firmly adherent to the muscular layer than the healthy gland, and remain permanently destitute of villi.

(*b*) *The Mesenteric and Mesocolic Glands.*—Just as inflammation of the tonsils induces vascular excitement and swelling of the lymphatic glands, situated about the angle of the jaw, so does inflammation of the solitary and agminate glands excite inflammation in the corresponding glands of the peritoneal folds. The swelling of the latter is always proportionate to the degree of the intestinal irritation; the glands, therefore, which lie in the angle of junction between the small and large intestine are those most affected. In every case of Enteric Fever we find that the mesenteric glands are more or less congested, swollen, and softened. They are usually of a dark purple color and of the size of hazelnuts. Some often attain the size of a walnut. Bisected with a sharp scalpel, the outer portions are seen to be of a uniform dark purple color, the central parts are less vascular, and the yellowish-white parenchyma is veined with diffuse purple streaks and a mottled appearance thus produced. The parenchyma seems yellower than usual, but this is simply the effect of contrast. The gland is so soft that it is difficult to make a thin section of any extent. Microscopically examined, we find it to be composed of an exceedingly delicate, friable, scarce stroma of indistinct fibres and of molecular corpuscles of various sizes. These latter constitute nearly the entire gland; they are for the most part spherical and nucleated: the most numerous average $\frac{1}{3600}$th of an inch in diameter; the larger present well-formed nuclei, and average $\frac{1}{2300}$th of an inch in diameter. (Fig. 9.)

Fig. 9.



Stroma and corpuscles from a mesenteric gland.

In the subsequent progress of the disease the glands may return to their normal condition, or the cells may break down to a creamy fluid. In one or two cases this puriform fluid has increased to such an extent as to rupture the peritoneal covering of the gland, and general peritonitis has followed the extravasation of its contents.

As soon as resolution of the inflamed, and cicatrization of the ulcerated, glands of the intestine have taken place, the mesenteric glands begin to decrease, and become for a time shrunken, flabby, and tough.

In those cases in which I have made the necessary examination, I have found the receptaculum chyli and thoracic duct empty and collapsed.

(*c*) *The Spleen* is severely congested in almost every case—probably, during the inflammatory period of the disease, in every case. It is usually enlarged to twice or thrice its natural size; occasionally it is found four or five times larger and heavier. Its color is uniformly purplish-black throughout, and it is so soft and friable that it may be reduced to a semi-fluid pulp with the greatest ease. Minute granular corpuscles, fibre-cells, and molecular branched fibres are the only structures I have been able to detect under the higher powers.

(*d*) *The Liver.*—A morbid condition of this organ and its secretion has been very generally observed. Forget does not specially mention the condition of the liver in many of his cases. Of others he records the following observations: "Liver normal, gall-bladder containing much or little, thin bile." (Obs. xlv. xlviii. lv.) "Liver voluminous, possessing a fatty appearance." (Obs. lx.) "Liver voluminous, gall-bladder almost empty." (Obs. lxviii.) "Liver presented a little softening in its right lobe; the gall-bladder contained a thin bile, slightly colored, like water." (Obs. lxxiii.) The liver was softer than natural in thirty-two out of seventy-three cases examined by Louis,

Jenner, and Murchison. (Murchison, p. 555.)

Louis states that the *volume* of the gland was augmented in $\frac{1}{10}$ of his cases, and in these it had lost its consistence; the *consistence* was diminished, the tissue of the organ being sometimes soft, sometimes friable, in the majority of his cases, and in none did it appear to him to be firmer than natural; *softening* existed in nearly half the cases, and in four to such a degree that the fingers sunk into the gland substance without resistance; the color was natural in only twelve of the subjects examined by him; it was redder than usual in eight, five of which were examples, more or less marked, of sanguineous engorgement. This appearance was noticed a little more frequently in those who died at an early period—from the eighth to the twentieth day. The *bile* was sometimes red, and very fluid in different degrees in about half the cases; in ten it was more abundant than usual. (Louis, Rech. Fièvre Typhoïde, vol. i. p. 269 et seq.) Another careful observer, Grossheim, remarked that, in all the cases observed by him, "the liver never retained its normal color, and the bile was always much thinner and clearer than in the healthy state. It was frequently transparent, sometimes clear yellow, sometimes of a dirty whitish color; in quantity, it was sometimes normal, rarely increased, but most frequently of all it was so diminished that scarcely any was left." (Edinburgh Med. and Surg. Journal, 1837, vol. xlviii. p. 178.) Stannius examined twenty-three cases of Enteric Fever. "In the majority, the liver appeared to be of normal consistence and color; not infrequently it was softened generally or partially. Almost always, both in those cut off at the height of the disease, and in those destroyed at later stages, the gall-bladder contained pale yellow, or yellowish-green, often watery mucous fluid, not reddening litmus paper nor tinging the skin." (Ibid. p. 174.)

My own observations agree with the foregoing; but as to the frequency with which the liver is found in a morbid condition, I am led to conclude that the gland *never* escapes without some alteration in its texture. In every case which I have examined, I have found the liver in a more or less advanced state of fatty degeneration, and in almost every case noted an increase of weight. Even when the gland is of normal size and to all appearance healthy, or only a little pale, microscopic examination will show very considerable degeneration of hepatic cells. In case 1, above recorded, the liver cells were greatly enlarged, averaging $\frac{1}{\overline{1000}}$ of an inch in diameter, and frequently containing spherules of oil $\frac{1}{\overline{5000}}$ of an inch in diameter. The bile in this case had the low specific gravity of 1018 and strongly reddened blue litmus paper. After depositing an abundant pale ochre-colored granular-looking matter, composed of columnar epithelium, it had the color of whey, or pale urine with a faint greenish tinge. I have constantly found the bile thin, watery, and easily filterable; in one case the specific gravity was as low as 1011·2. Filtered, and evaporated on a water bath, such altered bile yields only a small quantity of black solid matter, greenish-brown, by transmitted light, and wholly soluble in water. The bile itself, or this solution, gives slowly and faintly, sometimes imperfectly, the characteristic reactions of bile when tested with the mineral acids, or Pettenkofer's test. The bile has a strong post-mortem odor, and in one case which I examined twelve hours after death, when the viscera were still warm, and the blood steamed on exposure to the frosty air, it smelt strongly of sulphuretted hydrogen.

The morbid changes, above described as affecting the intestines, the mesenteric glands, the spleen, and the liver, are the constant and essential lesions of Enteric Fever. We now pass on to a cursory examination of such morbid phenomena as are exhibited by the other organs of the body.

Tongue.—The general condition of this organ has been described The characteristic features are, unusual redness of its edges, with enlargement and prominency of the fungiform papillæ, in the early period of the disease; and a wrinkling and cracking of the dry glazed surface, with contraction and reddening of the whole organ, at a later period. The cracks are very painful and often bleed. If the tongue remain moist, it is usually flabby, indented, and covered with white fur. In this condition it occasionally presents spreading ashy ulcers upon the tip and sides; and sometimes deep fissural ulcers, with pale everted margins, form across the dorsum. When nervous symptoms predominate, the tongue becomes covered with a thick, brown, firmly-adherent crust, very dry and hard, and reticulately fissured.

In several cases, I have observed great congestion and swelling of the follicular glands at the base of the organ.

The Lips and orifices of the *nostrils* are often cracked and inclined to bleed.

The Tonsils are rarely affected; abscesses have been observed in them in a few cases.

The Pharynx and Œsophagus.—Louis found small round or oval ulcers of the mucous membrane of the lower portions of the pharynx and œsophagus in about a sixth of his cases.

The Stomach, Duodenum, and Jejunum are usually healthy. In some cases they

present morbid conditions, such as softening and minute ulcerations of the mucous membrane, which are common to all inflammatory diseases.

The Pancreas.—I have usually found this gland harder and with the lobules more distinct than in health, as if shrunken. Otherwise it has appeared healthy.

The Urinary and Generative Organs are in the normal condition, or only slightly congested.

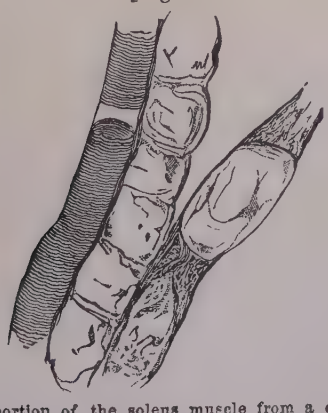
[The *Peritoneum* shows all the signs of acute inflammation when perforation of the intestine has occurred.—H.]

The Epiglottis, Larnyx, and Trachea are occasionally ulcerated. The mucous membrane of the bronchial tubes is usually red and swollen.

The Lungs present in almost every case evidence of pre-existing inflammation. (See Associated Pathology of Enteric Fever.)

The Muscular System.—Agreeably with what is observed in other protracted diseases of an acute character, the muscular tissue is found to be liable to degeneration in Enteric Fever. Zenker (Veranderungen der Muskeln in Abdominal. Typhus, 1864) describes two forms of muscular degeneration—granular and waxy. The granular form consists in the deposition of minute, highly refracting granules in the contractile tissue, giving to the fibres a dark appearance by transmitted light and obscuring the striæ. This molecular deposit is not wholly composed of fat. The degenerated fibres are very friable. The waxy form consists in the transformation of the sarcous tissue into a homogeneous colorless mass, glittering like wax, and causing a complete obliteration of the

[Fig. 10.



A portion of the soleus muscle from a case of typhoid fever. Preparation teased after treatment with Müller's fluid. × 200. Reduced ⅓. (Green.)]

striæ and nuclei of the fibres, the sarcolemma remaining intact. The waxy cylinders, thus formed, crack up into numerous fragments, which crumble down into a finely granular detritus, and this is gradually absorbed. The muscles most liable to degeneration are the adductors of the thigh and the abdominal recti. The affected muscles are of a pale grayish-red color. Rokitansky observed rupture of the abdominal rectus in Enteric Fever, and attributed it to spasm. Virchow noticed rupture of the muscles associated with friability of the muscular fibres in four cases of Enteric Fever. Zenker noted eleven such cases, all of which occurred in Enteric Fever. The rupture occurred most frequently, but by no means exclusively, in the rectus abdominis, pectoralis minor, triceps brachii, and psoas. The author last mentioned attributes the rupture of the muscles, and extravasation of blood into their substance, to the degeneration of the fibres above described. The rupture tends to produce hemorrhage, and this leads to the formation of collections of sanies or pus, which must be distinguished from general pyæmic deposits. Abscesses in the muscles are very rare in Enteric Fever.

The Skin presents us with one of the characteristic symptoms of Enteric Fever, the "*tâches roses lenticulaires*" of Louis. These spots closely resemble the papules of variola during the first few hours of their existence, but they are not quite so large nor so hard. They form slight, rounded, discrete elevations of a pale rose color, which fades away at the base, forming a moderately distinct circular outline. Each rose papule is a minute circumscribed inflammatory centre, from which the blush disappears on pressure. These spots usually appear on the abdomen and chest alone, but they are often found on the back. They are seen occasionally on the face and upper and lower extremities. The eruption is not always present. "Of 1820 cases admitted into the London Fever Hospital during ten years, it was noted in all but 224, or 12·3 per cent." (Murchison, p. 470.) The rash usually appears on the supervention of the acute febrile symptoms. It may be looked for at the end of the first week, and may continue as long as the febrile symptoms and diarrhœa persist. The total number of spots rarely exceeds fifty; in some cases they are innumerable. There is no relation between the quantity of the rash and the severity of the symptoms. It appears in successive crops; at first only two or three spots may be observed, next day four or five fresh ones, the next as many more. Each crop persists for a few days and then disappears. According to Barthez and Rilliet, and Murchison, the spots are fewer in children than in adults; and the former two observers state that in the same class of patients they are oftener absent in the severe cases than in the

mild. These rose spots occasionally appear in other acute diseases. In a severe case of typhus in a powerful fair-complexioned man I noted a very copious eruption of rose papules upon the chest and abdomen; they preceded the typhus rash, and had wholly disappeared when this became petechial.

The departure of the fever and the re-establishment of the cutaneous function is often announced by the eruption of *sudamina* over the whole of the chest and abdomen.

Roughness and minute desquamation of the cuticle, especially of that covering the abdomen, are observed after the cessation of febrile symptoms in severe cases. The desquamation occurs independently of the pre-existence of sudamina, which alone is sufficient to produce it.

The temperature of the skin usually undergoes a progressive increase during the first fourteen days of the disease, attaining, in severe cases, 104°, subject to the morning and evening vacillations, which are observable in other febrile conditions. If the abdominal or pulmonary symptoms undergo no amelioration, this temperature is often maintained during the earlier part of the day. When the intestinal inflammation proceeds to extensive ulceration, this high temperature may persist more or less continuously for weeks; but usually during the third week there are peculiar alterations of temperature, ranging from 4° to 6° per diem, the higher readings being observed during the hectic exacerbations which take place in the evening. Recovery in such cases is attended by a gradual diminution of temperature. In more favorable cases the resolution of the inflammation is declared by sudden falls of temperature.

When the Fever is prolonged, the pungently hot skin becomes very harsh, and the papillæ as prominent as in the *cutis anserina*.

The Lymphatic Glands are usually only secondarily affected in cases complicated with ulceration of the pharynx and erysipelas of the surface. In young children, suppuration of the cervical glands about the angle of the lower jaw is not very uncommon: three such cases have lately come under my care. Parotid inflammation, which is so common in typhus and in scarlatina, is rare in Enteric Fever.

Nervous System.—The only lesions discoverable are slight subarachnoid effusions, fulness of the bloodvessels, and slightly increased vascularity of the cerebral substance.

Circulatory Organs.—In protracted cases the muscular tissue is liable to fatty degeneration, and this change becomes first apparent in the left ventricle of the heart.

The Blood.—M. Trousseau, in speaking of intestinal hemorrhage in Enteric Fever, says the blood is exhaled by the mucous surface, as occurs in hæmatemesis, epistaxis, &c. "The proximate cause of this sanguineous exhalation," he goes on to say, "is a profound alteration experienced by the blood, and which is found in that state which one has termed 'the state of dissolution.'" (Clin. Med. p. 230.) M. Forget examined 123 specimens of blood, derived from persons in all stages of Enteric Fever. Of the blood drawn during the first period of the disease, only about $\frac{1}{15}$th of the specimens presented appreciable softening. In the second period $\frac{1}{7}$th of the specimens exhibited this change.

He concludes generally that an appreciable alteration of the blood in the several periods of Enteric Fever cannot be accepted as a general fact; that the blood is rarely altered in the first period; that the alteration is more marked in proportion as the disease is more advanced; that the alteration is not always in proportion to the gravity of the disease. (Forget, Sur l'Etat du Sang dans l'Entérite folliculeuse; Gaz. Médicale.)

My own observations of the condition of the blood of those who have died from Enteric Fever accord with those of M. Forget. In subjects dead in the third week of the disease, I have frequently found firm colorless clots of fibrin in the heart and roots of the great vessels. In protracted cases the blood not only becomes very thin, but is also much diminished in quantity, from sheer inanition.

Pathology.

If we carefully regard the incipient symptoms of Enteric Fever, we shall find that they have reference to derangement of the hepatic function. Often, long before the graver symptoms are developed, the patient loses appetite, the bowels are constipated, and the stools pale; the tongue is foul, and the digestion much impaired. All these symptoms point to a defective secretion of bile, and to a state of approaching inanition. Such a torpid condition of the liver may be produced in two ways in the development of Enteric Fever. It may result from severe or prolonged vascular congestion, in which the other internal organs participate; or it may be the effect of some morbific agent, carried by the portal vein from the intestinal surface into the liver, and causing, by a direct action upon its secreting corpuscles, derangement, or more or less complete paralysis, of its functions.

If in any case a poison be not decomposed in its passage through the alimentary mucous membrane, it must of necessity be admitted into the liver. We know how readily mineral poisons are conveyed and arrested here, and we recognize the

effects of certain vegetable substances upon the hepatic secretion. From these facts, and from its situation between the intestinal and general circulations, we may reasonably conclude, that it is one of the offices of the liver to arrest noxious matters in their way from the portal into the general circulation, to neutralize or decompose them, or to eliminate them from the blood, and throw them out again through the bile ducts into the intestine.

The very admission of deleterious agents into the portal circulation must lead, by diminishing the reciprocal attractions of the portal blood and the hepatic corpuscles, to congestion of the whole portal circulation.

Thus prepared, and by that concurrence of related actions which we everywhere witness in the body, the congested capillaries of the intestinal mucous membrane relieve themselves by a copious watery exudation, by means of which the poison set free by the liver is washed out of the alimentary canal. Such probably is the mode of action of elaterium, colchicum, &c. But it is the special function of the liver to prevent putrid decomposition within the body. If therefore the function of this gland be depressed, as in a case of simple vascular congestion from exposure to cold, for example, a septic poison may be generated within the body, and set up all the symptoms which follow the introduction of a similar poison from without. Doubtless, so long as the liver is in active healthy condition, any septic poison taken into the alimentary canal would generally be neutralized; but if the gland should happen to be torpid at the time, then the unaltered poison, upon admission into the liver, would possibly arrest the secreting corpuscles in the elimination of that very fluid which has the power of rendering it innocuous. Very little is known of the derangements to which the liver is liable, and of the alterations which its secretion undergoes. We readily obtain evidence of the grosser irregularities of the kidneys, but we can judge of those affecting the liver only by the color of the feces—a good general guide, no doubt; but only rarely is this means of diagnosis available in the incipient stage of diseases.

Primary vascular congestion of the liver, no matter how produced, leads to a vitiation of the secretions of the alimentary canal; nervous exhaustion results from arrested nutrition. Under these conditions the liver begins to degenerate, and the intestinal mucous membrane tends to ulcerate, the blood is imperfectly depurated, and general febrile disturbance ensues. Surely if high fever, violent delirium, and coma are the consequences of acute suppression of the bile, the pyrexia, headache, and the most severe delirium, which ever accompany Enteric Fever, may be fairly attributed to that diminution and derangement of the hepatic function which invariably accompany this disease.

That the liver is early and gravely deranged in Enteric Fever is proved by the facts already mentioned in the morbid anatomy of the disease, and by the prominency of those symptoms which have led observers in all ages to designate it by the terms, "bilious, gastro-bilious," &c.

In place of a thick, heavy, alkaline secretion, rich in biliary acids and coloring matter, we find a watery, neutral, or often excessively acid bile, notably deficient in its essential constituents, and sometimes putrid at its very source. M. Trousseau considers the flux from the bowels to be of the nature of a specific catarrh. But what is the *nature* of this specific catarrh? Is the bowel endeavoring to supply defective action of the liver by carrying away, in some unformed state, constituents of the blood which that gland should have removed as glycocholic and taurocholic acids? We do not think such a theory necessary. At the commencement of the disease there is probably some attempt at elimination, but in the subsequent stages we believe that the diarrhœa and intestinal lesions are rather due to congestion and mere local irritation than to any specific cause. This would appear to be the case from consideration of the fact that if we restrain the diarrhœa—the assumed means of elimination—we do not aggravate the general symptoms, but positively ameliorate them; and in most cases marked improvement follows the complete arrest of the diarrhœa.

Not the least important function of the liver is to prevent by its antiseptic properties the decomposition of the chyme; take away this preservative influence altogether from the system, and fermentation with the escape of gas and tympanitic distension follow. The impure chyme irritates the debilitated and congested mucous membrane, and what wonder then if inflammation, ending in ulceration of Peyer's patches and the follicular glands, should result?

But why should these particular structures suffer more than any other parts of the intestinal canal? For two reasons, we think: first, on account of the greater vascularity of these glands, whereby they most readily participate in local congestion, and, as has been shown, the arrangements of bloodvessels within them, which, when the circulation is obstructed, renders them liable to sloughing; and secondly, on account of their delicate cellular structure, for in febrile conditions it is the active growing corpuscles of the parenchymatous organs which most readily participate in the inflammatory process.

That the glands of the lower three feet of the ileum should be most affected may perhaps be regarded as a significant fact, and it is one for which it is difficult to find a satisfactory explanation. Anatomy will not allow us to ascribe a difference in function between the solitary and agminated glands lying near the junction of the small and large intestines and those removed to a greater distance from it; nor do we find that the glands of the upper and lower parts of the ileum have such a difference in their immediate associations as would account for unequal participation in general disease; and we should, therefore, be led to assume that if the solitary and Peyerian glands were employed in some general process connected with the elimination of a blood poison, they would be similarly affected. Such, however, is rarely or almost never the case in Enteric Fever, for the Peyerian glands of the lower third of the ileum are almost always found in a state of extreme inflammation when those of the upper two-thirds exhibit no morbid change, and we never find Peyer's glands of the upper portion of the ileum ulcerated when those of the lower are uninflamed.

The following considerations may afford some explanation of these facts. *First:* there appears to be a greater tendency to congestion of the lower than of any other portion of the ileum, due to the greater number of vascular solitary and agminated glands situated there, and also to the manner in which the small and large intestines are united. The abrupt fold forming the ileo-cæcal valve is similarly constituted to the anal sphincter, and, like it, necessarily causes some arrest in the flow of blood beyond its margins. We recognize, therefore, a predisposition in the lower part of the ileum, to participate in inflammatory action. *Secondly:* if we now regard the derangement which exists within the digestive canal we may be able to find an exciting cause in the altered action, which doubtless results from disturbance of the reciprocal action of parts engaged in the same function, but separated from each other by a considerable distance. Can we, for example, attribute the lesion of Peyer's glands in the lower portion of the ileum to defective action of the glandular apparatus situated in the higher portions of the alimentary canal? The liver, we have found, secretes bile deficient in those essential constituents which exert an important influence upon the digestive process. The defective bile probably contains sufficient of these constituents to maintain healthy action in the upper portion of the small intestine, but becoming exhausted of these in the lower, it there fails to exercise any antiseptic influence, and of itself induces unhealthy action.

But, it may be argued, if this were the true explanation of the lesions of the small intestine, how is it that the large bowel escapes; for, according to the theory, we should expect to find that the intestinal lesions would progressively increase from the lower third of the ileum downwards, instead of being confined, as is usually the case, to the lower third of the ileum and cæcum? The frequent immunity of the large intestine from any considerable participation in the disease may be explained by supposing that the irritation set up in the lower portion of the ileum, by the vitiated bile, causes such a copious exudation of fluid from this part of the alimentary canal, that the irritating matter is diluted, and at the same time so rapidly carried away through the great intestine, that the lower portion of the alimentary canal usually escapes any severe implication in the intestinal lesion.

There can be very little doubt that the dejections in Enteric Fever are chiefly thrown off from that part of the intestinal canal where the inflammatory irritation is greatest—viz. the lower portion of the ileum. The cæcum, where the secretions are necessarily retained for a time, is often as gravely affected as the last six inches of the ileum. In some cases, moreover, the large intestine is often severely involved in the disease, and occasionally, as we shall have an opportunity of showing, it is exclusively affected,—a fact quite consistent with the theory here advanced. *Thirdly:* the localization of the intestinal disease may be supposed to arise from derangement of that particular part of the sympathetic nervous system, which is distributed to the lower portion of the ileum, just as destruction of the eyeball may follow injury of the orbital branches of the fifth nerve. Morbid anatomy fails, however, to reveal such derangement of the sympathetic plexuses: and if it did, there would still remain the difficulty of accounting for a general febrile condition in such limited defect of nervous action. The question naturally arises to every inquirer, whether the symptoms of Enteric Fever are to be attributed to general blood-poisoning, or whether they secondarily arise as a consequence of a localized intestinal lesion. From the foregoing observations it will be seen that we are induced to conclude that the disease arises from a vitiation of only a portion of the venous blood, and that the constitutional symptoms are in many cases due to consequent derangement of the hepatic function. If we accept this view, we shall be at no loss to account for the great variation in the *nervous symptoms* observable in this disease. Some patients retain a clear intellect to the last hour of their lives (*e. g.* cases 5 and 6); others lapse into a state of stupor or coma at a very early period of

the disease (*e. g.* case 3); and the majority manifest great nervous irritability and prostration, and at some period or other, more or less delirium. In every case there can be no doubt that the derangement of the digestive, cutaneous, and pulmonary functions results in an impure condition of the blood; but we consider that in many cases the nervous symptoms are due to nervous exhaustion from inanition, or to active meningeal congestion, rather than to a specific blood-poisoning. The delirium partakes very much of the character of delirium tremens, and there is frequently very notable vascular excitement of the cerebral circulation. In those cases in which the cerebral symptoms are predominant, we are forced to recognize a general blood-poisoning, and then the question arises, Is this due to more or less complete suppression of the hepatic function, or to the admission of a specific poison into the general circulation? Probably it may be due to both of these causes. If the poison be arrested by and thrown out from the liver, no general blood-poisoning, and, therefore, no grave nervous symptoms, may ensue. If the liver be unequal to the arrest and elimination of the poison, it passes unaltered from the portal into the general circulation, and symptoms of general blood-poisoning at once appear; and if the gland be so far deranged in the process of elimination as to become almost paralyzed in its functions, more or less complete suppression of bile would be an additional cause of the cerebral symptoms.

Associated Pathology of Enteric Fever.

Pneumonia.—The lungs and the intestinal and mesenteric glands manifest very great sympathy in morbid action. In two, at least, out of every three of the many cases of pulmonary phthisis which I have examined, I have found the solitary and agminated glands of the lower portion of the ileum and the mesenteric glands more or less infiltrated with tubercle, and the former often very gravely ulcerated. The same sympathy is observed when the lungs are the seat of common inflammation, and in pneumonia we shall very often find corresponding inflammation of the solitary and Peyerian glands of the ileum. Reciprocally, of all the complications of Enteric Fever, pneumonia is the most common. In some stage or degree, I believe it is very rarely absent. In many cases the inflammation does not proceed beyond active congestion, the post-mortem evidences of which are engorgement with some friability, and the so-called "splenization or carnification."[1] According to the observations of Louis, inflammation of the lungs is more frequent in Enteric Fever than in any other acute disease. He found that splenization, simple or complicated with partial inflammation of the lung in the first or second degree, existed in twenty out of forty-six cases of Enteric Fever, and in seventeen there was actual inflammation. In only fifteen cases were the lungs healthy, or their alterations slight, little extended and consisting chiefly of change of color, due apparently to diffuse or partial congestions. Thirty-eight of his forty-six patients had cough at some period or other of the disease. (Louis, Recher. Fièvre Typhoïde, vol. i. p. 330 et seq.) These observations of Louis are in accordance with those of every other observer. In upwards of thirty cases examined by myself I have found the lungs free from the effects of more or less extensive inflammation only twice. [Such a frequency of pneumonia in Typhoid Fever is not observed either in private or in hospital practice in Philadelphia. Bronchitis, of moderate grade, is a usual symptom, beginning during the first week. Hypostatic congestion of the lungs occurs, in some cases, when the patient is allowed to lie continuously in one position, on the back. It affects the posterior lobes of both lungs; and may proceed to splenization or hepatization, sometimes with fatal result.—H.] The following case shows the intimate association of the two diseases. It is given by M. Forget as an example of "Follicular Enterite of the inflammatory form:"—

Case 7.—"A strong woman, aged 23, after exposure to hard work in the open air, experienced a sense of painful weariness, headache, nausea, vomiting, thirst, shiverings followed by heat, &c. *Third* day: diarrhœa. *Fourth* day: face flushed; skin hot and dry, pulse frequent, large, resisting; respiration frequent, without cough or pain; tongue, white at the centre, red at the edges; abdomen indolent; two liquid stools to-day. *Fifth* day: tongue red, denuded; meteorism; a liquid stool in the night. *Sixth* day: pulse 120, a little nocturnal delirium, dyspnœa, thoracic sibilance. *Seventh* to the *twentieth* day: continued in a typhous condition, with purging, dyspnœa, and more or less delirium. *Twenty-first* day: delirium, groanings during the whole night, deglutition difficult, several liquid stools, pulse frequent, thready: dyspnœa extreme; death.

"*Necroscopy.—Head*—notable injection of meninges. *Chest*—old pleuritic adhesions; both lungs engorged—a condition which

[1] M. Louis does not consider "splenization or carnification" as the result of inflammation, but such a condition developed during a general and continued febrile action cannot be regarded as being wholly independent of the inflammatory process.

appeared to have existed for some time—indurated, friable behind and at the bases. *Abdomen*—partial injection of the mucous membrane of the stomach and intestines. Towards the cæcum were met with, at first fine reticulated and swollen Peyer's glands, then rounded ulcerations, which became confluent, confused, fungous in the neighborhood of the ileo-cæcal valve and upon it: large intestine also presented traces of inflammation and numerous ulcerations; smaller, but more numerous than in the small intestine, and occupying almost its whole length. Mesenteric glands engorged, brownish. Spleen slightly enlarged, friable. Walls of the mouth and pharynx covered with a white pultaceous matter." (Traité de l'Entérite folliculeuse. Obs. lv. p. 414.)

In this case diarrhœa and dyspnœa appear to have commenced simultaneously. The patient died of pneumonia. Take away disease from the one lung, and truly we have, as far as the symptoms and morbid changes are concerned, a typical case of Enteric Fever. Are we, therefore, to attribute the lung disease in this case to a specific *typhoid* poison, the presence of which must be assumed to be proved by the intestinal lesion? Or may we not regard the pneumonia and enteric disease as mere local manifestations of one common inflammatory condition, probably produced by cold? We are inclined to adopt the latter view. In the outbreak of Enteric Fever in the two companies of soldiers under Dr. Grossheim's care, this acute observer could find no other cause for the disease, but "the violence and continuance of the military exercises, and the necessary exposure to great cold after being overheated by violent and laborious corporeal exertions." (Edin. Med. and Surg. Jour., vol. xlviii. p. 187.)

For the association of pneumonia and Enteric Fever, see also cases 6 and 19.

Pleurisy is almost as often present in Enteric Fever as pneumonia. Dr. Murchison observed recent adhesions, or effusion of lymph, in six out of nineteen cases; Sir W. Jenner, in six out of fifteen; and M. Louis, in two out of forty-six; but he found a greater or less amount of reddish, serous effusion in the pleural cavities in nineteen other cases. (Murchison, p. 560.)

Case 8.—Julia Hatch, aged about 30, died of pleuro-pneumonia on the *forty-fifth* day of the disease. She was admitted into the hospital on the *fourth* day, when the following note was made:—Pulse 120, tongue moist and furred, skin cool and moist, face flushed, respiration accelerated, slight dulness on percussion, and pleuritic friction sound at the base of the right lung behind; bowels regular. She improved, and took food with a relish until the *twenty-fourth* day, when the pleurisy attacked the left side; the febrile symptoms increased, respiration became hurried and oppressive. A second blister and mustard poultice, were applied to the chest, and on the *thirty-sixth* day pulse was 144, feeble; respiration much easier; face less livid; tongue clean; mucous crepitation and friction sound still heard, both before and behind, on both sides. Eats mutton-chops well. After this date she continues to get worse. On the *forty-first* day, pulse 144; respirations 50, labored; skin hot and very dusky; dulness, crepitation, and friction sound still heard. She continued in the same state till the day of her death. The condition of the skin, tongue, and abdomen was carefully noted from day to day, but, throughout, the digestive function was most regularly performed, the bowels acted naturally every day, and the motions were perfectly healthy. Considering her febrile condition, her appetite for food was unusual. On the *thirty-fifth* day she asked for meat, and enjoyed it. The abdomen was flat and natural, and there was never any appearance of rash.

Autopsy.—Body considerably emaciated; abdomen flat. *Chest*—costal cartilages partially ossified; extensive pleuritic adhesions on both sides, some of which were old, others evidently the result of the last illness; lower lobe of right lung adherent to the diaphragm and side of chest, soft, friable, and slightly crepitant, evidently recovering from recent inflammation. No trace of tubercle in any part of the lungs. Heart healthy, containing soft, yellow clots in the right cavities. *Neck*—fibrinous and serous exudation in the areolar tissue, around the trachea, and between the muscles in front of it. Slight œdema of the mucous membrane above the glottis, and redness of the trachea. Follicular glands at the base of the tongue much enlarged, with violet, swollen, everted margins and gaping orifices. Tonsils a little enlarged. Uvula much swollen. *Abdomen*—intestines undistended and undisturbed; the coils of the small intestine dark purple. The whole of the ileum was intensely inflamed, and every Peyerian gland swollen and prominent. Those in the lower three feet of the bowel, and also the intervening solitary glands, were greatly swollen and ulcerated—whole patches being excavated into ragged ulcers, with rounded, everted, intensely vascular borders, overlying the contiguous mucous membrane; the irregularly-excavated centres were deeply stained with bile. One ulcer, two inches from the ileo-cæcal valve, alone extended to the muscular coat, exposing a smooth surface, half an inch in diameter, of soft, swollen, muscular fibres. The non-ulcerated patches formed elevated fungous-like expansions. Many of the enlarged solitary

glands were deeply excavated at the centre. The intervening mucous membrane was excessively vascular. The cæcum was congested, the large intestine healthy, and contained well-formed, solid, bright, yellowish-brown feces. Mesenteric glands much enlarged, congested, and soft. The spleen, liver, kidneys, supra-renal capsules, and organs of generation were perfectly healthy. The gall bladder was full of healthy, green, viscid bile. Although the patient was subjected every day to close scrutiny, there was no suspicion of intestinal mischief at any time; there was not the faintest external indication of it, but the reverse. I examined the case chiefly with the view of ascertaining how far Peyer's glands are affected in acute disease, and I was surprised to find ravages much more extensive than are seen in ordinary cases of Enteric Fever, and such as would be considered to be eminently typical of the disease.

Laryngitis is a rare complication of Enteric Fever. I have noted it in two cases. In one it occurred during the height of the disease, and yielded to leeching and blistering. In the other it came on during convalescence. Suffocation impended for two days, but the patient escaped by the ejection of fragments of a tough, organized membrane.

[*Bronchitis* has been already mentioned as, in the United States, one of the usual symptomatic conditions, most marked during the first ten days.—H.]

Scarlatina and Diphtheria.—A very close relationship appears to exist between these diseases and one variety at least of Enteric Fever. All are, for the most part, autumnal diseases, and they may be observed to increase and decrease together, and all appear to arise spontaneously out of the same conditions. Stöber, Löschner, and Friedleben maintain that scarlatina and Enteric Fever prevail epidemically in an inverse ratio to each other, the one prevailing in proportion as the other declines. (Brit. and For. Med.-Chir. Rev., July, 1858, p. 162.) I have known several instances of scarlatina or diphtheria affecting one member of a family and Enteric Fever another, simultaneously. The day before C. B. (case 1) came into the hospital, her brother, aged 14, was admitted with "scarlatina in its most marked form." Sore throat, accompanied by the exudation of white pultaceous matter upon the mucous membrane of the fauces, frequently accompanies the early symptoms of Enteric Fever (*e. g.*, cases 4 and 7). Diarrhœa is often a severe complication in scarlatina, and in almost every fatal case of this disease inflammatory swelling of the solitary and agminated and of the mesenteric glands will be found. After most careful microscopical examination of these swollen glands, I have failed to distinguish the slightest difference between them and those of the first period of Enteric Fever. Scarlatina, I have reason to believe, often lapses into Enteric Fever, and such appears to have happened in the case of Julia Hatch (case 8). I have described this case under Pleurisy, but it is probable that its appropriate place would be under Scarlatina. I failed to get information as to her previous history, but the condition of the glands at the root of the tongue, and the neighboring inflammatory effusions, correspond exactly to the effects of scarlatina. Dr. Murchison noted the co-existence of scarlatina and Enteric Fever in eight cases, and the appearance of scarlet rash without sore throat in five other cases (pp. 518, 473). Other observers have frequently noticed the same facts. Barthez and Rilliet noted the co-existence of diphtheria and Enteric Fever in six cases, Forget in two, Louis in three, and Murchison in one.

The following case of "malignant scarlatina," associated with the anatomical lesions of Enteric Fever, is related by M. Forget:—

Case 9.—A strong man, aged 20, after his usual work, was seized with shivering; during the night, sensation of constriction in the throat, headache, fever, &c. *Third* day: carried to the hospital; hands, forearms, thighs, and chest "offrent une belle coloration scarlatineuse;" skin burning hot; pulse 140, small; tongue red, and covered, as well as the mouth, with a white pultaceous coating; throat painful; deglutition very painful; no diarrhœa; chest normal. *Fourth* day: partial stupor; eyes injected; coloration of skin persists; sudamina; pulse 160, thready; pultaceous coating of mouth diminished; back of throat very red, swollen; deglutition almost impossible; epigastrium tender; death.

Necroscopy.—*Head*—meninges injected. *Chest*—lungs engorged throughout, a little friable at the summit; heart filled with white clots. *Abdomen*—gastric and duodenal mucous membrane red, manifestly inflamed; small intestines presented, on approaching the cæcum, numerous Peyer's glands, of which some were simply dotted black, others were reticulated; the majority were red, swollen, firm, elastic, and prominent (gaufrées); an abundant miliary eruption (psorentérie) in a great extent of the small and large intestine, including the rectum.

"Ces caractères anatomiques sont tous ceux de l'entérite folliculeuse très développée, avant la période de gangrène et d'ulcération." (Forget, Obs. xix. p. 144.)

On the next page but one, the same observer gives the history of a case of "scarlatina suive d'entérite folliculeuse."

Such is the association which subsists

between scarlatina and Enteric Fever—an association closer and more frequent than is observed between the former and any other acute disease, and one which compels us to acknowledge some closer connection than mere accidental intercurrence.

Tracing the connection still further, we observe that the physiognomy, the character of the febrile action and delirium, and the condition of the tongue, are the same in both diseases. In both there is a tendency to epistaxis, cracking of the lips, desquamation of the cuticle, and dropsy (see Anasarca). In the other disease, the solitary and agminated glands of the upper part of the alimentary canal (the tonsils and intervening follicular glands of the tongue), and the neighboring lymphatic glands, are affected; in the other we find the corresponding parts (the solitary and agminated glands of the ileum, and mesenteric glands) of the lower portion of the alimentary canal diseased. Whether, therefore, we consider these two diseases in reference to their origin, their mode of development, or their physiological anatomy, we still find in either case a resemblance between them. It is only in their subsequent progress that we recognize a clear distinction.

This distinction has reference, *first*, to the nature of the contagious poison—scarlatina tends to spread as scarlatina, and contagious Enteric Fever as Enteric Fever; and *secondly*, to the progress of the two diseases—the one falls upon the cutaneous surface, the other upon the mucous. With regard to the first point, there is nothing in the history of the contagious diseases—at least of the diseases here compared—to dissuade us from the assumption that the contagious animal poisons are developed within the body, and derive their specific characters from the particular actions to which they may happen to be there subjected. Thus, for example, putrescent substances admitted, on the one hand, in a volatile form by the respiratory surface into the *arterial* blood, may be conceived to undergo, during the process of absorption, some special and definite change, whereby a specific poison is formed: and, on the other hand, if the same deleterious agent be taken in a liquid or solid form into the alimentary canal, and thereby admitted into that limited portion of the *venous* system—the portal circulation—we may reasonably assume that it may be peculiarly modified by the agency of the digestive secretions, so as to constitute, upon its admission into the blood, a poison different from that formed in the lungs, but somewhat related to it in its action.

But even assuming that the septic agent be not so diversely modified in the process of absorption, we may still find an explanation of the differences which ultimately distinguish the two diseases if we consider that, in the one case, the liver, a most potent converting agent, intervenes between the portal and systematic circulations, and that by its agency the skin and kidneys—the arterial organs most affected in scarlatina—may be in a great measure protected from a poison introduced by the alimentary canal. If the septic poison be simultaneously admitted into the blood by the lungs and intestinal surface, a mixed disease—scarlatina complicated with Enteric Fever, or the converse—may upon this theory result.

Those who have seen most of these two diseases, and have studied them side by side, will, we feel sure, be most ready to acknowledge how soon their distinctive characters become lost in the intermediate modifications which are observed to occur between them.

The allied affection, croup, is also an occasional associate of Enteric Fever. The following case is taken from M. Louis's work on Typhoid Fever:—

Case 10.—*Croup.*—A powerful man, aged 23, was attacked with slight pain in the throat, preceded by fatigue, lowness of spirits, anorexia, thirst, diarrhœa, and slight epigastric pains. *Third* day: shivering, heat, and sweating; diarrhœa each day very considerable; no sensible increase in the pains in the throat. *Fourth* day: considerable diarrhœa. *Seventh* day: considerable epistaxis; pains in the throat; soft palate red, without swelling; deglutition difficult and often excited; a sense of pricking and heat in the affected part. *Eighth* day: pain in throat continued; a shining semi-opaque false membrane upon the tonsils, sides of the uvula, which is inflamed, and upon the pharynx; voice a little changed; pain and difficulty of deglutition; four stools and copious sweats during the night; some lenticular rose spots upon the abdomen. *Ninth* day: false membrane more opaque, voice aginose, larynx a little tender, respiration a little accelerated. *Tenth* day: false membrane extending; deglutition causes insupportable anguish. *Eleventh* day: very fetid breath; croupal voice; deglutition impossible. *Twelfth* day: delirium and death.

Necroscopy.—Cervical glands enlarged to thrice their size and inflamed; false membrane upon the pharynx, the uvula, the soft palate, the epiglottis, and larynx; œsophagus healthy; mucous membrane of stomach and small intestine thickened and softened, and elevated by a kind of white granulations, miliary in the neighborhood of the duodenum, then proportionately larger as the cæcum was nearer; Peyer's patches more or less red and thickened in the ileum, their thickening

being due to swelling of the mucous membrane and subjacent cellular tissue; mesenteric glands large, of an amaranth red, especially near the cæcum, where they were softened; spleen thrice its natural volume.

"As to the symptoms peculiar to Typhoid Fever, if they were little marked, they announced, nevertheless, from their commencement, that the seat of the disease was in the abdomen." (Louis, Obs. xx. p. 187.)

Erysipelas is not frequently associated with Enteric Fever. Out of 199 cases of Enteric Fever, observed by Louis, Chomel, and Jenner, erysipelas was noticed in twenty. The following is given by Forget as a case of Enteric Fever. We would rather regard it as a case of erysipelas and phlebitis.

Case 11.—A strong man, aged 38, was under treatment in the surgical wards for erysipelas of the left hand, and on a certain day, when the inflammation was in process of resolution, he was seized, without known cause, with shivering, followed by heat, headache, vertigo, nausea, thirst, diarrhœa, and considerable prostration. He was transferred the same evening to the medical wards; expression stupid, sub-icteric tinge, abdomen tympanitic, gurgling, tenderness in right iliac fossa. *Second* day: same state: five liquid stools, *Third* day: pulse 100; six liquid stools. *Fourth* day: same state; diarrhœa; trembling of hands. The patient had been bled on the first day, and to-day one of the punctures is found gaping and exuding a puriform fluid, and the forearm and arm invaded by an inflammatory œdematous swelling very painful on pressure. *Fifth* day: pulse 120; prostration and stupor increasing; several liquid stools; cough; disseminated rales; sub-delirium, and death the same evening.

Necroscopy.—Jaundiced tinge of skin, right arm is considerably swollen; pus exudes on pressure from the gaping wound in the vein; vein thickened for length of two inches above the wound. *Chest*—lungs healthy, except a little posterior engorgement. *Abdomen*—alimentary canal healthy to within two feet of the ileo-cæcal valve; Peyer's glands are met with reticulated and swollen; in the intervals a slight psorentary; nearer the valve, and upon it, several patches are in the same state, but redder, more swollen, evidently inflamed, as were the surrounding parts of the intestine; mesentery contained glands swollen and reddish; spleen very large and friable, and no trace of purulent absorption anywhere.

"Voila, certainement, une entérite folliculeuse bien constatée, au cinquième jour." (Forget, Obs. xi. p. 119.) We confess that we cannot see more than the participation of delicate cellular organs—the solitary and agminated glands, the mesenteric glands and the spleen—in a general febrile condition. Had Peyer's patches been found in an ulcerated condition, the erysipelas, which is clearly the primary disease in this case, would probably have been considered to be a secondary complication of latent Enteric Fever.

The following case shows such implication of the solitary and agminated glands in a general inflammatory condition, as is very common in acute disease.

Case 12.—Mary W., aged 40, came under my care for erysipelas of the head and face, August 12, 1865. She had continued pyrexia and muttering delirium. Pulse 116 to 136; the urine was often retained, and the bowels were confined; tongue dry and brown; evacuations sometimes passed involuntarily; urine contained a little albumen; the stools were solid and natural; she died on the *tenth* day after admission. *Head*—brain healthy, but the vessels congested; two ounces of serum in the ventricles. *Chest*—lungs congested and carnified below and behind; healthy in front; heart normal. *Abdomen*—stomach, save a little finely dotted patch of ecchymosis, duodenum, and jejunum, healthy. Lower portion of the ileum and commencement of the large intestine injected. Peyer's glands in the last three feet of the ileum a little swollen and prominent; the intervening mucous membrane strewn with enlarged solitary glands the size of hempseeds, giving to the finger, as it passed over the inflamed membrane, a granular sensation. The mucous membrane of the transverse colon, corresponding to one of the longitudinal bands, was highly inflamed. Some Peyer's glands, higher up in the intestine, were stained with sulphide of iron, and were dotted with black. Here and there the contiguous mucous membrane was also stained black. She had taken perchloride of iron. The colon contained solid natural feces. Spleen weighed five ounces, and both it and the mesenteric glands were natural in size, color, and consistence. Kidneys and pancreas congested. Liver weighed two pounds fourteen ounces; it was pale, soft, and greasy; bile pale yellow.

Erysipelatous œdema of the glottis has occasionally caused death in Enteric Fever. Several cases are recorded by Jenner and Trousseau.

Case 13. — *Myelitis.* — "A delicate woman, aged 32, was seized at the catamenial period with sharp pains in the loins, the sides, and lower extremities. Menstruation continued the usual time, but the pains persisted and increased. *Fifteenth* day: dorsal decubitus, headache, prostration; face expressive of pain; moans and cries; the least movement is painful; the patient cannot be made to sit

for the examination of the spine, which is the seat of sharp pains in its whole extent; the joints are equally painful; the muscles and skin are everywhere extremely sensitive; prickling sensations in the hands and soles of the feet; she cannot stand; skin hot; pulse 100, small and hard; tongue furred; mouth clammy; anorexia; thirst; abdomen, like all the rest of the surface, tender; one stool a day. We diagnose a cerebro-spinal affection. *Seventeenth* day: acute pains; the patient says she cannot feel her limbs, but when they are touched she complains of extreme sensitiveness; spine painful on pressure throughout its whole extent; bowels confined. *Eighteenth* day: general pains; the patient cannot move. *Nineteenth* day: delirium, plaints; acute general pains; stools and urine involuntary; skin hot; sweating; pulse 112, large and supple. *Twentieth* day: same state; diaphoresis, sudamina, vomitings, and numerous stools. *Twenty-second* day: vomiting ceased; numerous involuntary stools; abdominal gurgling; immobility; acute pains provoked by movement; pulse 120. *Twenty-third* day: continued delirium; involuntary stools. *Twenty-fourth* day: the patient is pale, almost pulseless, and bathed in cold sweats; died this day."

Necropsy twenty-two hours after death. —*Head*—meninges slightly injected; brain of natural appearance and consistency; a little serum in the ventricles; coverings of the cord much injected; a great quantity of serum flowed from the spinal canal. The *spinal marrow* "est ramollie dans une grande étendue, sans changement de couleur; ce phénomène est évidemment cadavérique." *Abdomen*—stomach inflamed in patches. Small intestines present only some vascular ramifications to within two feet from the cæcum; then granulations (*psorentérie*) appear, then reticulated Peyer's patches, others ulcerated very numerous; some appear to be cicatrizing, and some already cicatrized; in the large intestine some isolated follicles appear to be affected; mesenteric glands engorged; spleen and liver present nothing of importance; the bladder contains turbid fetid urine, and its mucous membrane is strongly injected and dotted. (Obs. xxviii. p. 286.—Forget.)

M. Forget had headed this case "Entérite folliculeuse latente, prise pour une affection cérébro-spinale. Forme, rheumatismale." And after he has detailed the symptoms and post-mortem appearances transcribed above, he asks, "Where is the practitioner who would not have been deceived, as we have been, by appearances so fallacious? How recognize a case of follicular enteritis under such a predominance of sensitive nervous phenomena?" Only by regarding lesions of Peyer's patches so long and so exclusively that no other pathological condition can be conceived possible, we answer.

M. Forget attributes the softening of the cord to post-mortem changes, but the cord is not softer than the circumferential parts of the brain; it is equally well protected from maceration by its vascular coverings, and, from its situation in the axis of the body, it is less liable to post-mortem changes than the brain itself, which, in this case, is described "de consistance et d'aspect naturels." We know of no symptoms, or post-mortem appearances, which could more positively assure us of the existence of inflammatory softening of the cord, than those which the eminent Strasburg professor here places before us.

Phthisis.—In order to illustrate the similarity between the symptoms of tubercular ulceration of the intestines, associated with pulmonary tuberculosis, and those of Enteric Fever, I will here translate two cases from M. Louis's work on Typhoid Fever, and which this eminent author gives as examples, the one of ordinary Enteric Fever, accompanied by delirium, and the other of latent Enteric Fever, but which, with due deference to so great an authority on both phthisis and Typhoid Fever, we feel bound, from our own observations, to regard as cases of tubercular disease. We might have adduced instances in which the lungs were most extensively diseased, from our own practice, but we prefer to place before the reader the description and conclusions of some other author. The reader will form his own opinion on the nature of these two cases, bearing in mind the frequent co-existence of tubercular disease of Peyer's and the solitary glands in pulmonary phthisis, and the difficulty which exists in distinguishing acute tubercular inflammation and ulceration of the glands of the ileum, from the corresponding lesions of ordinary Enteric Fever.

Case 14.—A young woman, aged 17, "d'un embonpoint médiocre," had had cough for four weeks, and in consequence of afflicting intelligence suffered headache, loss of appetite and strength, thirst, increased heat, constipation; the headache was relieved by leeches, the other symptoms continued to the *eighth* day. She took some ipecacuanha, which produced bilious vomiting and purging, with pains in the hypogastrium; cough a little increased. *Ninth* day: sleepy; slight delirium at night; belly everywhere tender on pressure, supple; no tympanites; one stool; pulse 105; intense dry heat; some lenticular rose spots on the back and anterior and lateral parts of the chest; cough moderately frequent; oppression of the chest, mucous râles; in the evening tranquil but profound delirium. *Tenth* day:

partly recovered consciousness; tongue red and moist; abdomen tympanitic, a little tender on pressure; heat considerable; startings continue. *Thirteenth* day: pulse weak; mucous sputa, some streaked with blood; a little crepitation at base of right lung behind; continuation of the involuntary movements and meteorism; continued drowsiness during the day, and delirium at night. *Fourteenth* day: meteorism decreased, three or four involuntary stools; crepitant râles heard over the sides of the chest. *Fifteenth* to *nineteenth* day: profound drowsiness and delirium at night; on the evening of the *nineteenth* day respirations much embarrassed, 60. *Twentieth* day: delirium and ineffectual efforts to put the arms out of bed; death.

Necroscopy.—*Head*—brain and meninges apparently normal, only moderately injected. *Chest*—lungs free, filling the cavity of the chest, of a tender rose-color in front, a little engorged behind for some extent, strewn internally with a great number of gray semi-transparent granulations; bronchi injected, their last divisions covered with a puriform secretion. *Abdomen*—mucous membrane of the small intestine very soft, of an obscure red near the cæcum; Peyer's patches were only visible in the ileum; those nearest the jejunum were pale and obscure, the rest red and successively more developed, larger, and more thickened in proportion as they were nearer the ileo-cæcal valve; those in the last foot of the ileum were ulcerated, their mucous membrane more or less destroyed, and in some the muscular fibres, which were red and thickened, were discovered. The non-ulcerated plates were about a line in thickness. Between Peyer's patches were others much smaller, irregular, and otherwise resembling them, and some yellow, miliary granulations. The last two inches of the mucous membrane were entirely destroyed around nearly the whole of its circumference, and the submucous cellular tissue was more or less red and thickened. Large intestine: mucous membrane thickened and very soft, and presented a considerable number of grayish, lenticular spots, marked with a black point in their centre. All the mesenteric glands were red and livid, and those near the cæcum very large and soft. The mesocolic glands were in the same condition. The spleen was nearly double its volume. (Louis: Obs. xxxiv. p. 25, vol. ii.)

It is strange that Louis, of all other observers, should consider the foregoing to be a case of Typhoid Fever, when the history and anatomical characters are so plainly those of acute tuberculosis. The nature of the other case is still more apparent.

Case 15.—A spare man, aged 25, having short breath from the age of ten years, is taken with the following symptoms: Disgust of food, thirst, cough, shiverings followed by heat. These symptoms continued, with constipation, until the *twenty-first* day, when he was admitted into the hospital of La Charité. He presented the same symptoms with a slight oppression at the epigastrium; constipation still; heat of skin a little exalted, general moisture; pulse large and moderately full; cough infrequent; some mucous expectoration; natural respiratory murmur; moderate weakness. *Twenty-fifth* day: some lenticular rose spots on the abdomen and chest; pulse 86. Up to the *thirty-seventh* day the patient continued stationary, then for the first time he had spontaneous diarrhœa, and grew paler and weaker. *Thirty-eighth* day: a sudden and violent pain in right testicle and corresponding part of hypogastrium, accompanied by a little shivering. The pain came on again in the night, and his slender body was covered with large drops of sweat; he had neither nausea, nor vomiting, nor tympanites; pulse 104. *Thirty-ninth* day: copious diarrhœa; several vomitings of green bile; sweats and pain continued all day. These symptoms continued up to the *forty-fifth* day, when there was great abdominal pain and vomiting. He died this day.

Necroscopy.—Considerable emaciation. *Head*—slight sub-arachnoid effusion. *Chest*—the summit of the left lung presented some cellular adhesions, was a little hard and unequal, and offered for the depth of two inches a considerable number of gray, semi-transparent granulations, in the midst of which a tuberculous excavation was found, the size of a nut, partly empty, and communicating with the bronchi. Below, the pulmonary tissue was in the normal condition. The right lung was in the same condition. *Abdomen*—general peritonitis from perforation of the small intestine about five inches from the cæcum; red patches, due to injection of the peritoneum upon the external surface of the small intestine; internally, this part of the bowel presented, at about twenty-four inches from the cæcum, a transverse ulceration about ¾ths of an inch in extent, opposite the mesentery, having the attenuated muscular coat for its base, and the edges were not very prominent, and slightly grayish. Six similar ulcerations existed in the last six inches of the ileum, and in the centre of the first of them the perforation, measuring about a fourth of an inch in diameter, was found. The edges of this ulcer were very thin, and partly formed by the peritoneum alone.

The mesenteric glands were a little red, and three or four times their natural volume, and had only half their usual consistence; liver a little pale and soft;

spleen twice its natural volume, a little pale and soft.

"The thirst, anorexia, pains in the head, and the shiverings clearly indicate the commencement of the illness in this case. It was only after three weeks that the pains in the belly were experienced. Diarrhœa came on as late as the thirty-seventh day. The ulcerations of the intestine being the most profound and without doubt the oldest lesions observed, to these ought to be attributed, in great part at least, the febrile symptoms present at the commencement. It is requisite, however, to remark that the tuberculous affection commenced, according to all appearance, with the principal disease; yet as ulcerations of the small intestine had the characters of those which occur in the course of the typhoid affection, and as the state of the mesenteric glands[1] could only be referred to that condition, this disease has evidently had the greatest share in producing the symptoms and lesions observed, and we can only place this case in the chapter upon the latent typhoid affection." (Louis, Obs. xliii. p. 232, et seq.)

Endemic Intermittent and Remittent Fevers. — One of the most general facts observed in reference to Enteric Fever, is the frequent occurrence of intermittence in the pyrexial condition. The commissioners appointed to investigate the French epidemics of Enteric Fever, "call attention to the fact that a more or less pernicious intermittent or at least remittent character, was manifested under a great variety of circumstances." (De Claubry, Mém. de l'Acad. de Méd., tome xiv. p. 71.) "A great number of cases of Typhoid Fever presented, either at the commencement of the disease, transient symptoms of simple intermittent fever, or during its further progress, intermittent or at least remittent phenomena, which rendered the employment of quinine necessary." (Ibid. p. 11.) M. Trousseau (Clinique Médicale) records cases to show that "Enteric Fever may simulate at first intermittent fever, and reciprocally, an intermittent fever may assume at the commencement the characters of Typhoid Fever." (P. 247, 2d Edition.) "It is especially in countries where marsh intermittent fevers are endemic, and with individuals who have recently left their own country, that we see Enteric Fever assume at its commencement an intermittent type." (P. 250.)

Intermittent fever is, in the present day, nearly extinct in England. Its last strongholds are to be found in the north part of Kent. There, about the Isle of Sheppey, and on the marshy banks of the Swale, it still lingers. At Milton, for example, ague is still common in the autumn. "The drinking water is obtained from wells, and the general sanitary condition, as regards drainage and the non-removal of nuisances, is unsatisfactory, and there has been a considerable amount of Typhoid Fever at times, and scarlatina in a severe form was prevalent at the time of the inspection." (Rep. by Dr. G. Whitley, as to quantity of ague now prevailing in England. Sixth Rep. Privy Council, 1863, p. 432.)

At Holbeach and Long Sutton, Ague and Enteric Fever were both prevalent. The drainage of these places is bad, and the water supply bad, being from pits (p. 441). "Very nearly all the medical men who had had opportunities of forming an opinion concerning the co-existence of ague and typhoid fever in the same districts, were of opinion that the local conditions which produced the former are favorable to the development of the latter. Thus, Mr. Keddell, with forty years' experience in Sheppey, believed that when ague, from certain conditions of surface, is rife in summer, bilious, remittent, and typhoid fevers prevail in autumn." (Ibid. p. 452.)

My friend Mr. Charles Mayo informs me, from extensive personal observation, that the "camp fever" of the army of the Potomac was generally recognized as a "typho-malarious fever," in which the symptoms of typhoid fever, diarrhœa, rose rash, &c. were associated with those of intermittent fever. The typhoid symptoms occasionally predominated, and post-mortem examination revealed lesions of Peyer's glands. [See, on a subsequent page, a farther account of American Typho-malarial Fever.—H.]

It is evident from the foregoing observation that an investigation into the nature of Enteric Fever would be very incomplete without a brief consideration of the symptoms and morbid anatomy of the severer forms of intermittent fever. The Walcheren fever offers itself as a standard of comparison. Dr. Davis[1] has given a very clear and minute description of this disease. "The Walcheren fever," he says, "assumed the quotidian, tertian, double-tertian, and even remitting type. It did not uniformly declare itself with the same type, being one while continued, then remittent or intermittent, and changing its type again from these to the con-

[1] Louis appears to regard a vascular, swollen, and softened condition of the mesenteric glands as being inconsistent with the existence of tubercular disease. We have frequently seen the mesenteric glands purple, soft, and swollen in cases of advanced phthisis in which both lungs and intestinal glands have been affected. (See Diagnosis.)

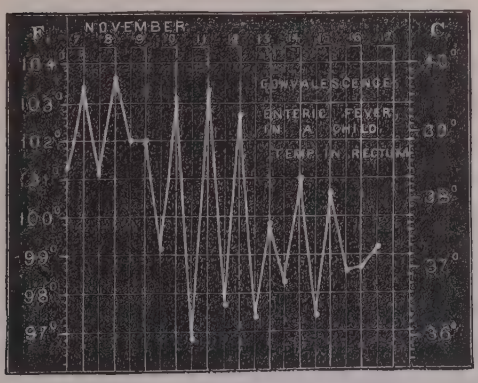
[1] View of the Fever of Walcheren and its consequences, by J. B. Davis, M.D. 8vo. Lond. 1810.

tinued character. I believe the Walcheren fever in many instances would have ceased but for the derangement it had occasioned in the abdominal viscera, becoming in some measure a secondary disease." (P. 12, et seq.) The *premonitory symptoms* were weakness, nausea, headache, universal languor, dejection of spirits, always combined with a vitiated state, suppression or diminution of the intestinal and biliary secretions.

After the paroxysms, headache, confused intellect for two or three days, ending in coma and stupor. At other times continued pyrexia, whiteness of the tongue, distension and uneasiness of the epigastric region, and anorexia. Then the bowels became painful, and there were diarrhœa, discharge of mucus, or much blood intermingled with feces, &c. &c. These symptoms would be obscured by the paroxysm, to reappear after it was over (p. 18). "All the patients with the quartan type under my care were very prone to diarrhœa and dysentery, thirst, pyrexia, emaciation, daily exacerbations of hectic, local pains, and general irritation, constituting an unmanageable disease which wore away the patient's strength, and utterly exhausted him" (p. 17). Delirium was seldom formidable; epistaxis frequently occurred. Hectic was almost uniformly the character of the pyrexia. Gray, clay-colored watery stools, and rapid marasmus, were common in cases tending to a fatal termination.

Such were the symptoms of "the continued fever or long-continued paroxysm,"

[Fig. 11.



Remitting lysis in enteric fever.]

when the order of the periods became so completely overturned that it was difficult to bring the fever to its proper type again (p. 21).

If we now turn to the morbid anatomy of the disease, we shall find positive evidence of lesion of the solitary and agminate glands of the small intestines, in at least six of the cases recorded by Dr. Davis. Usually both the small and large intestine were involved in the disease, and its ravages greatest in the latter. Thus, in case 29, we have a description of the post-mortem appearances of dysentery implicating the small intestine: — Colon ulcerated throughout its whole extent. Rectum much ulcerated and had sloughed near its termination. Jejunum and ileum "interspersed with black spots internally; the intervening spaces red, and raised up into little protuberances, resembling granulations of flesh, not unlike a cock's comb." Liver large, black, and soft. Spleen soft, of enormous size. Mesenteric glands enlarged (p. 173).

In case 32 the large and small intestines appear to have been pretty equally affected. "The small intestines were of a deep purple color, and interspersed on their inner side with tubercles and small ulcers, resembling chancres. The colon and rectum had numerous tubercles and ulcerations." Spleen weighed four pounds, and was uniformly soft throughout. Mesenteric glands enlarged (p. 175).

In case 34 the lesions were more apparent in the small than in the large intestine. The convolutions of the small intestines were united together by condensed coagulable lymph. They were of a reddish color, and interspersed with small red eminences on their inner surface. The coats of the colon were thickened. Liver large and black. Spleen firm and dark, weighed four pounds.

The particular characters of the intestinal lesions are thus clearly described by Dr. Davis. The ileum and jejunum were frequently interspersed with "tubercles inflamed and ulcerated in different parts. Here and there small eminences of the size of a pin's head, or round bodies with an ulcer at the point, or little ragged ulcers, excavated in the middle, resembling chancres, or one large, or a succession of small ulcers spreading wide upon, and deep into, the coats of the intestines. Color of these tubercles various, consistence firm. While their points were yellow their edges were hard, and their bases almost black, like a lump of decayed flesh. They did not come fairly to suppuration, but appeared gradually to crumble away and degenerate into a scabrous ulcer. These bodies had their origin beneath the villous coat of the intestine." (P. 191.)

From the above description we may infer that the morbid condition of the solid and agminate glands of the intestine in these fatal cases of intermittent fever is identical with that which is assumed to be characteristic of Enteric Fever. We have already seen that both forms of fever are developed amidst the same conditions, and

we therefore unhesitatingly conclude that Enteric Fever is often a part of intermittent fever, and the converse.

Dysentery.—After the foregoing observations it may appear superfluous to call special attention to the relation between Dysentery and Enteric Fever. But the connection between these two diseases is too important to receive only a cursory notice. Even in reference to Enteric Fever alone, it is important to observe that the ulceration sometimes spreads to the large intestines, when the lesions of the small intestine are in process of reparation. The following isolated case recorded by Forget, may be briefly mentioned to show how the enteric disease may be prolonged by subsequent lesion of the large intestine.

Case 16.—A patient was laid up with the usual symptoms of Enteric Fever for a month, then, after a few days' intermission, profuse dysenteric diarrhœa, tenesmus, and colic pains set in, and after continuing for about twenty days killed the patient. In the last two feet of the ileum, "numerous white shining spots, of variable extent, smaller than the ulcerated Peyer's patches, and evidently cicatrices, were found. The ileo-cæcal intestine was profoundly altered in all its extent from the great valve to the anus; it was brown, black, hypertrophied, vegetant, and softened, presenting ulcerations of various depth." (Forget, Obs. xlii. p. 351.)

Rokitansky describes "the typhous process in the mucous membrane of the small intestine," as distinct from "the dysenteric process" observed in the large intestine. But this distinction is purely artificial. In the following well-marked case of Enteric Fever which lately died under my care, the large intestine was the more extensively ulcerated, and the ulcers in both small and large intestine were indistinguishable from the so-called "dysenteric ulcers."

Case 17.—Catherine M., aged 23, residing at Stanmore, near London, was taken ill this autumn with headache, much shivering, pain in the back, and diarrhœa. Fever and diarrhœa continued; rose spots appeared on the abdomen from the *tenth* to the *twenty-second* day. Diarrhœa persisted, and there was much hectic. On the *twenty-fourth* day there was marked abdominal tenderness. The diarrhœa continued unchecked, and she died on the *twenty-seventh* day.

Necroscopy.—*Chest*—lungs congested, friable at apices, weighed thirty-three ounces. *Abdomen.*—liver enlarged, weighed three pounds five ounces, soft, greasy, and pale. Gall-bladder full of pale, thin, ochre-colored bile. Stomach, duodenum, jejunum, and upper portion of ileum healthy; last two feet of ileum presented sixteen ulcerations of Peyer's glands, varying from minute vascular abrasions to three-eighths of an inch in diameter. Four of these pale depressed ulcers were situated immediately above the ileo-cæcal valve, and were evidently in process of contraction and cicatrization. In the large intestine there were twenty-seven ulcers, twenty of which were in the cæcum; several were situated immediately below the ileo-cæcal valve, and one of these was as large as a shilling, and deeply excavated the muscular fibres; another, the size of a sixpence, was placed at the bottom of the cæcal pouch, and it lay upon the peritoneum, which presented externally a corresponding patch of opacity with vascular ramifications. Seven other ulcers occurred at intervals in the ascending and transverse colon, the last one occurring at a distance of two feet from the ileo-cæcal valve. All these ulcers were pale, with ashy or smooth bases lying upon the muscular fibres; their edges were not elevated, and often perpendicular. The solitary glands of the large intestine were enlarged, the central parts of many were eroded and in a state of incipient ulceration. The mesenteric and mesocolic glands were purple, much enlarged, and soft. The spleen weighed six and a half ounces, and was of normal color and consistence. Kidneys, pancreas, and the other organs healthy.

The day after this young woman died, a patient in the same ward, under the care of my colleague, Dr. Murchison, also died; and as the case illustrates very well how extensively the large intestine may be ulcerated by Enteric Fever, I have availed myself of Dr. Murchison's kindness in allowing me to make my observations of the case, and briefly detail them here.

Case 18.—Eliza H., aged 26, was admitted on the *tenth* day of her illness with fully developed Enteric Fever. She was taken ill with headache, heats and chills, and diarrhœa, and these symptoms continued to the time of her admission. Rose spots appeared on the abdomen from the *tenth* to the *eighteenth* day. The bowels continued very loose, and the stools were of a light yellow color. Medicines failed to restrain the diarrhœa, the abdomen became distended and tender, and the patient died exhausted on the *twenty-seventh* day of her illness.

Necroscopy.—*Chest*—lungs healthy, with only a little hypostatic congestion. Heart contained firm fibrinous clots in all its cavities. *Abdomen*—stomach, duodenum, jejunum, and upper portion of ileum perfectly healthy. In the last two feet of the ileum there were a dozen pale non-elevated ulcerations of Peyer's glands; six of them were in the immediate neighborhood of the ileo-cæcal valve, and the largest did not exceed three-eighths of an

inch in diameter. All were evidently in process of healing. The large intestine was in a state of ragged ulceration from the under surface of the ileo-cæcal valve to within an inch of the rectum. In the transverse and descending colon there were two rows of ulcers, each about a foot long; these ulcers were deeply excavated, and for the most part confluent, or only separated by narrow bands of hypertrophied mucous membrane. Each ulcer, or confluent patch, was about an inch wide. The edges were two or three lines thick, irregular and very vascular, and often black; the surface of the ulcer was chiefly formed of ashy sloughs of areolar tissue, or disintegrated muscular fibres. In the interval between these rows of ragged ulcers were a great many circular ulcers, and swollen solitary glands advancing to this condition. Nearer the cæcum and rectum the ulcers were fewer and more discrete. The mesocolic glands were greatly enlarged, purple, and soft. The spleen weighed eight and a half ounces, and was pulpy. The liver was very soft and greasy; it weighed forty-four and a half ounces. The gall-bladder contained half an ounce of pale yellow watery bile, which did not affect turmeric paper, but changed blue litmus to red. The other organs were quite healthy.

In the following case of Enteric Fever and pneumonia, the intestinal lesion was almost entirely confined to the colon:—

Case 19.—Phœbe Poole, aged 14, was admitted into the London Fever Hospital on the 8th of September, 1865, on the *fourteenth* day of her illness. She had had cough, quick breathing, and diarrhœa, accompanied by high fever. At this date the pulse was 150, tongue dry, brown, and cracked, skin pungently hot, respirations 58, cough, dulness with crepitation and bronchophony over the lower lobe of the right lung behind; there was retention of urine, the abdomen was tympanitic and tender, there were two rose spots upon its surface, and the bowels were very loose. The pulmonary and enteric inflammation progressed, tubular breathing was heard over almost the whole of the right side of the chest, and the bowels continued very loose. A few fresh rose spots appeared up to the *nineteenth* day. On the *twenty-ninth* day the respirations were 60, short and snatchy, the pulse 160, and the diarrhœa profuse. On the *thirty-fifth* day she died.

Autopsy.—Chest—right lung completely solid and firm, gray and gangrenous. Left lung a little engorged. Heart healthy. *Abdomen*—the last Peyer's patch near the ileo-cæcal valve had two minute ulcerations, but the rest of the gland, and all the other agminated and solitary glands, were perfectly healthy. The colic side of the cæcum, and the first five inches of the ascending colon, were in a state of ragged ulceration—long clean, transverse ulcers, laying bare and dissecting the muscular fibres, were repeatedly confluent in this part of the bowel, and were interspersed with islands of soft, greatly-swollen, mucous membrane. Lower down, were ulcerated solitary glands, and there were six more in the sigmoid flexure. The solitary glands and the mucous membrane of the rest of the large intestine were generally healthy. The mesenteric glands corresponding to the small intestine were quite healthy; the mesocolic glands in the neighborhood of the cæcum were purple, soft, and much enlarged. The rest of the viscera appeared healthy. The spleen was of natural size.

Such cases as the foregoing afford typical examples of acute dysenteric ulceration, and we may question whether the distinction between Dysentery and Enteric Fever is not somewhat artificial.

Cholera.—"In the delta of the Ganges, the Nile, and the Mississippi the three forms of disease called cholera, plague, and yellow fever, are constantly seen preceding, accompanying, and following intermittent fever, and constitute there the reigning endemic diseases; and one is forced to recognize a very great analogy, not to say an identity, of origin between marsh fever and the three great scourges above mentioned." (Traité des Fièvres intermittentes, rémittentes, et continues, par J. C. M. Boudin, p. 161.) A protracted attack of cholera bears a close resemblance to Enteric Fever; the intestinal lesions of the two diseases, moreover, are indistinguishable from each other. "The most frequent of all the abnormal conditions of the mucous membrane of the intestines was prominence of the intestinal glands, both aggregate and solitary, but especially the latter. This condition, the *psorentérie* of some French writers, was found in about two-thirds of the eighty-nine fatal cases examined." (W. T. Gairdner, M.D., Month. Journ. Med. Science, 1849.) M. Pirogoff examined 500 fatal cases of cholera. He observed, in the earlier periods of the disease, "thickening and swelling of the mucous membrane most often accompanied by swelling of Peyer's and the solitary glands, as well as swelling of the mesenteric glands. In the typhoid period, ulceration of these glands." (Anatomie Pathologique du Cholera Morbus. Folio, St. Petersburg, 1849.)

Scurvy is sometimes accompanied by ulceration of the solitary and agminate glands, with all the symptoms of Enteric Fever. An outbreak of scurvy occurred in the Milbank Penitentiary, in London, in the years 1822–23. An account of it was published by P. M. Latham, M.D. (8vo. Lond. 1825.) "In addition to the

ordinary symptoms of scurvy—purpura hæmorrhagica, spongy and even bleeding gums, &c.—there was every degree and species of flux ever seen or described. There were cases which corresponded with the descriptions of the Indian cholera, and there were some which corresponded with the common autumnal cholera of this country, except that they were accompanied by intractable diarrhœa. There was every kind and degree of dysentery." (P. 32, 33.) In some cases the abdomen was soft and natural; in others tympanitic. Post-mortem examinations revealed lesions of the intestines, which, from the descriptions at pp. 46–49, are clearly to be attributed to swelling and ulceration, even to perforation, of the solitary and agminated glands.

Varieties.—After the foregoing review of the associated pathology of Enteric Fever, can we adopt the dogma of Chomel?—"Quand nous trouverons dans les auteurs, soit anciens, soit modernes, des observations de maladies aigues à la suite desquelles on aura rencontre des ulceres à la fin de l'intestine grêle, nous aurons le droit de les considérer comme des cas d'affection typhoïde." (Fièvre Typhoïde, p. 113.) Or, going to the other extreme, shall we deny the existence of Enteric Fever as a specific disease, and regard the intestinal lesions merely as the result of an accidental but severe local complication which may arise in any general febrile condition of the body? If we accept the first proposition, we must include Tuberculosis under Enteric Fever. If we adopt the second, we may, with almost equal reason, deny the existence of scarlatina as a distinct disease. We can only avoid the dilemma by admitting that the enteric disease, and all its attendant phenomena, may occasionally become a part of some other more general inflammatory condition, and then, I think, it can hardly be denied that in other cases the disease is due to some poison or poisons introduced from without, in the elimination of which, the digestive organs especially are deranged. In order to include all the phenomena of Enteric Fever, I find it necessary to divide it into these three varieties: (1) Simple Inflammatory Enteric Fever; (2) Contagious Enteric Fever; (3) Paludal Enteric Fever.

It may seem paradoxical and unphilosophical to include under one kind contagious and non-contagious diseases; but since the question of contagion is still an open one with many, and neither symptoms nor anatomical lesions mark a distinction, we must be content thus to classify the disease for the present.

1. *Simple Inflammatory Enteric Fever.*—This variety is non-contagious, due to no specific cause, and may arise in any inflammatory condition of the body, such as accompanies pneumonia, erysipelas, pyæmia, &c. The common enteritis, which constitutes autumnal diarrhœa, if protracted, often lapses into this variety of Enteric Fever. Cases 6, 7, 11, 19, &c., furnish examples of this variety. The recognition of the intercurrence of enteric inflammation, with ulceration of the glands of the ileum, in acute diseases generally, is of very great importance; for of all the organs of the body, these delicate glands, from their situation within the thin and vascular intestinal wall, are less capable than similarly constituted parts elsewhere situated, of enduring prolonged inflammation, without risk of fatal accidents; and at any time the intestinal lesion may become much the gravest part of the more general disorder. The frequency with which the intestinal glands become implicated in acute disease is probably due to their exposed situation, their delicate corpuscular structure, their great vascularity, and the arrangement of their bloodvessels.

A well-developed healthy lad, aged 15, fell from a horse; the skull was fractured and the corresponding surface of the brain lacerated; febrile action followed, and he died on the third day. Before the body was cold I examined the small intestine. In the last nine inches of the ileum I found the solitary glands swollen, and of a delicate grayish-pink color, and semi-transparent appearance, forming rounded elevations of the mucous membrane, the size of hemp-seeds (psorentery). The mesenteric glands were a little increased in vascularity and size. There was no trace of disease in any part of the body. Here we recognize a condition of the solitary glands, which, under the continuance of the general febrile action, *might* have passed into the worst form of "typhoid ulceration."

"But surely," it will be said, "we can distinguish the true typhoid ulceration from any other at a glance; moreover, the typhoid ulcer is characterized by the deposit of a distinct morbid material—a specific *typhous cell.*" Having shown that the inflammatory swelling is due, not to the deposit of a specific morbid matter within the glands, but to the rapid growth of their normal corpuscular constituents under the influence of undue vascular excitement, we fail to recognize any character by which one form of inflammatory action in Peyer's glands can be distinguished from any other. Ultimately, we shall have little or no difficulty in distinguishing a tubercular ulcer from any other; but between the lesions assumed to be characteristic of Enteric Fever, and those arising from ordinary inflammation, which, of course, may affect the intestinal glands

in common with every other part and organ of the body, there is, I conceive, no distinction. As a result of common inflammatory action in the glands, we may find Peyer's glands swollen into cock's-comb or fungus-like elevations, and excavated into ragged ulcers, with red everted edges, or occupied by sloughy cores. (See case 8.) In a well-marked case of Enteric Fever, in which the diarrhœa, rose spots, and abdominal pain and swelling call attention to the abdominal lesion, we may, on the other hand, find, as in a case which I examined two days ago, a dozen angry-looking ulcers in the last foot of the ileum, varying in diameter from two lines to three-fourths of an inch, and exposing the red-streaked muscular fibres, each ulcer sharply cut, and the irregular margins *not* raised above the general level of the intervening dark-red mucous membrane. Higher up were seen glands level with the inflamed mucous membrane, and presenting sloughy erosions, like an aphthous ulcer of the mouth.

2. *Contagious Enteric Fever.*—Of this variety I can say but little. I am not *sure* that I have seen it; but that it exists, appears to be an indisputable fact. Eight of the sixty-eight patients referred to below came from houses in which other residents were affected with the fever. The disease may have been propagated by contagion in some of these cases, but in two instances, in each of which three members of the same family were affected, I found from personal observation that an endemic cause existed in impure drinking water. See also cases 4 and 5, and the observation upon "Contagion." The associations of this variety appear to be, as I have already pointed out, with scarlatina and the allied affections, diphtheria and croup. Exudations upon the faucial and laryngeal mucous membrane appear to be frequent in this variety, and the course of the disease more rapid than in the third variety.

3. *Paludal Enteric Fever.*—This we believe to be the common form of the disease. It arises from putrescent animal and vegetable substances. It is non-contagious, and its course is usually slow. Case 2 may be taken to illustrate this variety of the disease.

In thus unreservedly recognizing the connection which I cannot doubt exists between Intermittent and Enteric Fever, it may appear to some that I transgress the facts which have been adduced to illustrate this view; but I feel sure that justice has not been done to the numerous observations which abound in medical literature, and which, if collected, would together form irresistible evidence of the direct connection between these two diseases. In the low-lying districts on the banks of the Thames, within and about the metropolis, where ague was formerly so rife, Enteric Fever prevails continuously, becoming very abundant in the autumn, while the higher situations are comparatively free from it. Of sixty-eight cases of well-developed Enteric Fever which have come under my care during the present autumn (1865), fourteen came from the districts of Stanmore, Chelsea, Lambeth, Southwark, Stepney, Hackney, Bethnal Green; thirteen from the lowest part of the parish of St. Luke alone, where cesspools and pumps are still in use, and where drainage works are now in progress; twenty-three from the filthiest and most crowded parts of the parishes of St. Clement Danes, Holborn, and St. Giles; and only five from the more elevated localities of Soho and Marylebone, Islington, Holloway, &c. The remainder resided in Maidstone, Croydon, Mitcham, Edmonton, and various other country districts near London.

We find Enteric Fever remarkably prevalent in the spreading outskirts of the suburbs, where new houses and streets are constantly springing up beyond the limits of the drainage works.

We acknowledge as modifications of the same disease, that intermittent form to which, when London had its cesspools and pumps, and retained all its filth within its undrained area, James I. fell a victim, and that continued modification which still lingers in a subdued form in the same locality, and to which a good Prince has succumbed in our own generation.

[*Typho-Malarial Fever.*—In the States which were the seat of conflict during the American civil war, and especially in Virginia, in 1862, many cases occurred amongst the soldiers of the Union army, for which the above is the most fitting name. "Chickahominy Fever" was a hospital designation, derived from a locality in which hundreds of cases took their origin during the "peninsular" campaign of McClellan.[1]

Three morbific elements appeared to combine in the causation of these cases: malaria, camp or "crowd" poison, and the dietetic deficiency which produces scurvy, and gives the scorbutic taint to other diseases. According to the predominance of one or another of these etiological elements, the resulting malady varied. The following brief account is cited from a record taken by me upon the observation of a large number of these cases in two of the Philadelphia Hospitals.

"Of the form in which the malarial element prevailed, the somewhat abrupt commencement, gastric disturbance, and icteroid skin and tongue, with remissions tolerably distinct, were predominant fea-

[1] See Woodward's "Camp Diseases of the United States Army."

tures. The lenticular spots of typhoid fever, and the sudamina and tympanites, were often wanting altogether.

"A slower onset, less distinct remissions, more cerebral disturbance, and diarrhœa, with epistaxis and bronchitis sometimes, but with both less constantly than in civil life, marked the predominance of the typhoid pathogenetic element. Deafness, under my observation, was less frequent than in civil life, but was sometimes very well marked. The aspect of the countenance, and the character of the somnolence and delirium, were precisely the same as in ordinary typhoid fever.

"The scorbutic complication was recognizable, in the third group of cases, by the peculiar mental and bodily prostration which preceded and followed the disease —the remarkable irritability of the heart, the state of the gums, tendency to hemorrhage, discolorations and petechiæ, pallid, large and smooth tongue, and extremely protracted convalescence.

"*Morbid Anatomy.* — Most important was the intestinal lesion, similar to that of typhoid or 'enteric' fever, though not identical. The following account of this is from Dr. Woodward.[1]

"'In the earlier stages there was little to distinguish the intestinal lesion from the corresponding process of ordinary enteric fever, except, perhaps, the great tendency to the deposit of black pigment in the enlarged follicles. In the latter stages certain peculiarities are often distinctive enough to enable the anatomist to recognize typho-malarial fever by the post-mortem appearances alone. The tumefaction in typho-malarial fever rises very gradually from the surrounding mucous membrane, and attains a moderate degree of thickness (three to six lines) on the edges of the ulcer. In this it differs materially from the ordinary typhoid ulcer, in which the enlarged patch rises abruptly from the mucous membrane in such a way that the summit is often larger than the constricted base, giving rise to the comparison made by Rokitansky, who likens the shape of the tumefaction to that of flat sessile fungi. The umbilicated depression, so frequent in the ordinary typhoid patches prior to ulceration, has never been observed in typho-malarial fever. The ulcer itself presents ragged, irregular edges, which are often extensively undermined in consequence of the erosion extending more widely in the submucous connective tissue than in the glandular tissue of the mucous membrane. This characteristic undermining of the edges is much more extensive in these than in ordinary typhous ulcers.'

"*Pathology.*—Doubting not at all the presence of the malarial element, the question occurs, was the modifying 'febrile' cause of the *typhous* or of the *typhoid* character? Granting, that is, that these are pathogenetically distinct, we should expect that the typhus or 'crowd-poison' element must result from the circumstances, as from those which made typhus or 'camp fever' the scourge of armies in Europe. Only, against this, we have the local lesion, of the glands of Peyer and mucous membrane of the bowels, recalling enteric or typhoid fever.

"But—as, where typho-malarial fever occurred, causes of intestinal irritation (bad water, deficient food, &c.) were present—I am not satisfied that such an appearance (not, as we have seen, identical with that of typhoid fever) should exclude the idea of the zymotic action being that of the *typhous* cause. In that opinion, as a probability, not, of course, now demonstrable, I rest.

"*Treatment*—From the above view of the hybrid and threefold nature of the disease, came its rational treatment. More *quinine* than in typhus, more *alcohol* than in remittent, more *fresh vegetable food* and fruit than in either. Experience justified this plan. In our hospitals in Philadelphia, few died from fever who were not moribund on their arrival from the seat of war."[1]—H.]

DISTRIBUTION. — Enteric Fever prevails in every inhabited part of the world. No situation is secure from it. In the report of the epidemics which have occurred in France from 1841 to 1846, De Claubry (Mém. de l'Académie de Méd., tome xiv.) observes: "The situation of the twenty-eight departments which have been the frequent theatre of destructive epidemics of typhoid fever, was such that it appeared impossible to conclude that it had any influence whatever in the production of these epidemics." (P. 4.) "If one finds typhoid fever on the one hand in villages situated in deep valleys, in narrow gorges; in lowlands, where the water-courses frequently overflow, making the submerged soil, upon which the miserable dwellings are built, extremely damp: one sees it, on the other hand, in villages situated, one upon the most elevated points of a high chain of mountains, and constantly exposed to every wind, and having no unhealthy condition in its neighborhood; another situated 600 feet above a little flowing stream, commanding an extensive view of perfectly cultivated fields; a third, in a very salubrious position, upon an undulating soil, where the flow of water is perfect." (P. 8.)

Enteric Fever, moreover, attacks every class of society indifferently. On one

[1] Op. citat., pp. 102–3.

[1] Essentials of Practical Medicine, 4th ed., pp. 367–9.]

hand, we find it associated with the most abject poverty, damp, filth, overcrowding, and defective ventilation; and, on the other, we witness the disease making havoc amongst the wealthy residents of spacious, dry, well-built houses, isolated, or united to form wide open streets, or elevated terraces.

CAUSES: (*a*) *Predisposing.* — Of the causes which predispose to Enteric Fever, *youth* is usually considered to be one; but young people are not more liable to this than they are to other inflammatory diseases. Dr. Murchison states (page 409), that slightly more than half of the cases of Enteric Fever admitted during ten years into the London Fever Hospital were between fifteen and twenty-five years of age; one-fifth were under fifteen; less than one-seventh above thirty: and only one-sixty-eighth exceeded fifty. Similar statistical results may probably be found in many other acute diseases. [More than one attack in the same individual is rare; but instances of recurrence do occur.—H.]

Seasons have a marked influence on the increase and diminution of Enteric Fever. "Out of 106 times in which an exact indication of the epoch when the epidemics of typhoid fever commenced, the reports of the years 1841 to 1846 give the following results:—First yearly quarter, twenty epidemics; second quarter, twenty-one; third quarter, twenty-nine; fourth, thirty-six: or, summer (April to September) sixty; winter (October to March) fifty-six. Seventy epidemics commenced in the four months of August, September, October, and November; while only forty-six commenced in the other eight months of the year, from December to July." (De Claubry, op. cit. p. 8.)

"In New England Enteric Fever is not infrequently called the autumnal or fall fever." (Bartlett on Fevers, p. 101.)

On examining the accompanying Table (*vide* Table) of the cases which have occurred at the London Fever Hospital during the last 18 years, the following facts appear:—*First*, that the greater number of cases occur during the autumn and winter months, and the average of 17 complete years[1] shows that more than twice as many cases, or a proportion of 2·1 to 1, occur during these periods, as compared with those happening during the other six months of the year. *Second*, that of all the seasons autumn is the one in which Enteric Fever is most prevalent. In fourteen out of the seventeen years, the number of autumnal cases exceeded that of any other season. In one of the three remaining years, 1851, an equal number of cases occurred in the summer and autumn respectively. In the other two years, 1860 and 1862, the greatest number of cases occurred during the winter, exceeding the autumnal cases by sixteen. *Third*, that the disease is least of all prevalent in the spring. Excepting the years 1852, '56, '59, '60, '61, and '63, the least number of cases occurred in spring, and in all these exceptional years there were only thirty-four spring cases in excess of the summer ones.

It appears from these general facts, and from a little closer examination of the table, that Enteric Fever obtains its maximum development in the months of September, October, and November, declines slowly during the winter and spring, and reaching its minimum in May, then begins to increase progressively with the advance of summer.

Let us now go a step further, and endeavor to find out the conditions which, prevailing most in autumn, render this season most favorable to the existence of Enteric Fever.

Temperature.—In every year but 1852, the combined temperature of the autumn and winter was less than the combined temperature of spring and summer, and in this exceptional year the mean temperatures were as 50°·3 to 50°·9. And in every year but 1851, '54, '55, '56, and '61, the temperature of autumn was less than that of summer, and in no year did the autumnal temperature exceed that of summer more than 1°·1. Again, if we except the years 1851, '55, '56, and '61, October—the month when Enteric Fever is most rife—was cooler than May, when the disease is at its lowest ebb. It has been already stated that in five of the years, the number of cases occurring in the spring of those years was in excess of the number which happened in the warmer summers.

Apart from any other cause, it cannot, therefore, be concluded that temperature has any influence on the increase of Enteric Fever.

Rainfall.—In considering the influence of rain upon the quantity of Enteric Fever, attention must be given, not so much to the total yearly amount, as to the quantity which falls in each month. The average amount of rain for each of the seventeen years is 23·1 inches. If only one inch fall during a period of two months, that must be regarded as a season of drought.

Of these seventeen years the most rain, 34·4 inches, fell in 1852, and in this year there were 140 cases of Enteric Fever. Next stands the year 1860, when there was a rainfall of thirty-two inches, and only ninety-five cases—a total considerably smaller than that of any other year. But on further comparison we find that in 1860 the rain was not only abundant,

[1] From 1848 to 1864 inclusive.

TABLE *showing the number of cases of* ENTERIC FEVER *treated in the* LONDON FEVER HOSPITAL, *and certain attendant atmospheric conditions, during the last eighteen years. The analytical observations have no reference to* 1865, *which year was incomplete when they were made*

The *first column* gives the number of cases admitted during every month, season, and year. It is partly derived from p. 17 of Dr. Murchison's work, and partly from the London Fever Hospital Reports. The *second column* gives the mean temperature of the air during each month, season, and year. The *third column* shows in like manner the mean degree of moisture contained in the air. The numbers have reference to 100, which is taken to represent the greatest quantity of aqueous vapor which the air can at any temperature retain. The *fourth column* gives the total rainfall in each month, season, and year. The second, third, and fourth columns are from the Registrar-General's Returns, and are due to Mr. James Glaisher, F.R.S

	1848.				1849.				1850.				1851.				1852.				1853.				1854.				1855.				1856.			
	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.
January....	9	34°6	83	1·2	9	40°1	88	1·6	6	33°7	89	1·2	13	42°9	85	2·7	10	42°0	84	3·6	17	42°4	80	2·0	13	39°0	91	1·7	16	34°9	91	1·0	12	39°4	91	2·6
February ..	9	43·4	86	2·6	7	43·2	86	2·2	5	44·7	83	1·3	8	40·1	87	1·2	12	40·8	87	0·9	7	33·3	80	0·9	13	39·5	84	1·0	9	20·4	91	1·4	10	42·0	87	1·1
March	7	43·8	83	3·1	5	42·5	80	0·5	5	39·9	77	0·3	12	42·6	84	4·1	6	41·3	81	0·2	14	38·5	78	1·5	7	43·8	79	0·4	5	37·9	86	1·5	8	38·7	82	1·1
April	4	Mean temp. & hum. of suc. qr. & total rain			3	43·2	86	2·2	7	48·5	79	2·3	8	44·7	82	2·3	7	45·9	75	0·5	5	45·2	77	3·1	6	48·4	77	0·6	8	45·8	78	0·1	8	46·8	74	2·3
May	4	54·3	81	7·3	4	54·0	70	3·9	14	51·3	76	2·4	16	50·9	76	0·8	9	51·5	77	1·9	6	52·0	74	1·6	8	50·9	85	3·3	10	48·8	80	1·8	7	49·5	77	3·5
June.....	13	..	..	..	12	57·9	71	0·2	11	60·8	70	0·9	24	58·9	73	1·3	9	56·1	78	4·6	17	58·2	70	2·8	10	55·7	82	1·0	7	56·9	74	0·7	7	58·5	79	1·6
July.....	16	..	..	..	16	62·1	71	2·9	15	62·2	88	2·9	29	60·1	77	4·2	4	66·6	73	2·3	11	60·3	76	6·0	15	60·3	78	1·7	25	62·1	80	5·0	7	61·1	79	0·9
August.....	17	58·6	78	8·7	16	62·9	77	0·45	13	60·2	80	1·9	18	62·3	76	2·6	22	62·1	72	4·5	33	60·0	77	2·2	20	60·9	77	2·9	40	62·1	76	1·1	28	63·6	78	2·4
September .	26	..	..	..	19	58·8	77	3·3	13	56·4	75	1·3	27	56·9	76	0·5	19	56·8	77	3·9	33	55·3	84	2·4	49	58·1	77	0·7	26	57·1	80	1·1	14	55·2	75	2·8
October	17	..	..	..	25	51·1	81	2·7	17	47·0	82	1·4	24	52·6	81	1·8	12	47·9	80	3·8	29	50·9	90	4·3	51	49·4	84	2·6	25	51·2	89	4·9	15	51·7	87	1·6
November..	19	45·9	88	7·3	16	44·1	86	1·5	17	46·5	85	2·5	30	37·9	82	0·6	12	48·9	82	6·0	26	42·1	93	1·5	20	40·5	91	1·4	22	41·3	92	1·3	28	40·7	88	1·0
December..	11	..	..	..	6	39·1	90	2·4	14	40·6	92	1·3	25	40·5	69	0·6	18	47·6	79	2·2	13	34·0	91	0·7	16	41·3	87	1·4	24	35·6	79	1·2	5	40·2	90	1·3
(*a*) Spring..	20	..	..	..	15	42·9	84	4·9	17	44·3	79	3·9	28	42·5	84	7·6	25	42·7	81	1·0	26	39·0	78	5·5	26	43·9	80	2·0	22	37·7	85	3·0	26	42 5	81	4·5
(*b*) Summer	33	..	..	..	32	58·0	70	7·0	40	58·1	78	6·2	69	56·6	75	6·3	22	58·0	76	8·8	34	57·0	73	10·4	33	55·6	81	6·0	42	55·9	78	7·5	21	56·3	78	6·0
(*c*) Autumn	60	..	..	..	60	57·6	78	6·4	43	54·5	79	4·6	69	57·2	77	4·9	53	55·6	76	12·2	95	55·4	84	8·9	120	56·1	79	6·2	91	56·8	81	7·1	57	56·8	80	6·8
(*d*) Winter .	39	..	..	..	31	41·1	88	5·5	37	40·2	88	5·0	68	40·4	79	3·9	40	46·2	82	11·8	56	39·5	88	4·2	49	40·2	89	4·5	62	33·9	87	3·5	45	40·1	89	4·9
Total ..	152	..	..	23·3	138	49·9	80	23·8	137	49·2	81	19·7	234	49·2	79	22·7	140	50·6	79	34·4	211	47·7	81	29·0	228	48·9	82	18·7	217	46·0	82	21·1	149	48·9	82	22·2

TABLE *showing the number of cases of* ENTERIC FEVER *treated in the* LONDON FEVER HOSPITAL, *continued:—*

	1857.				1858.				1859.				1860.				1861.				1862.				1863.				1864.				1865.			
	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.	Cases.	Temperature.	Moisture.	Rain.
January....	8	36°6	92	2·6	32	37°5	86	0 7	13	40°4	88	0·8	14	39°7	88	1·8	8	33°9	85	0·5	17	39°0	85	1·9	20	41°8	85	2·6	6	36°5	82	0·9	14	36°3	89	3·3
February ..	5	39·2	87	0·2	7	34·6	84	1·7	10	43·1	81	0·9	6	35·7	80	1·1	8	42·1	91	1·8	6	41·1	84	0·5	21	42·1	86	0·6	13	36·0	88	0·8	18	36·6	83	1·9
March.....	8	41·8	84	0·8	13	41·4	78	0·9	12	46·4	79	1·3	11	41·1	79	1·9	6	43·8	90	2·2	17	43 1	86	3·7	15	43·9	78	0·7	9	41·3	83	2·7	3	36·6	82	0·9
April.....	4	45·7	82	1·4	5	46·2	76	2·4	5	46·6	78	2·2	7	42·9	79	1 0	3	44·3	85	0·8	4	48·4	81	2·8	4	49·1	78	0·4	14	48·2	74	0·7	6	52·3	73	0·4
May.....	1	54·0	74	0·6	9	51·7	75	1·8	5	53·1	77	2·4	4	53·8	75	3·9	1	51·9	74	1·8	5	55·4	84	2·8	9	52·0	78	1·3	11	53·8	73	1·9	5	56·1	75	4·4
June.....	9	61·8	72	2·7	7	64·9	67	1·2	8	61·4	77	1·4	2	54·8	82	5·8	2	59·1	81	1·9	21	56·3	77	1·8	8	58·1	75	3·9	12	57·4	72	0·9	27	60·2	70	2·4
July	19	64·5	71	1·1	13	60·6	72	2·9	12	68·1	70	3·3	7	57·6	83	2·8	10	60·9	78	2·2	21	59·1	78	1·7	14	60·8	72	0·9	18	61·8	70	0·3	26	63·8	72	2·3
August	26	65·8	73	2·6	29	62·0	70	1·6	20	63·5	72	1·1	10	57·7	83	3·7	18	63·2	76	0·6	14	59·5	81	3·0	12	61·9	74	1·8	51	59·6	65	1·4	34	59·9	80	4·0
September .	34	59·7	86	3·4	22	60·3	78	0·9	20	56·7	75	3·8	9	53·4	88	3·1	31	57·1	78	1·5	16	57·7	83	1·6	22	53·7	77	3·2	42	56·9	77	2·8	52	63·9	76	0·2
October.....	38	52·9	92	4·2	20	50·8	85	1·2	33	50·9	89	3·6	6	50·6	89	1·6	30	54·9	87	0·9	31	51·8	89	4·0	24	51·6	87	1·7	39	50·5	78	1·1	85	51·4	87	5·9
November..	33	45·9	94	1·3	15	39·6	86	0 4	27	42·1	87	2·9	6	40·8	93	2·5	31	40·8	87	5·2	34	39·8	92	1·0	17	45·7	88	1·8	24	42·0	86	2·6	128	45·0	88	2·3
December..	29	45·9	90	0·5	8	41·0	89	1·5	11	36·8	88	2·2	13	36·3	92	2·8	13	41·0	87	1·3	18	43·6	88	1·6	9	43·2	83	1·1	10	38·5	86	0·6	106	43·5	90	0·6
(*a*) Spring .	17	42·2	84	2·4	25	40·7	70	5·0	27	45·9	79	4·4	24	39·9	79	4·0	17	43·4	88	4·8	27	44·2	83	7·0	46	45·0	80	1·7	36	1·8	81	4·2	27	41·8	79	3·2
(*b*) Summer	29	60·1	72	4·4	29	59·1	71	5·9	25	60·9	74	7·1	13	55·4	80	12·5	13	57·3	77	5·9	47	56·9	79	6·3	31	56·9	75	6·1	41	57·6	71	2·1	58	60·0	71	9·1
(*c*) Autumn	98	59·4	83	10·2	71	57·7	77	3·7	73	57·0	78	8·5	25	53·9	86	8·4	79	58·4	80	3·0	61	56·3	84	8·6	58	55·7	79	6·7	132	55·6	73	5·3	171	61·7	81	10·1
(*d*) Winter.	70	42·5	93	4·4	55	39·3	87	2·6	51	39·8	87	5·9	33	38·9	91	7·1	52	38·5	86	7·0	69	40·8	88	4·5	46	43·5	85	5·5	40	39·0	84	4·1	266	41·6	89	6·2
Total...	214	51·0	83	21·4	180	49·2	78	17·2	176	50·2	77	25·9	95	47·0	84	32·0	161	49·4	82	20·7	204	49·5	83	26·4	175	50·2	82	20·0	249	48·5	77	15·7	522	51·2	80	28·6

(*a*) Spring—February, March, April. (*b*) Summer—May, June, July. (*c*) Autumn—August, September, October. (*d*) Winter—November, December, January.

but that each month had a due share, while in 1852 the still more abundant rain was unequally distributed throughout the year, the spring and early part of summer being unusually dry. If now, on the other hand, we regard the influence of drought, we find that the largest number of cases, 249, occurred in the driest year, 1864, when only 15·7 inches of rain fell. The next driest year was 1858; there were only 17·2 inches of rain in this year, and one-third of it fell in the summer months; the winter was the season of drought, and this was associated with an unusual increase of Enteric Fever in January.

With the same amount of rain in the years 1850 and 1854, there is a difference of ninety-one cases of fever, and this may be attributed to the inequality of the distribution of rain throughout the year, which may possibly be greater than is indicated in the Table. Thus, for example, the Table is not sufficiently detailed to show that, in 1854, the inch of rain for June did not fall on the first day of that month, and the 1·7 inch on the last day of July, leaving a long interval of drought between—as may have been the case. Mr. Glaisher's laborious and valuable observations are deserving of more detailed study in the elucidation of these questions.

The dry winters of 1851, '53, '55, '58, and '62, were associated with an increase of Enteric Fever.

The opposite effects of drought and rain have been well illustrated during the present summer (1865). Towards the end of July there was a great want of rain, but from the 30th of this month and throughout August there was an unusual amount, larger quantities having fallen almost every day. As shown by the admissions into the London Fever Hospital, Enteric Fever was very prevalent during the dry season, but after a fortnight's heavy rain its further progress received a sudden check, which continued until the effects of the succeeding drought became manifest.

It appears clearly from the foregoing observations that the absence of rain furnishes conditions most favorable to the increase of Enteric Fever; and since drought is necessarily associated with dryness of the air and exalted temperature, we must consider it, thus combined, as the one predisposing cause of Enteric Fever.

Other Atmospheric Conditions.—Too little is at present known respecting the influence of ozone in the production of disease; but as this body has been observed to be absent, or nearly so, from the air during the prevalence of cholera and other intestinal affections, the following general statements made by Dr. Moffatt (Chemical News, September, 1861) may be borne in mind. The quantity of ozone varies according to the time of year, the direction of the wind, temperature, atmospheric pressure, and the pressure of decomposing substances. Rain, a south wind, fall of the barometer, and increase of temperature, separately or combined, are associated with an increase of ozone, and the reverse conditions with its decrease. "Ozone periods terminate with increasing barometer readings, decrease of temperature, and wind from N. points of the compass." Ozone is most abundant in January, February, and March; less so in April, May, and June; and least of all in July, August, and September. "The greatest number of ozone days is in April, and the smallest in August and November. Whatever tends to a deflection in the direction of the wind leads to a corresponding result in ozone observations; and a town, chemical works, drains and cesspools, &c. deozonize the air, or wind passing over them" (p. 167).

Change of Residence, &c.—Both Louis and Chomel have observed that the greater number of the patients who came under their treatment in Paris had resided there only a short time. But change of residence, apart from the excitement and fatigue, the irregularity of living, and the distress which very commonly attends it, can hardly be considered a predisposing cause of Enteric Fever. In the autumn of 1861 a case of Enteric Fever which terminated fatally came under my care in Paris. The patient, a robust, newly-married lady, had been resident there only a few weeks, but during the whole of this time she had voluntarily lived a life of daily excitement and fatigue; the digestive functions were deranged by an unusual diet and irregular mode of living, and to these causes the disease was probably attributable.

M. Chomel found that one-third of the 115 cases to which special inquiry was directed had been exposed to sudden cold, to want of food or to bad diet, to excessive fatigue, to mental depression, and to debility produced by other diseases.

In his account of the outbreak of Enteric Fever in a garrison of 306 soldiers, Dr. Grossheim says: "It is difficult, if not impossible, to ascribe any deleterious influence to the food—all shared alike." He attributes the disease to the effect of military exercises in a changeable season, with night bivouac in the open air. (Edin. Med. Jour., vol. xlviii.)

(*b*) *Exciting Causes.—Contagion* is supposed by MM. Leuret, Bretonneau, Gendron, Dr. William Budd, and other physicians, to be the means whereby Enteric Fever is propagated. The following examples of the spread of the disease furnish the strongest proof that can perhaps be adduced in support of this view:—

Five persons were successively attacked with Enteric Fever in a certain house in Geneva. A sixth inhabitant of that town spent two nights with the third patient, soon contracted Enteric Fever, and died of it in the hospital. "At the autopsy, ulcerations of the ileum, and all the other lesions characteristic of dothinentery, were found." A clergyman who visited the third patient—a little girl—took the disease and died with all the symptoms of typhoid fever; his nurse was also attacked with typhoid fever, and died in the third week. A young lady also paid the third patient a visit, and rendered her some service in the sick-chamber, and this person soon fell ill with symptoms of typhoid fever, in another house, in which five other persons were subsequently attacked by the same disease. (M. Lombard, Gaz. Méd., 1839, p. 138: quoted by M. Piedvache, Mém. de l'Acad. de Méd., tome xv. p. 294.)

Dr. W. Budd had seventeen cases of Enteric Fever under his care in the hamlet of North Tawton, Devon, and during the prevalence of the disease three persons left the hamlet ill of the fever.

A went to Morchard and died there, and ten days after his death two of his children had the fever in the same house.

B also went to Morchard, and three cases of Enteric Fever afterwards occurred in the house where he lay ill.

C went to Chaffcombe, seven miles from North Tawton, and nine other cases of Enteric Fever appeared in the farmhouses to which he went. One of these nine left Chaffcombe and went four miles away, to Loosebeare, to be nursed. Several inmates of the house into which this patient was received were subsequently attacked with Enteric Fever, and from this house the disease extended over the whole hamlet. An infected boy also left Chaffcombe, and took the fever to a cottage midway between Bow and North Tawton, and five persons subsequently fell ill of Enteric Fever in the house into which he was received, and in the adjoining one. Besides these there was no single case of the sort nearer to Chaffcombe than North Tawton. "There were twenty or thirty hamlets in the neighborhood similar in all respects to Loosebeare. From the soil of all, through month after month of the same fine, dry, autumnal weather, human and other exuviæ exhaled into the air; and yet, while at Loosebeare a large proportion of the inhabitants were lying prostrate with intestinal fever, in not one of the exactly similar places was there a single case." (Dr. W. Budd, *Lancet*, July 9, 1859, p. 8.)

It is reasonable that those who have witnessed such instances as these should be fully persuaded that Enteric Fever is propagated by contagion. But there are many physicians, and amongst them those who have had the most extensive experience of Enteric Fever, who conclude either that the disease is destitute altogether of contagious properties, or only possesses them in a very slight degree.

[Dr. Austin Flint,[1] of New York, who has contributed one of the most remarkable series of facts showing the influence of tainted drinking water in Typhoid Fever, nevertheless writes thus: "Under ordinary circumstances it is not diffused by contagion;" "facts appear to show the spontaneous generation of the causative agent in the great majority of cases."—H.]

That form of Enteric Fever which prevails continuously in London is certainly non-contagious. I have never had cause for the slightest suspicion of contagion in any case which has come under my observation, either in King's College Hospital or in the London Fever Hospital, and this generally is the experience of hospital physicians, both in London and in Paris.

Since writing the above, the following instance, in which there is evidently a strong probability of contagion, has come under my notice. In the present summer (1865), four members, A, B, C, D, of a family of six adult people, residing at St. Peter's Terrace, Notting Hill, were attacked with Enteric Fever. A and B had been residing at Ryde for a month. They returned to town with two other members of the family on the 26th of May. A suffered ever since his return with diarrhœa, which continued, and the symptoms of Enteric Fever became well developed. "On the 28th of June he had a severe attack of intestinal hemorrhage, which recurred two or three times for several days; eventually he recovered." About the 7th of June, his sister, B, fell ill of the disease, and she died comatose on the 29th of June. On July 10th, C and D, who had not been absent from London, manifested symptoms of Enteric Fever, and were sent into King's College Hospital, where they came under my colleague Dr. George Johnson's care. I saw them frequently. They had well-marked Enteric Fever, and are, at this present date (August 22, 1865), slowly convalescing in the Twining ward. On the 8th of August, a nurse in attendance upon these patients was taken ill with the same disease, and now is under my colleague Dr. Beale's care, in the same ward, in a very precarious state.[2] This last patient, it

[1] Practice of Medicine, 4th Edition, p. 683.]

[2] She died on the 25th of the above month, and I witnessed the post-mortem examination. There was extensive ulceration of Peyer's patches and enlargement of the soli-

must be observed, slept every night at St. John's House, situated at a distance from the hospital, and between it and the Thames; and a great many isolated cases of Enteric Fever have occurred in London during this month. All but the last patient came under the care of my friend, Dr. Easton, of Connaught Square, and to him I am indebted for the particulars having reference to the period before B and C were admitted into the hospital. Dr. Easton carefully inquired into the sanitary condition of the house where the disease broke out. No cause could be found, and care was taken to mix the evacuations with Condy's fluid and dispose of them immediately they were passed. C and D had scarcely any communication with A and B.

In considering the question of contagion it must be granted that the occurrence of any number of cases simultaneously or successively in any given house or hamlet, can never prove the fact of contagion. The general conditions, moreover, favorable to the increase of the disease, are so common and wide-spread, that one who entertained limited views of the nature of the disease, might account for its propagation among the inhabitants of Geneva, above referred to, for example, by assuming—and not improbably—that the conditions favorable for the spontaneous development of Enteric Fever existed in each of the four or five houses in which the patients resided, and that the intercommunication which took place between them was a curious coincidence—each patient in reality being independently affected. If this explanation be thought satisfactory in the first instance adduced, it cannot be considered otherwise than unlikely and superfluous in the second, in which the general evidence of contagion appears to be complete. It is true that the conditions favorable for the outbreak of Enteric Fever appeared to exist equally in all these little hamlets; and if it had arisen in any or in all without the intervention of an infected person—and it must have so arisen in the first case—the disease in each individual might reasonably have been referred to a spontaneous origin.

M. Gendron himself was unable to account for the first cases in many localities, and M. Piedvache, after a careful examination of the whole question of contagion, "feels almost sure that typhoid fever, under some circumstances, declares itself at once and in sufficient numbers to constitute an epidemic, independently of contagion." (Mém. de l'Acad. de Méd., tome xv. p. 137.)

We have now to inquire into the conditions with which Enteric Fever is immediately associated, and the mode of its spontaneous origin.

Spontaneous Origin.—The majority of the French physicians who witnessed the epidemics which occurred in France, from 1841 to 1846, "signalize, amongst the causes to which they attribute the manifestation of these epidemics, the following conditions:—The more or less immediate vicinity of stagnant waters, marshes, or bogs, from which, chiefly under the influence of summer heat, effluvia arise and spread over the people—effluvia which tend to produce affections of a periodic type: the presence of dunghills, often accumulated and allowed to remain for a long time upon a public way, before houses, or the single door of some wretched hut; wells of water, level with the ground, permeated with water infected by drains and dunghills; an infected pool existing in the midst of a commune, and furnishing only a brackish, muddy, stinking water for the common drink of men and of animals who come to allay their thirst there; or springs, containing, it is true, a pure water and fit to drink, but disturbed by all kinds of animals who go there to drink and corrupt the water with their dung; or further, conduits of impure water, which become a source of infection to the houses near which they pass, or, discharging their contents upon the public ways, form puddles of stagnant water in streets badly kept and unprovided with suitable means for carrying off the fluid accumulations." (M. de Claubry, op. cit. p. 11, et seq.)

The occupants of a farmhouse are attacked with Enteric Fever, and the only discoverable cause is an over-flowing cesspool, or a stagnant, offensive pond, or a low-lying fold-yard, covered with a thick bed of dung and other refuse matter, from which putrescent runnings sink and saturate the soil in which the well, supplying the house, is excavated.

The accumulated or pent-up sewage of a town escapes into the subjacent soil within and about it, soaking into the wells and defiling the drinking water, and giving off filthy emanations into the air, and an outbreak of Enteric Fever follows.

A particular drain becomes obstructed, bad odors arise into the houses in communication therewith, and sicken their inhabitants, and Enteric Fever soon manifests itself.

Such are almost constantly observed conditions with which Enteric Fever is associated, and we must therefore conclude that this disease has a spontaneous origin in putrescent matters—that these, when preserved from the purifying influences of air and water, generate a poison, which, when admitted into the body, pro-

tary glands in the last three feet of the ileum, and these constituted the chief anatomical lesions.

duces Enteric Fever. Leaving for the present all speculation as to the nature of the poison thus generated, we will now proceed to inquire how the poison or poisons gain admission into the system—whether by the lungs or by the alimentary canal. The inquiry is an important one, and we must be careful to avoid the influence of partial views. If we except the most thoroughly-drained towns (such as London, where, perhaps, Enteric Fever attains its minimum development), wherever the air is vitiated with filthy odors, the subjacent soil is permeated by the putrescent source from which they arise, and the water is also contaminated. Hence, in many cases, it may be difficult to determine whether the poison were conveyed by the air or by the drinking water. Dr. Murchison, in his elaborate work on the Continued Fevers of Great Britain, endeavors to prove that Enteric Fever arises from "sewer emanations." "So far as we know," he says, "it is necessary for the production of the poison of Enteric Fever that the matter undergoing fermentation be either in a confined space, as in a drain or sewer, or that it be in a state of stagnation. Free exposure to the atmosphere, or constant dilution in a running stream, may not only render the poison inoperative, but may altogether prevent its formation. A privy outside a house is much less dangerous than a badly-appointed water-closet within." (Pp. 452, 453.) In the instances which he adduces to support this view nothing is said respecting the water-supply. Assuming that there were no sources of water within the precincts of the buildings, the outbreak of fever in the Westminster School—shortly after the exhalation, from a foul and neglected sewer, of a disagreeable stench, so powerful as to induce nausea—appears, pretty clearly, to have been due to the foul air. In outbreaks of disease amongst bodies of soldiers, school-children, &c. we must be prepared to look for the cause abroad as well as at home. A body of young cricketers, for example, may quench their thirst in some impure stream, and thus contract a disease, for which some apparent cause may be readily discovered at home. There can be little doubt that Enteric Fever is occasionally generated by exhalation from putrescent matters, but usually, we believe, they are ineffectual in the production of the disease. It has never yet been proved experimentally that Enteric Fever may be generated by emanations from decomposing animal and vegetable matters. Nor does it appear from the observations of those who have made special investigation in this direction that individuals whose occupations require them to spend much of their time in an atmosphere thus contaminated evince an unusual proclivity to Enteric Fever. Dr. Guy compared the past and present condition of ninety-six nightmen, with about the same number of bricklayers, laborers, and brickmakers, and after a most critical investigation, he says: "An examination of the tabulated results of his inquiries must convince the most sceptical that the health of scavengers is fully equal to that of the laboring man with whom they are compared." (Journal Statis. Soc., 1848, vol. ii. p. 79.) This agrees with the observations of M. Parent du Chatelet and others on the health of men who work in sewers. In the country it is a common practice to empty the privies and spread their contents over the gardens or adjoining fields. The filthy odors are endured as a temporary inconvenience, but experience proves that the practice is a harmless one. In two instances I have known the inhabitants of a house to be exposed for months to the direct emanations—in the one case, of a large cesspool; in the other, of a common-sewer. In both cases a leaden pipe, in communication with the water-closets, was carried down inside the house and thence into the sewer or cesspool. After several unsuccessful explorations it was discovered that rats had eaten a large hole in the pipe leading to the cesspool, and through this aperture the fetid exhalations from the surface arose directly into one of the sitting-rooms and a passage of the building, and on hot and damp days the effluvia were intolerably offensive. The cesspool was emptied at this time, and I witnessed the process. The contents were semi-fluid, of a dirty-black color, and the surface of the pool was covered with large bubbles of gas. Now, although several persons were continually exposed to filthy exhalations, in the one case for many months, and in the other for several years, none suffered from diarrhœa or Enteric Fever. Numerous examples of this kind teach us that we must not be too ready to attribute Enteric Fever to foul air, but that we should be prepared to acknowledge other means by which the disease may be introduced into the system. Of these we may consider two,—impure water and bad food.

[In some inquiries into the statistics of Typhoid Fever in Philadelphia, obtained at my request from the official records of the Board of Health by Dr. B. Lee, it was ascertained that the largest mortality from this disease during a series of years occurred in two kinds of localities. These were: 1, those districts adjoining the Schuylkill and Delaware rivers, near the outlets of the city sewers; 2, those whose sanitary conditions were otherwise of the worst kind, from poverty, ignorance, vice, and neglect.—H.]

The following instance is worthy, from the simplicity of the attendant conditions,

of attentive consideration with reference to the spontaneous origin of Enteric Fever. An outbreak of Enteric Fever occurred at Stangenrod, "a village chiefly seated on a naked, exposed, isolated eminence. Trees, excepting young brushwood, are not seen in the neighborhood, and there are but few marshes; a small stream has its source at some distance, but scarcely touches the extreme boundaries of the district. In several places are flats covered with broom and used as pasture grounds. The basis is mostly basaltic rock, covered with a layer of clay and loam mixed with sand and flint. The air is keen and pure, and the atmosphere rarely disturbed by clouds. The position of the town is healthy as to local influences, and not favorable to the production of endemic disorders, which, it appears, had never been observed in it. The inhabitants were poor, and chiefly occupied in agriculture and pasturage; comfort and wealth were exceptions. The houses were small, low, dirty, and surrounded by dunghills. The general want of spring-water the inhabitants attempt to supply by collecting rain-water in bad reservoirs." (Dr. Ebel, Ed. Med. and Surg. Jour., 1837, vol. xlviii. p. 160.) The disease appeared at the close of July 1833, and terminated in April 1834. The spring of 1833 was dry, followed by a dry summer, with insufferable heat, continuing to autumn without rain, and with little wind (p. 187). 157 out of 318 inhabitants were attacked, and 19, or 12·1 per cent., died. Dr. Ebel believed that the chief cause of the disease consisted in "the entire want of good fresh water and the use of corrupted water. The inhabitants of the village used for domestic purposes rain-water obtained from marshes, and preserved in insufficient reservoirs until required" (p. 188).

In the latter part of 1859 a severe outbreak of Enteric Fever occurred at Bedford, and there was every reason to believe that it was due to fecal matter soaking into the wells from the numerous cesspools of the town. The water from these wells was found to contain a large quantity of decaying animal matter, evidently derived from the sources alluded to. (Simon, Report to Privy Council, 1860.) Early in October 1847, "intestinal fever" broke out almost simultaneously in thirteen houses in a certain terrace at Clifton. The houses were far apart in the terrace, and there was little or no intercourse between their inmates. The inhabitants of these thirteen houses drew their drinking water from a well situated at one end of the terrace, and at the end of September it became evident, from the taste and smell of the water from the pump, that it was tainted with the contents of the sewer. The remaining twenty-one houses were supplied with water from another source. (Dr. W. Budd, *Lancet*, 1859, p. 432.) Other instances of the direct association of Enteric Fever with defective drainage and contaminated well-water may be found in the Sixth Report to the Privy Council, 1863.

Rain, the natural preventive of Enteric Fever, may, under certain local conditions, be the means of diffusing it. This appears to have been the case at Festiniog.

The houses in which the disease appeared are situated on the slopes or bases of mountains on the sides of the valley stream. "The majority have no privies nor ash-pits, nor have their inhabitants access to places of this sort. The custom of the neighborhood is to use the fields, or when house utensils are employed to empty them at a distance from the houses. Houses in a row are generally provided with one or more privies, with cesspools common to the row. The air is pure and the natural drainage good; the valley stream is exposed to constant contamination. The inhabitants stated that water for internal use is got from wells on the mountain side, presumably situate above any possible source of pollution" (see the Report to the Privy Council by my colleague, Dr. Buchanan; Sixth Report, 1863, pp. 787–8); but they acknowledge that they use the water of the stream in the valley for some domestic purposes, and it is obvious how readily this may be substituted for the purer, on all occasions, and particularly when the distant spring fails, and laziness or lack of time constrains the water carrier.

Under such conditions as the above, filth accumulates upon the surface during a dry season, and remains harmless there till drenching rain gradually washes it down into the common stream, which thus becomes continuously contaminated for weeks or months. Dr. Buchanan has pointed out these conditions, and is inclined to attribute the prevalence of the disease during an unusually *wet* autumn to water contamination, combined with exposure to cold and wet.

Impure water appears to be equally a cause of intermittent fever and its complications. The Inspector of Hospitals writes of Walcheren during the prevalence of the severe intermittent fever there: "The bottom of every canal that has communication with the sea is thickly covered with an ooze, which, when the tide is out, emits most offensive and noisome effluvia; every ditch is filled with water which is loaded with animal and vegetable substances in a state of putrefaction; and the whole island is so flat, and so near the level of the sea, that a large proportion of it is little better than a swamp; there is scarcely a place where

water of a tolerably good quality can be procured." Sir John Pringle mentions "that the men-of-war which lay all the time at anchor in the channel, between South Beveland and Walcheren, even during the worst period of the distemper, were not affected with either flux or fever, but enjoyed the most perfect health." (Davis on the Walcheren Fever, 8vo. 1810, p. 15.) These sailors were doubtless provided with a supply of good water.

The inhabitants of a marsh, seated in a basin of clay, or level with the bed of a river, must of necessity drink water contaminated by their excretions and other impure matters, if the water be derived from the marsh itself, and the drier the season the more concentrated the poisonous impurity.

Dr. W. Budd most strongly insists that the essence of Enteric Fever is contained in the alvine dejections of the patient, but we cannot adduce any facts recorded by himself that give material support to this view, and our own observations lead us to the conclusion that the intestinal discharges do not contain any *volatile* poison which is capable of generating Enteric Fever. [Dr. Ballard and others[1] have reported several instances in which outbreaks of typhoid fever, in London and elsewhere, have been traced to a close coincidence with the distribution of milk, furnished to a number of families from the same dairies. The contamination of the milk by foul water has been inferred; and, in some of the cases, the existence of typhoid fever near the inculpated dairies has been shown. It appears, however, to be still an open question, in some minds at least, whether these instances are to be considered as giving evidence of the diffusion of a specific *contagium* of Enteric Fever, or whether they ought not rather to be referred to the category of facts, otherwise numerous, showing the promotion of the disease by *foulness* of all kinds, in air, water, or food.—H.]

Food in an incipient stage of putrefaction is also capable of generating symptoms and intestinal lesions apparently identical with those of Enteric Fever.

Dr. Kerner of Weinsberg has collected 35 observations of poisoning from eating a certain kind of smoked puddings, which he regards as putrid food. They are chiefly composed of animal matters, and have a putrid savor and odor. Post-mortem examinations often revealed traces of inflammation of the œsophagus and pharynx; gangrenous patches of the stomach; intestines inflamed in divers places, or even gangrenous in part; lungs strewn with black spots, or hepatized, (Orfila, vol. ii. p. 636, 1843.) Dr. Schumann records the symptoms and post-mortem appearances produced by eating similar food. The symptoms were those of Enteric Fever, *plus* inflammation of the pharynx, œsophagus, and larynx. Post-mortem examinations revealed inflammation of the intestinal canal: "L'intestin grêle, quelquefois très distendu par les gaz, présenti des traces d'inflammation très intense, et souvent des plaques gangréneuses." Lungs gorged with blood. (Archives Générales de Méd., tome xxii.)

Granting that Enteric Fever may be produced by the ingestion of putrid animal substances, we shall be at no loss to find a cause for the origin of many of the isolated cases which occur, for such may exist in every household. There is nothing, for example, more essentially putrid than the *decomposed cheese* with which many persons habitually indulge their appetites, and persons unaccustomed to such food can hardly be supposed to partake of it with impunity. It is a matter of common experience that an *egg* will sometimes produce vomiting and purging.

With regard to the identity and nature of the poisonous agent or agents which produce Enteric Fever, we know nothing. It has never been demonstrated that any particular gaseous body can induce the lesions found after death from Enteric Fever. There are, however, both mineral and vegetable substances which, when introduced into the stomach, produce symptoms and morbid changes, if not identical with those of Enteric Fever, at the least hard to be distinguished from them. Thus, to take vegetable substances:—Twenty-four hours after the ingestion of *poisonous mushrooms*, the members of a family were taken with nausea, and the next day frequent vomitings of bilious matters and abdominal pains, of which symptoms three children died. The other members had insupportable pains in the stomach, loins, and abdomen, meteorism, retention of urine, tenesmus, glairy sanguinolent stools, vomiting, and great thirst. On the fifth day, shivering, collapse, and death.

The stomach and intestines were found covered with whitish or yellowish mucus, the valvulæ conniventes and mucous follicles generally very prominent. (C. P. Galtier, Traité de Toxicologie, tome ii. pp. 564–569.)

The effects of *colchicum* still more closely resemble those of Enteric Fever. Two young women took each about ʒiv of tincture of colchicum. Both died of violent gastro-intestinal inflammation. The viscera of one were tied up and sent away for analysis. In the other, who died twenty-eight hours after taking the poison, the lungs were healthy, stomach distended by gas, and the mucous membrane evidently softened. Throughout the

[1 British Med. Journal, Nov. 28, 1870; Lancet, April 5, 1873; Ib., Aug. 16, 1873, &c.]

whole extent of the intestine the muciparous follicles were of the size of millet seeds, and in the inferior third of the ileum the plates resulting from the agglomeration of the mucous crypts were also much developed and of a violet color. (Galtier, loc. cit. p. 322.)

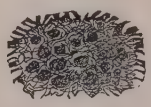
In poisoning by *Cicuta virosa* similar symptoms and swelling of the glands of the intestine are produced.

Has the poison which so frequently generates Enteric Fever any relation to the elaborated narcotico-acrid principles of these or other plants? If so, is it generated in the process of decomposition, and is it possible to obtain it by lixiviation of the impure soil, or by precipitation from the contaminated water? Or again, is the poisonous agent merely a common product of decomposition, and destitute, like that common contagious sanies which so readily induces phlebitis or erysipelas, of any specific character? Such are the speculations into which further inquiries respecting the origin of the disease lead us.

Returning now to the facts above detailed, we may conclude, (1) that refuse animal and vegetable matters, if allowed to accumulate and decompose in seasons of draught, generate a poison which, if not washed away or diluted by sufficient rain, rises into the air, or becomes diffused in the water; and which, when introduced into the body by these media, may produce Enteric Fever; (2) that food or water rendered impure under conditions less general, may also be the means of inducing the same disease; (3) that there can be little doubt that the usual symptoms and post-mortem appearances of Enteric Fever may arise during the progress of several other acute diseases as a consequence of a general inflammatory condition.

Diagnosis.—The indications of inflammation and ulceration of Peyer's patches are the following: Pain and tenderness in the right iliac fossa; general derangement of the alimentary canal, accompanied by persistent diarrhœa; light ochre-colored watery stools; hectic fever; and the eruption of rose-colored papules. If these symptoms be associated with any febrile condition or complication whatever, we may be sure of the presence of enteric inflammation. If they are absent, the case may be one of commencing pneumonia, erysipelas, hernia, puerperal fever. But each or all of the symptoms of enteric inflammation may be latent, and our attention may, therefore, be altogether called away from the abdomen to the head or the chest. In the absence of diarrhœa, the other characteristic symptoms being present, we may assume, generally, that the intestinal glands are simply inflamed and swollen; the presence of the pale, watery, flocculent stools, on the other hand, may be taken as indicative of the ulcerative stage. Diarrhœa alone, or even associated with a febrile state, does not furnish conclusive evidence of intestinal lesion; but there is one unmistakable evidence occasionally present, from which, in the absence of every other symptom, we may positively declare that the agminated glands of the ileum are in a state of sloughing inflammation; it is the presence of a fragment of a disintegrated Peyer's gland in the stools. If the floculent debris, when examined under water by the aid of a pocket lens, present a number of minute, closely-placed follicular depressions with minute circular orifices, loosely embedded in a ragged fibrous stroma (fig. 12), we have direct

Fig. 12.



Fragment of a Peyer's gland.

and positive evidence of the nature and progress of the disease. Even while the pale stools are still solid, a shreddy fragment presenting these unequivocal characters may be found adherent to their surface. To apply the test, the stools should be strained through a little cap net, the matters arrested thereby should then be washed, floated in clear water, and examined with a common pocket lens. The structure of the agminated gland is quite distinct from that of any other tissue in the body, not even excepting the thyroid gland and tonsils. Cellular vegetable structures, such as the rind of an orange, may be mistaken for the intestinal slough, but these are distinguished by their softer and more pulpy texture, and by their vegetable qualities. If there be much fever, with headache and delirium before the abdominal symptoms are developed, Enteric Fever may be mistaken for several other acute diseases, such as scarlatina, variola, typhus. In the first of these diseases we have the same bright cornea and flushed face, and the same lively character of the delirium. When characteristic symptoms are absent, we must suspend our diagnosis for a day or two, using at the same time precautionary measures against contagion. If the rash and sore throat of *scarlatina* be but slightly developed, or if the rash have receded, the diagnosis will be difficult and unsatisfactory.

If, in the accession of *variola*, the severe lumbar pain be absent, and the eruption make its first appearance as a few isolated papules upon the chest and abdo-

men, the case may be doubtful for a few hours, but the hardness of the varolous papule will speedily remove all doubt.

Chronic tubercular peritonitis presents many of the symptoms of Enteric Fever. Hectic flush, pinched features, abdominal pain, tenderness, gurgling, and diarrhœa are common to both diseases. But in tubercular peritonitis the tongue is clean and moist, there is no eruption, and frequently the abdomen is distended with fluid effusion; moreover, there may be evidence of tubercular deposit in the lungs.

Tubercular ulceration of the intestines, with tubercular deposit of the mesenteric glands, is a condition frequently mistaken for Enteric Fever. Two of M. Louis' cases (Ob. xliii. xliv. vol. ii.), given as examples of "latent typhoid fever," are instances of general tuberculosis simultaneously affecting the lungs and the intestines. I have already included another case (see case 12) recorded by this author (Obs. xxxiv.) under Tuberculosis, on account of the co-existence of miliary tubercles in the lungs and ulceration of the glands of the ileum, regarding the lesions in the chest and abdomen as simultaneous manifestations of the same pathological condition. If the lungs had presented evidence of only common inflammation, there would have been no reason for distinguishing this case from Enteric Fever. Indeed, the distinction between acute phthisis simultaneously affecting the lungs and intestines, and Enteric Fever, will more often turn upon the difference between tubercular and simple pneumonia than upon a difference in the character of the intestinal lesions. Inasmuch as the lungs are so commonly affected with inflammation in Enteric Fever, and the tubercular ulceration induces all the more prominent symptoms of Enteric Fever,—such as hectic, abdominal tenderness, tympanites, diarrhœa, and even intestinal hemorrhage,—the diagnosis is often exceedingly difficult. If the patient have been long declining in health, with emaciation, sweating, cough, and expectoration, and we find dulness at either apex of the lungs, gurgling, or amphoric breathing, we may set down the case to be one of phthisis. Doubtless, it is possible for tubercle to be deposited nowhere else in the body but in the solitary and agminated glands of the intestine, and in the neighboring mesenteric glands; and, in such a case, the diagnosis between the two diseases, hardly or not at all to be obtained during life, would turn upon the distinction between tubercular deposit and ulceration, and the nature of the inflammatory process and ulceration in Enteric Fever. I have examined the intestinal lesions of individuals in whom, after death, the lungs have been found consolidated with tubercles, and excavated into ragged cavities at their superior parts, side by side with the corresponding abdominal lesions of Enteric Fever. In the early period of the tubercular disease, I have been unable to detect any difference in the morbid phenomena; both diseases fall with greatest severity upon the solitary and agminated glands of the lower third of the ileum, and the appearance of the ulcers is similar, except that in Enteric Fever the inflammatory action and swelling is usually greater; in both diseases the contiguous mesenteric glands have the same violet color, soft consistence, and increased size. The diseased parts offer the same microscopical appearances; but the corpuscles of the inflamed glands within and without the intestine are a little smaller in Tuberculosis than in Enteric Fever. In the advanced period of the tubercular disease, however, the ulcers are very distinct from those of Enteric Fever; they have an irregularly circular, thick, often indurated, elevated, angry-looking border, inclosing, and here and there continuous with, large interrupted irritable granulations, between which little yellow masses of tubercular matter, firmly attached to the base of the ulcer, are to be seen. In chronic cases, moreover, and before ulceration has begun, the swollen intestinal and mesenteric glands are free from inflammation, and the tubercular matter within them has a white, opaque appearance. It frequently becomes hard, gritty, and cretaceous, and sometimes forms compact stony calculi.

The disease, however, with which Enteric Fever is most frequently confused, is *typhus*.[1] In our own country, Willis, Sydenham, Huxham, and other acute observers in every generation, have recognized and asserted the distinction between these two fevers, but a general confusion of the diseases has, nevertheless, prevailed amongst the members of the profession, until within some twenty years ago, and we are greatly indebted to Dr. A. P. Stewart of the Middlesex Hospital, and to Sir W. Jenner, for reasserting and proving that a distinction really exists, and for rendering the diagnosis between the two diseases more clear and distinct. What these observers have done for England, Dr. H. C. Lombard of Geneva, Messrs. Gerhard and Pennock of Philadelphia, Hildenbrand and Griesinger in Germany, and Forget, Godélier, Barralier, and others in France, have done for their countries.

Still, there are physicians who, either from partial views, or from insufficient experience of the two diseases, do not recognize any specific difference between them, and regard the abdominal lesion merely as a complication of typhus. From

[1] See article Typhus Fever in this volume.

what has been said on the associated pathology of Enteric Fever, it may, perhaps, be argued that the abdominal lesion, which is assumed to be characteristic of Enteric Fever, is nothing more than a local result of a common febrile disorder of the system; yet it is remarkable that lesions of Peyer's patches, which may sometimes be found in every other acute disease, are never, I believe, found in typhus, in which the febrile condition is both very general and very prolonged. I have examined the intestines in a great many cases of typhus, with the exclusive view of detecting, if possible, some participation of Peyer's patches in the general inflammation, but have always failed to detect either swelling or ulceration.

Case 20.—Three months ago, a woman, aged 30, and her little daughter, came under my care, with well-marked symptoms of typhus. The hot dusky skin of both patients was maculated with a distinct mulberry, typhus rash, which in the mother was petechial on the twelfth day of the disease. On the following two days the rash was still more plainly marked, and the case offered a good example of "spotted fever." There were no gastric or intestinal symptoms; sordes formed on the teeth, and the tongue was dry and brown; the eyes were dull and suffused; the mind heavy and confused—in short, all the symptoms of typhus were most distinctly marked. On the fifteenth day the bowels became loose, and a considerable quantity of blood was passed *per anum* the same day. At 11 A. M. on the sixteenth day intestinal hemorrhage was again declared by the passage of much clotted blood. The hemorrhage continued during the day, and at one time the bed was saturated with it and it ran down upon the floor. She sank and died at 5.45 P. M. on the same day. I was induced to re-examine carefully the skin of her little daughter who lay in an adjoining bed. A dark typhus rash was still out upon the chest and abdomen, and there were no traces of abdominal mischief.

On post-mortem examination of the body of the mother, which was well developed and well nourished, I found the small intestine perfectly healthy and pale; every Peyer's gland was also pale and healthy, and no solitary gland was either enlarged or prominent. The mesenteric glands and spleen were normal in size and appearance. In the cæcum, an inch and a half beneath the margin of the ileo-cæcal valve, I found a circular ulcer of the mucous membrane a quarter of an inch in diameter; the surface was grayish-white, like an aphthous ulcer of the mouth; the margin was slightly excavated, slightly raised, and vascular. On the opposite wall of the cæcum, a little higher up, where it becomes ascending colon, there was another much larger ulcer; it was an inch and a half long by half an inch wide, and it lay across the bowel; the surface was irregularly excavated, clean, and pale; the margins irregular and sharp; it extended below the mucous membrane, and lay in the thickened areolar tissue. In the transverse colon there were eight other small ulcers, resembling the first one, but deeper, and with margins more sharply cut and vascular. Excepting the largest, all the other ulcers were placed over one or other of the bands of longitudinal muscular fibres. Seven were arranged in a row upon one of them, at distances of about an inch. The intervening mucous membrane was congested, but not swollen; the ulcers were plainly the source of the hemorrhage.

Such are the characters and situation of the intestinal lesions in typhus fever when they occur, but they are very uncommon. They do not appear to commence, like tubercular ulcers and the ulcers of Enteric Fever, in the solitary and agminated glands. In the case last described, the solitary glands contiguous to the ulcers were perfectly healthy. If we now compare the general symptoms of the two diseases, we shall find equally well-marked differences. The countenance in typhus is dusky, or suffused with a dusky flush, which spreads continuously over the face, neck, and shoulders; the eyes are injected and the pupils contracted; the expression is heavy, the intellect dull, and the delirium is generally quiet. In Enteric Fever, the expression is bright, the pupils are dilated, and the hectic blush patchy, and limited to the cheeks; the delirium is often intermittent, becoming worse at night, and disappearing during the day. Its character is more lively than that of typhus. The eruption in typhus is a mere indistinct passive congestion, soon becoming a minute extravasation, and forms no distinct elevation. The rose spots of Enteric Fever are raised inflammatory papules. Diarrhœa, often profuse, frequently accompanies typhus, but the stools are always dark; the bile is thick and dark colored. The patient has no peculiar odor in Enteric Fever, but in typhus the disgusting exhalations from the skin are so strong and peculiar that we may often diagnose typhus by means of the nose alone. Typhus runs a regular course, and is terminated in the third week. Enteric Fever has no regular course, no certain date of termination. Typhus kills by coma or congestion of the lungs, Enteric Fever by asthenia, pneumonia, diarrhœa, hemorrhage, or perforation—very rarely by coma.

PROGNOSIS.—The prognosis in Enteric Fever must be formed with extreme cau-

tion; the worst accidents of the disease sometimes occur when all appears to be going on well. The mortality, however, is not great. Out of 18,602 cases, there were 3,447 deaths, or one in every 5·4. (Murchison, p. 529.) A pulse continuously frequent, and much hectic or obstinate diarrhœa, are very unfavorable symptoms. Hemorrhage, to any considerable extent, associated with tympanites, and occurring at a late period of the disease, is an unfavorable symptom. If there be excessive tympanites and abdominal pain, there is little hope. A fatal termination must also be anticipated if the stupor or delirium becomes continuous, and associated with muscular twitchings. The delirium sometimes assumes the traumatic character, or the patient becomes obstinate as well as stupid: such features also forbode an unfavorable issue.

On the other hand, we hail a fall in the temperature of the skin and the appearance of a copious eruption of sudamina over the abdomen and chest as most favorable indications.

Treatment.—The early recognition of enteric disease is of the utmost importance, for its progress is frequently so very insidious that many patients go about their usual occupations at a time when Peyer's glands would present very grave lesions (*e. g.* case 4), and complaining of nothing more than loss of appetite and debility. Our suspicions must be on the alert in such cases, and, before all things, we must ascertain the condition of the alvine secretions. Long before diarrhœa sets in, the well-formed feces may be observed to be of a light-yellow or ochre color—a condition in which they may exist in the absence of diarrhœa, but at a time when the intestine may be gravely ulcerated. The indications in the early period of the disease are to relieve internal congestion, and to revive the function of the liver. If the bowels be confined, a dose of castor oil, or of compound rhubarb powder, should be given. If the skin be inactive, the hot bath should be used, and a diaphoretic, composed of aromatic spirit of ammonia and acetate of ammonia, prescribed. With the view of arousing the liver to activity, I have given the following every three or four hours, for two or three days, until some improvement was manifest in the condition of the alvine secretions. When necessary, I have continued the administration of the mercurial until its constitutional effects—slight tenderness of the gums and fetor of the breath—began to appear.

℞. Hydrargyri cum cretâ, gr. iij.
Pulvis cretæ aromaticæ cum opio, gr. v.
Sodæ bicarbonatis, gr. xx. Misce, et fiat pulvis.

If this relaxes the bowels, I have combined it with a little more of the chalk and opium powder, or suspended its use altogether, and trusted to infrictions of mercurial ointment into the groins or arm-pits. I have employed this treatment in all cases which have come under my care in the early period of the disease, before diarrhœa had become severe, and have reason to believe that marked benefit has resulted therefrom, for the subsequent course of the disease in these cases has been uniformly mild. Calomel should be avoided; its action is too irritant. Saline purgatives and the vegetable cathartics must never be employed in the treatment of this disease, at any period. A single dose of jalap, scammony, and the like, may suffice to develop the worst features of the disease. If, at an early period, we succeed in producing a flow of healthy bile, with moderate action of the bowels, we may arrest further progress of the disease and restore the appetite. If there be diarrhœa and sickness from the commencement, we may prescribe an ounce of chalk mixture with ten grains of subnitrate of bismuth and five minims each of tincture of opium and dilute hydrocyanic acid, every two or three hours. This usually succeeds in allaying the vomiting. For simple vomiting of sour bilious fluid, a mixture, containing thirty grains of bicarbonate of soda and five minims of dilute hydrocyanic acid is very serviceable. The acid condition of the secretions in the upper portion of the alimentary canal, and the deficiency or total absence of alkali from the bile, are facts to be borne in mind in the treatment of Enteric Fever.

If the disease exhibit an intermittent character, quinine, gr. v to x may be given every evening. In the absence of cerebral or gastric disturbance, quinine is a most valuable remedy for subduing the evening exacerbations of fever so frequently present. Grains iij to x may be given in a glass of water, with this view, every or every other day at noon.

In the further progress of the disease, our treatment will have almost exclusive reference to the abdominal lesion.

Diarrhœa must be restrained, some physicians think checked, if possible, altogether. My own experience, however, leads me to believe that moderate diarrhœa in the early period of the disease is beneficial in some cases, and more especially in those in which delirium appears early. In the congested condition of the abdominal viscera which exists in Enteric Fever, one or two liquid stools in the course of the twenty-four hours doubtless acts beneficially in all cases, and our object in the early period of the disease should be to keep the diarrhœa within moderate limits, rather than to stop it

altogether. In the early period, chalk and bismuth, with catechu and opium, is usually all that is needed to check the diarrhœa and allay irritation. As soon, however, as the diarrhœa becomes excessive, or we have reason to suspect ulceration, stronger astringents must be given. Some physicians use sulphuric acid with opium—℞. Acidi sulphurici diluti, ♏xxx; tincturæ opii, ♏x; decocti cinchonæ, ℥iss; fiat haustus, quartis horis sumendus. We prefer the styptic salts, having found them much more efficacious: indeed, the acid mixture often increases the purging and pain. Acetate of lead, nitrate of silver, and sulphate of copper are employed. The first may be given in the form of mixture—℞. Plumbi acetatis, gr. iij–v; acidi acetici, ♏iij; morphiæ acetatis, gr. ⅛th; aquæ cinnamomi, ℥iss; fiat haustus, quartis horis sumendus. Acetate of lead is a very suitable and efficacious remedy, but its continued use in Enteric Fever should be avoided, as it may subsequently affect the system injuriously. Dr. Tweedie and M. Trousseau speak in high terms of nitrate of silver. It may be given combined with a grain or two of compound soap-pill in doses of a quarter of a grain to one grain, every three or four hours. Dr. Tweedie says: "I have prescribed it extensively in Enteric Fever, and continued its use for a considerable time, and have never witnessed any approach to discoloration of the skin." (Lects. on Continued Fevers, p. 233.) Of all medicines, we consider sulphate of copper to be the most efficacious in restraining the diarrhœa of Enteric Fever. We may give it in quarter-grain doses, combined with two grains of compound soap-pill, to be taken every two, three, or four hours. If need be, the dose may be increased to a grain, a day or two afterwards. For children, it may be prescribed in doses of the eighth or sixth of a grain. If too large a dose be given at first, it may excite vomiting. In small doses we have often prescribed it when there has been considerable irritability of the stomach, in which case it appears to act as a sedative. Alum, catechu, tannic and gallic acids, krameria, hæmatoxylon, &c., are of comparatively little value in the treatment of Enteric Fever.

[Oil of turpentine, the use of which in Typhoid Fever was urged by the late Dr. G. B. Wood, of Philadelphia, has sometimes an exceedingly good effect, apparently as an alterative to the diseased intestinal mucous membrane and glands of Peyer. It acts most favorably in small doses (ten or twelve drops every 3 or 4 hours), in mucilage, with a few drops of laudanum.—H.]

Starch and opium enemata—(Mucilaginis amyli, ℥iv; tincturæ opii, ♏xv–xxx; fiat enema, nocte vel nocte maneque injiciendum)—are of great value in allaying that irritability of the lower bowel which often induces purging. When enemata cannot be retained, we may still use suppositories. (Pilulæ saponis compositæ, gr. v to gr. x.)

Abdominal pain and tenderness.—The disease being localized in the right iliac region, we must direct our remedies to this part. Hot sedative fomentations, turpentine stupes, or poultices containing an admixture of mustard, should be frequently applied to the abdomen. If there be much tenderness four or six leeches should be applied, partly to the right iliac region and partly around the orifice of the bowel. Leeching of the anus is the most effectual mode of relieving the intestinal congestion. If the pain be great, an occasional full dose of opium will be needed.

Tympanites.—If there be any increased fulness of the abdomen, a flannel or linen bandage should be placed around it. In commencing tympanites we regard this as a very important part of the treatment, as it at once diminishes the congestion of the inflamed part, and prevents injurious distension. It also gives support to the painful abdomen in the process of respiration. Folds of wet cloths may be interposed between the bandage and the abdomen.

If the tympanites be considerable, it becomes a most distressing symptom, and the life of the patient is in great danger from distension of the diseased and attenuated bowel; laceration of its ulcerated coats being imminent so long as the distension continues. To relieve this painful and dangerous condition, turpentine stupes should be applied over the whole abdomen, and a gentle support given by means of a thin flannel bandage. An assafœtida enema (℥xij ad ℥xx enematis assafœtidæ P. B.) often gives much relief. If we should fail, however, to cause expulsion of the air by this means, a long elastic tube with wide side openings may be passed into the colon and retained there at intervals. Dr. Tweedie speaks well of the use of the stomach pump, per rectum, in the relief of this condition. "I have applied it," he says, "in some cases with happy effects, and withdrawn the accumulated air which may be passed through the lower tube of the stomach pump into a basin containing water." (Op. cit., p. 237.) Oil of turpentine (♏x–xx), or oil of rue (♏iii–v), combined with opium and given by mouth, are often serviceable in the relief of pain and flatulent distension.

Intestinal Hemorrhage.—Moderate capillary hemorrhage from the general mucous surface of the bowel must be regarded as beneficial, and we should employ no means to check it, but if the blood be clotted, in large quantity, and unmixed

with mucus, we must fear the erosion of a large vessel, and treat for such an accident very promptly. A bladder of ice bandaged upon the right side of the abdomen, and the internal administration of gallic acid, solution of perchloride of iron, acetate of lead, or turpentine, are the most hopeful means of arresting it. Sulphate of copper in combination with soap-pill is a very valuable remedy in this condition also, and one upon which we are inclined to place most reliance. If the patient have been previously taking the copper salt, the dose may be increased at once to one or two grains. Turpentine, in doses of ten or fifteen minims, given every half-hour or hour, is often effectual in stopping the hemorrhage, and is especially useful in cases where there is a tendency to syncope. The solution of perchloride of iron of the British Pharmacopœia is a very valuable remedy for intestinal hemorrhage; ℳxx–xxx in a wineglassful of water may be given by mouth every two or three hours. If the hemorrhage be slight and the arterial action much excited, ℳxv tincturæ digitalis, with ℳxxx tincturæ ferri perchloridi, ℥iss aquæ menthæ piperitæ, may be given every four hours. This may be administered alone or in combination with thirty minims of dilute sulphuric acid. If we fail to arrest the hemorrhage by these means, the bowel may be injected with one or other of the following enemata. ℞. Plumbi acetatis, gr. x; acidi acetici, ℳx; morphiæ acetatis, gr. ½; aquæ tepidæ, ℥iv; misce. ℞. Liquoris ferri perchloridi, ℳxv; morphiæ hydrochloratis, gr. ½; aquæ tepidæ, ℥iv; misce.

Cerebral Symptoms.—The indications in the treatment of cerebral symptoms are to relieve congestion and procure sleep. If there be much pain and heat of the head, cold water may be applied as an occasional douche, a gallon being poured in a gentle stream upon the head as often as the heat becomes excessive. Rags wetted with water, or spirit and water, may be applied in the intervals. If this treatment fail to restrain the vascular excitement, a few leeches should be applied behind the ears, or a blister upon the nape of the neck. As often as they are required, full doses of opium should be given to procure sleep. In the majority of cases the cerebral affection is mild and requires no direct treatment, and the sedatives given to relieve the abdominal symptoms are usually sufficient to calm the nervous irritability and procure sleep. When the pulse is fast and there is pulmonary inflammation, we must be careful to avoid large doses of opium. In some cases the delirium makes the patient obstinate, and he persists in refusing food and drink, and keeps the teeth firmly clenched. In such a case, with diarrhœa present or impending, we cannot feed *per rectum;* we must therefore gag the patient and use the stomach-pump.

In such a state, too, we should daily examine the pubic region. Now and then we are painfully reminded of the negligence of those in close attention upon the patient, by discovering, after death, the bladder distended almost to the umbilicus, and with its attenuated coats inflamed and softened.

Pulmonary Symptoms.—Bearing in mind the frequency of pulmonary complications, we should carefully regard the breathing, and occasionally examine the chest. If pain and crepitation be developed in any part of the chest, a blister should be applied and mercurial infrictions used. Cough and bronchial dyspnœa may be treated with ipecacuanha and senega, and the application of mustard poultices and turpentine stupes. [Prevention of hypostatic pulmonary congestion and splenization may be promoted by care in changing the position of the patient every few hours; especially not allowing him to lie very long at a time on the back.—H.]

Food.—While we are thus combating the disease, the most unwearied attention must be given to the support of the patient. The blood impoverishes, and the body emaciates very rapidly, and our endeavor must be to introduce such food into the stomach as will be most easily digested. All nourishment must be given in a fluid or pultaceous form. Eggs, milk, vermicelli, arrowroot, or ground rice, beef-tea, gelatin—alone, or in various combinations—will be the most appropriate articles of diet. The eggs must be given in the form of emulsion in a little wine whey, tea, or cocoa. Two or three should be given daily. Milk-arrowroot, containing a little brandy, is a very appropriate nutriment. The beef-tea must be[1] thickened with well-stewed vermicelli, or isinglass. Small quantities of food should be given at a time, and repeated every one or two hours.

Stimulants, in any considerable quantity, are not needed in the early period of the disease. When required they should be given well diluted. A few ounces of wine in the form of wine whey, or dry port mixed with an equal quantity of water, may be given with a little sponge-cake at intervals. Effervescent wines must of course be avoided. If the heart's action be weak, or the patient tends to lapse into the typhous state, brandy may be freely given, carefully avoiding excess. The following general rules may be observed in the administration of alcoholic

[1 May be, rather; as, if well made, some patients will prefer it without such additions.—H.]

stimulants in this disease. As long as the pulse remains under 120 and retains moderate force, six to eight ounces of wine, or four ounces of brandy, given within twenty-four hours, will be sufficient. When the pulse ranges between 120 and 130, and is small, we may double these quantities; and if the heart does not respond to the stimulant after twelve hours, thrice the original amount may be given. The bulk and force of the pulse must be our chief guides; and if these notably fail from day to day, we must daily increase the quantity of the stimulant until the patient is supplied with as much as half an ounce every half-hour, always diluted with a little milk, tea, water, &c. When there is much hectic, and the pulse is small and sharp, strong stimulants often appear to increase the irritability of the system, and in such a case we should give them sparingly and in the early part of the day, trusting to a dose of quinine, with or without opium, according to circumstances, in the evening.

Excepting a little custard, solid food of all kinds must be absolutely avoided, until a week after the diarrhœa has ceased, and the stools become solid. Then we may venture to order boiled fish with bread. A boiled egg, a little fish, or a ripe pear or plum, taken too early, will almost certainly bring back the diarrhœa with a complete relapse [and danger of intestinal perforation.—H.].

The patient must return very gradually to ordinary diet, and he should be directed to eat slowly and masticate the food thoroughly. At first boiled rice should be taken in place of potatoes.

Convalescence is sometimes very slow, and often retarded by the occasional recurrence of diarrhœa. The styptic should be continued a week after the stools have become solid. At first the bowels are usually constipated, and this condition we shall do well to maintain for days. Subsequently, it will be advisable to relieve the bowels occasionally by a dose of castor oil.

As soon as the digestive function is restored, we prescribed cod-liver oil as a supplement to the diet, in all cases where there is much emaciation, and if, as is rarely the case, the oil does not digest, we may direct it to be rubbed into the abdomen.

[Amongst physicians in America, of latter years, *expectancy* has grown more generally in favor in the treatment of Typhoid Fever than in that of almost any other disorder; as no malady more evidently presents a self-limited character. Cases of an ordinary grade are often conducted through their whole course to a good convalescence, with no medication, or almost none. Great importance belongs, under such management, to the regulation of the diet of the patient. This must be liquid and concentrated; milk and (unfiltered) beef-tea being the chief articles for this use; these must be given in small quantities at short intervals (a tablespoonful or two every two or three hours), in the more feeble cases, day and night. Also, of course, the bed-clothing must be attended to; requiring change in the garments next the skin every two or three days, or oftener, if soiled by perspiration or other discharges. The state of the bladder must be watched, to anticipate prolonged retention of urine. Change of position, from one side to the other, or from the back to the side, should be insisted on, to avoid hypostatic pulmonary congestion. Ventilation of the chamber, without direct draughts, must be secured. These, with the quiet always essential to the sick room, and other parts of "good nursing," will suffice in the care of not a few cases of well-marked, uncomplicated Typhoid Fever, without even the addition of alcohol to the food. A large minority of cases, however, will call for this addition.

Within a comparatively few years, very positive treatment for fever as such, in all its forms, is advocated by a number of medical authorities. Prominently, cold bathing has been brought forward, by Drasche, Brand, Liebermeister, Ziemssen, Wilson Fox, and others. The patient, according to this method, is immersed, for ten minutes or more at a time, in water at about 70° Fahr., to reduce the temperature. Ziemssen's plan is undoubtedly the safest, of placing the ill person in water at 95°, which is gradually lowered to 80° or 75°, watching the effect upon the pulse, respiration, and countenance. The indiscriminate use of the sudden cold plunge bath in fever is certainly not free from danger by excessive depression.

Quinine is now employed by quite a number of practitioners as a direct *antipyretic*. In order to produce a positive effect in lowering the temperature of the body, it has to be given in rather large doses; ten grains or more, repeated. Salicylic acid acts in a similar manner. It remains to be shown with certainty that this mode of reducing temperature has, on the whole, a favorable effect upon the progress of Typhoid Fever, when it is not complicated by the influence of malaria.—H.]

PROPHYLAXIS.—Sufficient evidence has been adduced to prove that Enteric Fever commonly arises from the retention of refuse animal and vegetable substances within an undrained, or imperfectly drained soil. If, therefore, the contamination of the soil be prevented by the construction of sufficiently inclined sewers with impermeable walls, and the inhabitants be provided with abundance of pure water, Enteric Fever may be expected to disappear almost entirely.

The requirements for the prevention of the disease are sufficiently simple, but they are not easily fulfilled in every place where living beings are congregated. Nature, indeed, has provided these sanitary conditions almost everywhere, and if man would be more mindful of them his life would be rarely sacrificed to Enteric Fever. A house built upon a hillside, with its spring of pure water above the foundation, and its cesspool below it, would be free from this disease, as far as external conditions are concerned. But reverse the position of the cesspool and the spring, and the disease may appear at any time.

If the dwelling be built upon a low-lying flat, and there is no near spring or flowing stream, these two necessaries—a pump and a cesspool—must needs co-exists side by side. In such a case they should be as widely separated as possible, and the sides of the well should be thickly covered with concrete. Whenever the premises are small, and it can be so contrived, a water-closet should be provided, and the excretions carried in an impermeable drain to a distance from the pump. In towns and large villages both pumps and cesspools should be abolished, and every house provided with a water-closet in communication with a sewer.[1] The water should be derived from a distant elevated spring or reservoir, preserved from contamination at its source, and conveyed in well-joined iron pipes to its destination. The soil should be well-drained, and during the continuance of dry weather the drains and sewers should be regularly flushed.

We cannot positively say that Enteric Fever arises from the ingestion of diseased meat, but there is a strong probability that it does sometimes originate in this cause. Whether this cause has been in operation during the present year (1865), when "contagious typhus" has been so generally prevalent amongst, and destructive of our horned cattle, there is no direct evidence to show; but it is remarkable that, co-incidently with the spread of the cattle disease, there has been a great increase of Enteric Fever. On turning to the Table at p. 237, it will appear that the number of cases admitted into the London Fever Hospital in 1865 is more than double that of every preceding year, and more than treble that of the majority. The only effectual way of preventing the admission of diseased meat into the markets would be to establish a limited number of slaughter-houses, where the animals, previous to being slaughtered and afterwards, could be inspected by proper officers. The experience of the present severe epidemic of cattle disease has taught us, that, *after death*, it is exceedingly difficult and, to an inexperienced eye, impossible, to distinguish positively between the flesh of an animal which has died of contagious fever, and that of one slaughtered in perfect health. In the absence of that more general protection which is so urgently required, two precautions should be taken: first, flesh of a flabby consistence and of a dusky, dead hue should be avoided; and secondly, all meat should be so thoroughly cooked that the fibre is quite firm and free from juice, which, on exposure to the air, becomes red. In the treatment of the contagious variety of the disease, the ordinary precautions against contagion must be taken, viz., the isolation of the patient and the disinfection of everything that has had contact with him.

TYPHUS FEVER.

By George Buchanan, M.D.

Definition. — Typhus Fever is an acute specific disease, lasting from fourteen to twenty-one days, characterized by an eruption of its own that appears between the third and sixth day, eminently contagious, and forming strongly-marked epidemics.

Under the name "Typhus," the writer of one of the Hippocratic treatises describes a disease that agrees in its essential features with typhoid fever. But the term was not afterwards used to signify a special disease until the time of Sauvages, in whose nosology it is adapted to certain forms of continued fever, while the name Synochus is used for another class of cases.

[1 The frequent imperfection of water-closet connections with sewers, allowing the escape of sewer-gas into houses, has lately been credited with the production of a large number of cases of Typhoid Fever.—H.]

The disease as above defined, and now known as Typhus, has been separated from other forms of continued fever within the last thirty years.

Typhus Fever has received a multitude of names, almost every epidemic having added some fresh one. In English, Spotted Fever, Petechial Typhus, Epidemic or Contagious Fever, Putrid or Malignant Fever, Camp or Jail Fever, are samples of the names that have been conferred on the disease, from various considerations of its nature or cause.

Etiology.—The causes that predispose to Typhus may be considered as affecting the individual and the community.

In the individual, sex and age have no influence in determining an attack. In the writer's experience, at the London Fever Hospital, very nearly equal numbers of each sex, and persons of every age, from a fortnight to over eighty years, have been attacked. Upon the authority of death registers and hospital statistics, the statement is constantly made that Typhus attacks adults more than children; but the evidence furnished by these data is quite untrustworthy as showing the relative proclivity of different ages to an attack. Typhus, as it appears on the death registers, is indeed incomparably more frequent among adults than children; but that is because children rarely die of it, not because they are rarely attacked. And in hospital records a much greater proportion of adults than of children are seen to be admitted; but this is because of obvious domestic reasons, because of the slightness of the fever in children, and often because of the rules of the institution. When inquiry as to age is made to include every case of attack, children and adults are found to be quite equally susceptible; the actual incidence may even be observed to be strongly upon the young, partly because of their greater numbers, and partly because adults are frequently protected by previous attack.

Depressing mental influences, overwork, and anxiety, appear to be causes that render the system more liable to contagion. Fear of contagion is often alleged, and perhaps justly, to be another such cause. It is especially among persons of better rank of life that these influences have been observed to operate. Depressing bodily influences are, however, of far greater moment. With persons temperate and provided with sufficient food, the contagion of Typhus, even though intense, is usually resisted for some time; but with intemperate and ill-fed persons, contagion is received so readily, and so small a quantity of it produces an attack, that it is constantly difficult to find out whether in the particular case there has been any exposure to contagion at all. It is essentially among the poor underfed population of large towns that Typhus epidemics occur. Paupers and the class just above paupers are the chief, and except in intense epidemics almost the only, sufferers from the disease. People whose earnings enable them to get more than the bare necessaries of life from hand to mouth do not suffer from Typhus save in exceptional instances, and usually as a consequence of constant communication with the sick.

The most violent epidemics of Typhus have been among communities that were fed more badly than usual, either through social difficulties, or through failure of crops; special hardships in war, and in civil life, strikes, and commercial distress, have at different times determined an epidemic prevalence of the fever.

The experience of Ireland in 1818 and 1847 illustrates the influence of privation in predisposing a community to Typhus. In each of those years an epidemic of this fever prevailed (along with relapsing fever), as a consequence of the almost complete failure of the potato crop; and it is estimated that on each occasion an eighth part of the entire population was attacked. But it must not be supposed that serious epidemics of Typhus require exceptional destitution as a necessary condition of their occurrence. A marked instance to the contrary is afforded by a recent intense outbreak at Greenock, where circumstances of special commercial prosperity had (by causing exceptional overcrowding) conduced to the epidemic spread of Typhus.

The next predisposing cause of Typhus is probably the most important of all, and consists in the association of conditions known as overcrowding, crowd-poisoning, or ochlesis. These conditions are scarcely to be separated from each other, but may be enumerated as overcrowding of dwelling-houses upon too limited area, overcrowding of rooms by too many occupants, bad ventilation of streets and houses, domestic and personal dirtiness. It is to the operation of this series of conditions that the special incidence of Typhus upon the laboring population of large towns is to be ascribed.

Illustrations of the effect of crowding too many inhabited dwellings upon a limited area might be gathered in abundance from the experience of camps, where the superficial space per man has been less than in the densest cities with their tall houses, and where Typhus has carried off large proportions of many armies. But they may equally be drawn from the experience of civil life. The town of England which habitually has most Typhus, and in which the most serious epidemics occur, is Liverpool. Here the huddling together of houses with insufficient space

around them is carried to a greater degree than in any other town in the kingdom. In Liverpool, a large number of the houses are built back to back, in unventilated courts, and the population is so dense that, in some districts, each person gets only eight square yards of superficial space. In these parts it is that fever specially flourishes, and in epidemic periods passes by none but those who are protected by previous attack. Glasgow is another instance of a town in which the packing together of houses reaches an extreme extent, and in which Typhus correspondingly prevails; its distribution following so exactly the degree of density of population in different parts of the town as to leave no doubt of the connection between the disease and this condition.

Overcrowding of the interior of houses by too many occupants, with deficient ventilation of rooms, may be illustrated as a cause of Typhus by the experiences of the common lodging-houses of English towns. Before the regulation of these by law in 1851, dwellings of this class were in a state of most miserable filth and overcrowding. In London and Liverpool, especially, there is evidence that they were peculiarly infested with Typhus, far more cases of this fever occurring in them than among an equal population residing in poor tenements of another class. Since 1851, the number of fever cases in common lodging-houses has been accurately ascertained, under the same Act of Parliament that has diminished their overcrowding and improved their cleanliness; and it is found that in some thousands of such houses in London, hardly any Typhus exists in non-epidemic times, and that in epidemic times they suffer now much less than other houses inhabited by the poor. In Liverpool, it was upon the overcrowded lodging-houses that the chief force of the epidemic of 1847 fell; the cases of Typhus that occurred in them being numbered by thousands. During the year 1863, when the fever again became epidemic, in a thousand regulated lodging-houses of Liverpool, only twenty-four cases occurred, a quite inappreciable fraction of the whole number of fever cases in the town.

Doubtless both sorts of overcrowding act chiefly by facilitating communication between the sick, and contagion from person to person. And hence comes one explanation of the different degrees in which overcrowding favors Typhus in town and country. In many country cottages, very considerable crowding of rooms, no ventilation, and habitual dirtiness exist as constant conditions, and yet Typhus is practically unknown.

Other predisposing cases of Typhus require brief consideration. Persons of all countries and races exposed to its influence contract Typhus with equal readiness. But the disease is essentially one of cold and temperate climates. Within such climates there is no country, whose epidemics are accurately recorded, that does not suffer more or less from Typhus; and, on the other hand, there is no sufficient evidence that this fever occurs within the tropics. Of all countries, Great Britain and Ireland are the chief seats of Typhus, which occurs here more constantly from year to year, and with severer accessions of epidemic force than elsewhere.

In the United Kingdom, the large trading ports are especially prone to Typhus; but this must be ascribed less to their position on seaboard or river than to the greater communication they have with localities from which infection may be derived, and the extreme density of population in such places.

By season and meteorological influences Typhus is not known to be very much influenced. It is a very common, though not an invariable occurrence, that the last month or two of the year is the chosen period for an increase in the prevalence of Typhus. In the main this connection appears to be established through the influence of cold upon the vital powers of the individual, and upon the social and domestic conditions of the poor community. People suffer more from scantiness of food and get less ventilation in their crowded rooms as winter sets in. Drought, again, has sometimes appeared, as recently in Bristol, to predispose to Typhus, through reducing the supply of the element necessary to cleanliness. Low elevation of site, again, is a condition that renders a place less easily purified by currents of air than if it lay higher, and may in some minute degree assist in the development of an epidemic. But to such atmospheric and climatic conditions as these, and they are the most weighty ones of their kind, an inferior degree of importance only is found by experience to attach.

As to the exciting causes of Typhus, the great, if not the only one, is the specific poison of the disease transmitted from person to person by contagion or fomites. Evidence of propagation of the fever by communication between the sick is seen in its epidemic spread when it enters a community of susceptible persons, and even more conclusively in the way in which persons exposed to none of its predisposing causes catch the fever when they are in close attendance upon cases of Typhus. Nurses in hospitals, where many cases of Typhus are received, invariably get Typhus, no matter under what sanitary conditions they are placed. There appears to be no exception to this rule, unless, indeed, it be that the nurse is personally insusceptible of the disease from a

previous attack of it. Medical men and Catholic priests in attendance upon numerous Typhus cases are also almost sure, sooner or later, to get the Fever, and that they do not fall ill with so much certainty or rapidity as the nurses appears due only to their contact with the sick being less constant and intimate. The contagious matter of the disease seems peculiarly capable of destruction when it is diluted with air. Thus tolerably close communication with the body of a Typhus patient appears requisite for the reception of contagion from him. Casual visitors to fever wards very seldom get Typhus, and in private houses of the better class the disease rarely spreads to the attendants. Extension of Typhus from a hospital to the adjacent streets is unknown, even though there should be hundreds of cases congregated within a very short distance of other buildings. In these respects Typhus differs much from smallpox and some other diseases of its class.

There appears reason to believe that Typhus can be communicated apart from actual intercourse with the sick by residence in the house where the fever has recently existed, and by the use of articles of bedding and clothing that have been recently used by Typhus patients. But, as compared with scarlatina, for instance, the degree to which the contagious matter of Typhus can be thus conveyed by fomites is very inconsiderable, and, in fact, it appears to be very seldom thus conveyed if the simplest means of purification by air and water are employed.

Great immunity from an attack of Typhus is obtained by a person who has once suffered under it. Some, but very few, well-authenticated cases of second attack are on record.

There are many instances where Typhus Fever occurs in individuals who cannot be ascertained to have been exposed to any contagion, and where the readiest explanation of the occurrence of the fever is that it has originated *de novo* from the intense operation of its predisposing causes. Cases of this kind, happening in the absence of epidemic influence, and constituting the first instance in a community among whom the disease afterwards spreads by contagion, have been collected by Dr. Murchison, and must be allowed to have weight in favor of the theory advocated by him, that destitution and overcrowding are by themselves capable of generating afresh the contagious matter. If, to this view, the speculative objection be opposed that a specific self-multiplying matter can only be allowed to have a specific origin, the speculative answer may be given that at some time or other there must have been generated a first case of Typhus, and that the same *de novo* production may therefore occur again. Considerations of this kind, on one or the other side of the argument, are of little importance by the side of Dr. Murchison's actual observations. The most serious obstacle to the reception of this theory arises from the analogy of other specific diseases as to the present production of which by contagion, and contagion alone, there is no question. Thus, in many outbreaks of scarlatina and smallpox, the source of infection in the first instance is often as obscure as in the cases of apparently spontaneous Typhus cited by this author, and there are cases even of children's syphilis originating under circumstances that the most experienced investigator has failed to connect with exposure to the poison. Another consideration which weighs somewhat against the belief that destitution with overcrowding is the condition required and sufficing for the *de novo* production of Typhus, is that outbreaks of Typhus, apparently spontaneous, sometimes occur in persons under excellent hygienic circumstances. Thus in a case that came under the notice of the writer, two boys living in an institution where every advantage of diet and lodgment was afforded (as may be held proved by the fact that the fever did not extend) were attacked within a few hours of each other with Typhus, and the most careful inquiry failed to show that either of the boys had had the opportunity of getting the fever by contagion.

Symptomatology.—The period of *incubation* of Typhus is not satisfactorily determined. It is so rare for persons to fall ill of this fever after a single exposure that opportunities of ascertaining the point in this way do not often occur. And in practice it is also extremely difficult to get, with any accuracy, at the limits of the incubation period from the times of first and last exposure. It is probable that this period is not constant, but that it varies from a few hours to several days.

The *invasion* of Typhus is generally marked by headache, more or less severe, loss of appetite and general malaise. For a day or two, and in the absence of information respecting exposure, there is nothing to distinguish the outset of Typhus from that of any other fever, unless it be the absence of the positive symptoms of other specific illness. It is particularly difficult to separate the invasion of Typhus from an attack of acute dyspepsia. Rigors are of frequent occurrence, but they are not so definite or so severe as in smallpox, or in the pyrexia accompanying internal inflammation. The sense of chilliness commonly complained of along with the early headache may not amount to actual shivering, and

it is often wholly absent. In slight attacks, especially in children, it may not be possible to settle the actual time of invasion. On the other hand, in severer cases the disease begins very suddenly with shivering, headache, and it may be vomiting. For three or four days the symptoms of the invasion period get worse, and are accompanied by sleeplessness and general pyrexial symptoms, thirst, heat of skin, pretty complete anorexia, and usually a very peculiar prostration. In a case of Typhus of any gravity the patient gives in to the disease within the first three days, leaving off his work and commonly taking to his bed by that time. Patients with an actual Typhus rash on them, and having been ill five or six days, do indeed sometimes apply as out-patients of dispensaries and hospitals, but this is incomparably of rarer occurrence with Typhus than with typhoid or smallpox patients.

Before proceeding to state the symptoms of a fully developed case of Typhus, under the heads of the organs and systems affected, mention must be made of *the general appearance* of the patient. This is so peculiar as to constitute to the practised eye a very ready means of diagnosing Typhus, and frequently even an important element in deciding on the nature of a doubtful case. In an average attack the patient lies prostrate on his back with a most weary and dull expression of face, his eyes heavy and with a somewhat dusky flush spread uniformly over his cheeks. In the advanced stages of a severe attack, he lies with his eyes shut or half shut, moaning and too prostrate to answer questions, to protrude his tongue, or to move himself in bed; or the mouth is clenched, the tongue and hands tremble, and the muscles are twitching and half rigid. The dryness of the mouth, the sordes on the teeth and lips, the hot dry skin, and the deafness are other symptoms which strike an observer so immediately as to deserve to be included in the physiognomy of the disease.

The maximum *temperature* reached in the course of the disease is rarely less than 104·9 or 105°, and in many cases it reaches 106·5–107°, sometimes even a higher point than this. This high maximum is, as a rule, in favorable cases only attained once or twice throughout the fever, and generally of an evening; the highest morning temperature very rarely exceeds 106°. The temperature begins to rise at the commencement of the disease, and has been observed as high as 103·8–104·9° the first evening; it continues rising till the third day, when it often reaches 105–107°. The maximum is generally attained in the middle of the first week, between the fourth and sixth days and generally on the fourth day, and then a slight though appreciable fall takes place. There is generally a well-marked remission about the seventh day. In Typhus, though less than in other forms of fever, there is an exacerbation in the evening, and the remission about the seventh day is, in some cases, only indicated by the comparative slightness of the evening elevation which then takes place. In the more severe cases there is no trace of remission at this period, but the temperature maintains itself steadily or even rises a little. The absence of this remission marks the case as likely to be a severe one.

In the second week the temperature rises again, but only for a day or two, and rarely reaches the maximum of the first week. The elevation may be from half a degree to two or three degrees, but averages about three-quarters of a degree, often lasts but for one evening, sometimes continues longer.

Between the twelfth and fourteenth day there is a remission, in both the mild and severe forms of the disease; even in cases about to prove fatal, and in those other severe cases which showed no decrease of temperature at the end of the first week. But notwithstanding some remission, the temperature in fatal cases often remains high (rarely, however, above 105°), and shortly before death a very rapid rise occurs, indicating that the fatal termination is approaching, in some cases the temperature being sometimes higher at this time than at any previous date of the disease. In cases of recovery from an average attack, defervescence generally occurs some time between the thirteenth and seventeenth day, and its approach is sometimes announced by a slight exacerbation which renders the subsequent fall more conspicuous. The return to the normal temperature takes place very quickly. In the majority of cases it is completed within twenty-four hours, often in twelve hours; it begins very frequently in the night, and the abrupt manner in which the fever leaves is one of the peculiar features of the disease, the temperature sometimes falling as much as three or four degrees in the course of a night. By the end of the second half of the third week the temperature has usually returned to its natural standard.

The difference between the morning and evening temperature is smaller in the first week and the first half of the second, not amounting to more than one degree or one degree and an eighth; somewhat more considerable from this time to the termination of the disease, averaging about one degree and a half. The evening rise is much less than in cases of typhoid fever, in which disease the great elevation of temperature which precedes the fatal issue of the disease, is not so marked as in Typhus.

Symptoms referrible to the circulatory system.—The pulse of Typhus is always accelerated; in a case of medium severity in an adult being about 120, in slighter cases not exceeding 80 or 90, while in children (by reason of their age), and in more serious attacks in adults, the pulse reaches up to 140–150, even beyond this to a number which cannot be estimated by the finger. From the time of attack on the one hand to that of improvement or death on the other, the pulse seldom fluctuates or falls; it rises steadily up to its maximum in favorable cases, maintains the same moderate excess for several days, and then subsides uniformly and rapidly. In graver cases the pulse continues to rise until the crisis of the disease is reached. A fall in the frequency of the pulse indicates, in the very great majority of instances, the commencement of convalescence, and a subsequent rise signifies the accession of some local complication. It is said that a sudden fall in the pulse, especially when it has been excessively high, occasionally precedes death. Probably this is sometimes the case when there are obvious symptoms of impending dissolution, but diminution in frequency of the pulse has not been observed to precede other fatal symptoms. The difference between the morning and the evening pulse appears to be only a slight exaggeration of that which is observed in health. Change from the lying to the sitting posture increases the frequency, but not to any remarkable degree.

The character of the pulse is peculiar; it often strikes the observer as being very large and strong, but very slight compression is ordinarily sufficient to obliterate it altogether. In other cases it is distinctly feeble and small, and when most rapid is often scarcely perceptible to the finger. Irregularity of rhythm is observed in some severe cases. A dichrotous pulse is occasionally found, but more rarely than in typhoid. On the other hand, instances are sometimes seen when every second beat of the heart only gives a pulse at the wrist. This circumstance is only transient, and must not be confounded with a real reduction in the heart's frequency.

The heart sounds in Typhus may be natural, but in severe cases they are weak and distant, the first sound especially, as has been shown by Stokes, being deficient in tone even to the point of being quite inaudible.

The capillary system exhibits also important changes in Typhus, showing themselves clinically on the conjunctiva and skin among the external parts, and in congestions of various internal organs. The eyes are often bloodshot, and the skin much injected, symptoms more observed in persons below the middle period of life than in old persons whose circulation is inactive. The injection of skin is sometimes carried to such a point, that the finger drawn lightly over the surface causes a white stripe to appear in the course of a few moments. Besides this general injection, a special eruption results from congestion and extravasation of blood in the vessels of the skin.

This eruption, constituting the measley or mulberry rash of Typhus, is present, at some time or other of the disease, in about 95 per cent. of cases, and forms the principal diagnostic evidence of the fever. It has been described by Jenner and most subsequent writers as consisting of two portions, but between the two every intermediate link may be found. The one is a faint, irregular, dusky-red, fine mottling, looking as if it lay some little distance below the surface of the skin, and were seen through a semi-opaque medium. This appearance is well expressed by the name (which is otherwise inexpressive enough) of "subcuticular" mottling. The other part of the eruption is formed by separate spots of small size and purplish color, scattered over the mottled surface, and looking more or less superficial. These are the "maculæ" of Typhus. They are irregularly roundish in shape, and in color vary from brightish-red to livid, fading into the color of the adjacent portion of skin. At their first appearance, they are often a little elevated, and, exceptionally, are found to be as much so as the spots of typhoid. In the course of a day or two they are no longer felt raised above the surface. The mottling often exists without the distinct spots; the spots rarely without a considerable degree of mottling. Usually, the two exist together, but in slighter cases (in children especially) the greater part of the eruption is formed by the general mottling, while in old persons it consists mainly of the distinct maculæ.

As a rule, the eruption of Typhus appears on the fourth or fifth day; it may, however, be met with as early as the third, and rarely is delayed as late as the seventh day from the onset of fever symptoms. It comes first on the backs of the wrists, the borders of the axillæ, and about the epigastrium; in many cases it covers the whole trunk, and frequently also the arms and legs. More rarely it is met with on the face and neck, but in children especially it may be so copious on the face as to resemble measles.

The eruption takes a variable time, under forty-eight hours, for its complete development, and then undergoes certain changes, which, if life is enough prolonged, end in its disappearance; but from the establishment of the eruption in the first week of the disease, no fresh spots are seen. The spots are at first wholly obliterated by pressure with the finger, but after a few days there is com-

monly some little yellow color left when the finger is removed, looking as if the coloring matter of the blood had stained the skin at the injected spots, and later in severer cases pressure fails to remove the maculæ to any considerable extent, owing to an actual escape of blood from the vessels. The duration of the eruption varies according to the relative amount of mottling or maculation, and according to the degree to which the spots become ecchymotic. In slight cases with few distinct spots, and occurring among children, the mottling may not last more than two days, and the skin be then left quite clear. In cases of medium severity, the greater part of the mottling disappears within a few days, going first from the face and wrists; the skin of the trunk still showing a crop of the irregular maculæ, half ecchymosed, and, in their later stages, apparently seated at various depths below the surface. In such a case the whole eruption lasts till the twelfth or fourteenth day. But in severer cases, especially when the general eruption is livid and the maculæ immovable by pressure, the typhus spots persist later than this, and the small ecchymoses may not disappear until the twenty-first day or even later. Desquamation of the cuticle is not observed as a consequence of the eruption.

Another lesion, of rare occurrence, connected with the circulatory system, is local gangrene. This is more commonly observed in winter than in summer. The toes, the legs, and even the nose may suffer. More frequently sloughs on the sacrum and heels are seen as the result of pressure combined with defective circulation. Large, heavy people, much prostrated, who lie helplessly on their backs day after day, suffer most from these accidents.

Other alterations, connected in nature with the circulation, will be considered symptomatically under the headings of the Brain and Lungs.

Symptoms referrible to the Digestive System.—The tongue of Typhus Fever presents every variety of appearance. In the earlier stages it may be unchanged or covered with a thick white fur. Among adults uniformly, as the disease advances, the tongue becomes dry, the fur forming a rough brown coating over a red mucous membrane. Often the tongue is so hard and the whole of the mouth so dry, that from this cause alone there is difficulty in protruding it. At the end of the second week, in favorable cases, the edges get moist, and the tongue clears, the fur disappearing molecularly or else in patches, leaving the mucous membrane shiny and red. In severer forms of Typhus, with a variable amount of fur, the dry tongue cracks and bleeds, giving rise to black sordes during the disease, and to fissures of which the remains persist after recovery. The tongue may be intensely red and cracked, without there being much fur, and in such cases the characteristic tongue of typhoid is closely simulated. The papillæ are rarely enlarged at any period.

The mucous membrane of the mouth and throat gets, like the tongue, dry and covered with sticky masses of mucus. The lips, in bad cases, become covered with black sordes like the tongue.

Thirst is a symptom met with very uniformly, and from the earliest period of Typhus. It does not give way until, as convalescence begins, the natural secretion of the mouth suffices to keep the surface moist. Extreme loss of appetite is another symptom of equal constancy. For a few days, indeed, in mild cases, the patient can be persuaded to take light food, but as a rule refuses everything solid, retaining a desire for stimulants only. These, too, are soon distasteful, and then the only thing relished is cold water. Vomiting is a less uniform symptom. It sometimes, indeed, occurs at the outset, and occasionally forms a troublesome complication in the progress of the fever.

Diarrhœa, with tympanitis, is another digestive symptom of not infrequent occurrence. It appears to have much to do with the regimen under which the patient is put, inasmuch as it certainly occurs more in the practice of some institutions than of others in the same epidemic.[1] It is worth while to insist upon this cause of diarrhœa, since its occurrence at one time, or in one place, might otherwise be mistaken for a special type of the disease there. Thus in the practice of the London Fever Hospital, of recent years, diarrhœa has been seen in at least one-third of the cases of Typhus. In the epidemic of 1856, it was practically absent there; at Liverpool, during the present epidemic, there has been little of this complication; and even in the present London epidemic it has been absent in the Typhus treated at some workhouses. The symptom appears to be ascribable in some measure to the greater amount of liquid food that is forced upon the deranged stomach in the practice of some physicians. The plan of the London Fever Hospital is to give at short intervals as much liquid nourishment as the patient can be made to take. If the occurrence of diarrhœa do really result from this circumstance, it must be confessed to constitute a slight drawback to a plan of treatment, which for its aggregate results is not the less to be warmly advocated. When diarrhœa is present there is often

[1] It is quite uncommon in Hospital practice in Philadelphia.—H.]

considerable tympanites, and some gurgling may be felt, but it is seldom fine or confined to the cæcal region. The abdomen is in such cases slightly tender on pressure, but such tenderness is more about the epigastrium than elsewhere.

On the other hand, in many cases of Typhus the bowels are constipated, but they are readily acted on by purgatives. The stools in this fever differ from those of typhoid, even when diarrhœa is present. They are usually dark, and are of every consistence, but they do not exhibit the appearance of powdery matter suspended in liquid. Their reaction is stated to be acid.

There is an obscure connection between dysentery and Typhus, the fever appearing under certain circumstances to be generated by persons suffering from dysentery. When this connection has existed, the tenesmus and frequent bloody stools of dysentery have been observed to complicate cases of Typhus.

The writer has seen peritonitis occurring in one instance only among some thousands of cases of Typhus that have come under his notice; in that case it resulted from the rupture of a multiple abscess in the spleen following on endocarditis.

Symptoms referrible to the respiratory apparatus are so common and so important that they must be regarded as essential parts of the disease. In most cases of Typhus, during the second week there is some little dry rhonchus found at the posterior bases of the lungs. The chest should be examined daily for this condition, even when there is no objective lung symptom. By care in this respect, the accession of the next series of symptoms may constantly be prevented. These consist in increased duskiness of the face, livid flush on the cheeks (not specially on the malars), dusky color of rash—conditions indicating defective aëration of the blood—and these may exist without any cough, but are almost always accompanied by some increase in the frequency of respiration. The patient will make no complaint, but the smallest degree of any of these symptoms should at once point attention especially to the lungs. Often dry rhonchi at the bases will alone be found, but frequently also dulness of one or both bases. About the lower six inches of one base is the amount of dulness usually reached, but it may extend up to the spine of the scapula on each side. With the dulness are found increased vocal fremitus, high-pitched respiration, and at the earlier and later stages of the dulness, coarse muco-crepitation. Fine dry crepitation is less frequently heard over the solidified lung. It cannot be too strongly insisted on that, with all these evidences of lung mischief, there may be no cough, and no expectoration, up to the time that redux crepitation begins, and very likely nothing beyond the duskiness that has been mentioned to call attention to the state of the chest. Upon the occurrence of secondary crepitation, cough often becomes more troublesome, and if there have before been no expectoration it now appears, and consists of a semi-transparent tenacious mucus, scantily aërated, and frequently discolored with varying blood-tints, as in idiopathic pneumonia.

Bronchitis and consolidation of the lung in Typhus are very apt to improve about the period when the fever itself reaches its turning-point, which has been stated to be usually about the fourteenth day in adult patients with moderately severe Typhus, and these lung states do not often constitute a superadded disease after the end of the third week. But when they are present they constantly obscure the occurrence of a favorable crisis, and protract the total convalescence of the patient for several days. The physical signs in cases of recovery usually disappear pretty rapidly, but when dulness has been considerable it may not be quite got rid of for some weeks, although the patient be gaining strength, and have no other evidence of chest mischief beyond this dulness, and a pulse that keeps up above its natural standard. In cases of Typhus fatal from lung complication, the lividity of surface and the physical signs get gradually worse, and generally (but even then not invariably) there is visible embarrassment of respiration. [The origin of pulmonary embarrassment in *hypostatic congestion*, due to prolonged recumbency without change of position, has an important practical bearing in Typhus as well as in Typhoid Fever.—H.]

The nervous symptoms in Typhus are constant and prominent, and it is probably through the nervous system that the poison of the disease primarily operates. It is from certain of the nervous symptoms that the name of Typhus was originally conferred upon continued fever.

From the very outset of the illness these symptoms occur, consisting in rigor, headache, and weariness of body and mind. The amount of head symptoms is greater in proportion to the severity of the attack and the age of the patient. Restlessness and loss of sleep are, even in the slight attacks of children, pretty constant from the first. Sleeplessness often continues up to the time of crisis, and constitutes one of the most distressing parts of the illness to the patient; and even if he gets a good deal of intermitting sleep, he will frequently protest, with many complaints, that he has not slept a wink. The headache is often intense, but is a dull and heavy, and not a sharp pain, and is accompanied with some giddiness and with

noise in the ears. It gets worse through the first week, and then gradually disappears, rarely lasting longer than the tenth day. Before the cessation of headache, the intellect is heavy, the faculty of appreciating dates and intervals of time being notably confused.

In a large proportion of cases, delirium is a symptom of Typhus. It supervenes usually between the fourth and eighth days, the headaches going off as the mind begins to wander. Subjectively, as learnt from the statements of convalescents, the delirium is formed by utter confusion about time and place and people, and even about personal identity. The patient often has fancied that he is two or three people, and is undergoing several sets of miseries and horrors. Objectively delirium is of very various amount, and in character may be active and maniacal, or low and muttering. Much active excitement is rare in Typhus, but extreme degrees of it are occasionally seen, the patient praying, bawling, blaspheming, according to his habitual turn of mind, or leaving his bed to escape from imaginary ill-treatment. Suspiciousness is a not infrequent form of delirium, and the obstinate refusal of food that comes of this mental state may be carried to a degree that itself is fatal to the chance of recovery. Acute delirium commonly passes after some days into the low muttering form, the form which is more usually the character of the delirium from the first. In this the patient lies talking quietly to himself about matters that interested him at the time of his seizure, or on subjects suggested by what is going on, or he supposes to be going on around him. In severer Typhus, the muttering delirium passes into a heavy stupor, and tremulousness of the tongue and hands, with twitching of the muscles (*subsultus tendinum*), is then commonly observed. In very bad cases the patient cannot be roused from his coma, or a few days before his death he falls into the state known as coma-vigil, staring vacantly and with fixed eyes while in a state of complete unconsciousness. To the severe coma, rigidity of the muscles, fumbling at the bed-clothes, and loss of power of swallowing are added before death. Convulsions are another symptom of occasional occurrence, and of almost certainly fatal augury. They are epileptiform in their character, lasting a few minutes only, and giving place to profound coma, in which the patient dies, with or without a repetition of the convulsion. Convulsions in Typhus are almost always associated with albuminous urine, and, in a few cases where this has been investigated, with urea in the blood.

If the head symptoms have been prominent, and yet the patient recover, a childishness of intellect is often left for a few weeks. In very rare cases, the patient has remained insane for some time, but in such of these cases as have come to the writer's knowledge the previous history of the patient's mind was not satisfactorily made out. As a very rare condition indeed, softening of the brain, proving fatal, shortly after the fever has subsided, has been witnessed.

All cerebral symptoms are severe, and the delirium is commonly earlier and more active in persons of the better class of life, when they happen to contract Typhus, probably on account of the habitually greater activity of their brains.

Of the special senses, that of hearing is chiefly affected; besides noises in the head, deafness is of very frequent occurrence, beginning at the end of the first week, being slight, or nearly total in amount, and persisting even after the advent of convalescence. Nothing is to be seen in the ear to account for it. The eyes are suffused, and the conjunctival vessels injected. The sight is rarely affected, but much light increases the headache. The pupils vary a good deal from a condition of medium dilatation to one of great contraction. Their size has not, in the writer's experience, a very constant relation to the mental state of the patient, nor to the severity of his disease; but small pupils are the rule when coma is present. Some sluggishness of the pupil to the action of light is then also frequently observed.

Kidneys.—The urine of Typhus has not been sufficiently investigated, and many of the following statements rest wholly upon the writer's own observations.

In quantity it directly represents the amount of fluid ingested, regard being had to the other ways in which the body gets rid of water. The quantity has been found greatest in the first week of the fever; about the same or slightly less, in the second; and notably less during the third week after the commencement of convalescence; the mean of several cases and of several days being taken for comparison. A belief exists among good authorities that much ingested water is retained in the body during the whole stage of pyrexia, but of this there appears to the writer to be no evidence that will bear scrutiny. The occurrence of diarrhœa diminishes *pro tanto* the amount of fluid carried off by the kidneys; as to the perspiration, it is not yet demonstrated what alteration in its amount goes along with the increased temperature of the body; it is commonly assumed, and is probable, that very little water is got rid of by the dry-feeling fever skin. An increased amount of urine is said often to accompany the crisis; this has been observed occasionally by the writer, but with no approach to constancy.

The color of the urine in Typhus is variable. In most cases it is darker than usual up to the turn of the disease; it then becomes natural in color, and after the third week it is commonly pale. It is very rarely pale throughout. The reaction is acid, probably not more so than in health; and in a case quantitatively examined by Parkes, the free acid reached to only half the normal standard.

The twenty-four hours' urine is often quite free from deposit; but at some period or other of the disease it is usually turbid from lithates. A deposit of lithates has been said to occur as a critical discharge; but in the cases examined by the writer, this was observed quite as often at earlier as at later periods, and most frequently a day or two before convalescence. The daily amount of uric acid at the height of the fever appears (from one analysis by Dr. Parkes, and one by myself) not to be in excess of the healthy quantity. In Parkes's case the sulphuric acid excreted was rather high; the phosphoric acid has not been estimated.

The chlorides of the urine are greatly reduced during the pyrexial period of Typhus. This partly results from salt not being ingested; but there must be some other cause for their disappearance from the urine, since all chlorides may be withdrawn from a person in health, and yet the urine will continue to contain considerable quantities for some time after. In pretty severe cases of Typhus the abolition of the chlorides may be total; but usually there is a small amount secreted, estimated volumetrically at from two to three grains in the twenty-four hours, and just giving an opalescence when acid nitrate of silver is added to the urine. Before the advent of convalescence, the diet remaining the same, the chlorides reappear in some quantity, undergoing a gradual increase, though the quantity ingested remains the same from day to day. It is not ascertained how far their previous diminution is made up for by greater excretion of them during convalescence. Their disappearance is not connected with diarrhœa, nor is it due to the accession of pneumonia.

When common salt is taken into the stomach as a medicine in the early days of Typhus, while the natural chlorides are being excreted in diminished quantity, it does not pass out by the urine. When taken at a later period, just before convalescence, it is found freely in the urine. In fatal cases, it does not appear up to the time of death. In one such case, where twelve drachms of salt were given to a patient on the ninth and tenth days of Typhus, mere traces of it were excreted by the kidneys, up to the time of death on the seventeenth day; and upon examination of the blood, a certain small excess, but not accounting for one drachm of salt in the whole volume of the blood, was detected. It is probable, therefore, that the chlorine attaches itself to some solid tissue of the body; or that the excess is diffused alike through all the tissues, and that its retention is intimately connected with the febrile condition.

The daily excretion of urea in Typhus, as deduced from sixteen cases accurately observed through the several stages of the disease, is at first considerably above the normal amount. Taking one case with another, the daily quantity during the first week—the patient being fed on low diet, milk and beef-tea—may be stated as about double that of the fourth week, when he is sitting up and eating his fill of meat. The increase is found at the earliest day at which the urine has been examined. In three fatal cases, it did not diminish in quantity during the time that the urine could be procured. In cases of recovery the amount of urea gets gradually less, sometimes, but not always, showing a special decrease about the time of crisis.[1]

The urine of Typhus is occasionally albuminous. In fifteen male cases of various severity, examined throughout their course (some as early as the third or fourth day, and most as late as the fourth week), albumen was found in two cases. One of these was fatal on the tenth day without complication, and the urine contained much albumen on the fourth, fifth, and sixth days, the only occasions when it could be collected. In the other case, a trace was found on the eleventh day, but none before or after; here the patient died on the twenty-fifth day, from one of the rarer sequelæ of the disease. Albumen is only found in Typhus of considerable severity; but in some of the cases examined, though the fever proved fatal, there was none in the urine at any stage. The proportion of cases here stated to have exhibited albumen is below that which is reported as the experience of most observers.

A trace of sugar was observed at one time or another in nine cases out of fourteen, when it was sought for. It appeared at any period between the sixth and

[1] It must not be forgotten that some portion of the excess is due to gelatine taken in beef-tea. In one case it was attempted to estimate the influence of this element. For two periods the patient was fed on milk, and no gelatine, and between these periods he was fed upon three pints daily of the strongest beef-tea ("so strong it was quite a jelly when cold"), and nothing else. The mean of the two milk periods was compared with the beef-tea period, and an average daily excess of 11·5 grammes (174 grains) of urea was found in the latter. The writer would also like to see the influence of alcohol excluded in certain observations on the urines of fevers.

twenty-seventh days, and only lasted a day or two. It was probably no more than often occurs in health, and was clearly of no clinical significance.

Convulsions, as connected with kidney disease, have already been considered under Brain Symptoms. Retention or else involuntary passage of urine is frequent in severe cases.

Generative System.—The catamenia are sometimes present in patients on their admission to the London Fever Hospital; occasionally to a considerable degree. If not present at the earlier stages, the catamenia do not usually appear during the progress of the fever nor during the portion of convalescence that the patient passes in hospital. When pregnant women get Typhus, if they are past their sixth month, they frequently miscarry, but this accident adds very little to the danger of the case, Typhus Fever herein differing much from typhoid or scarlatina.

Duration.—The duration of Typhus Fever may be measured by the fading of the eruption, by the fall in the pulse, by the decrease of temperature, and by the general improvement in the aspect and condition of the patient. As a rule, amendment begins in all these ways pretty simultaneously, the fall in pulse and temperature being, however, the most reliable indication of approaching convalescence. Measured by these tests, the duration of the uncomplicated fever varies from twelve to twenty-one days, in mild cases (among children particularly) being sometimes less than twelve days, but only in very rare instances reaching twenty-two or twenty-three days. When the fever is complicated, for instance with erysipelas or parotid swelling, the pulse and temperature may keep up beyond this latter date. In about half the cases, they fall on the thirteenth or the fourteenth day. Except when petechiæ on the skin have been very distinct and irremovable, the eruption usually fades about the same time, and, with very rare exceptions, is altogether gone by the end of the third week.

Relapse in cases of Typhus, meaning by the word recurrence of the specific disease after apparent convalescence, is of excessively rare occurrence. Once only has it happened to the writer to see a true Typhus rash recur, and this was in the case of a woman who lost the eruption on the fourteenth day, and appeared to be getting rapidly well, when, after a few days' ailing, the eruption reappeared copiously on the twenty-fifth day and remained visible for more than a week, the patient passing through a second and very severe attack of the fever.

The duration of fatal cases of Typhus extends from two or three days to twenty-one days, which appears to be the limit of risk in cases of uncomplicated Typhus. In civil practice it is uncommon for the fever to prove fatal before the seventh day; but in certain epidemics that have occurred during warfare, death has frequently occurred at an early period, before the development of the eruption. The ordinary duration of fatal cases is twelve or fourteen days. When parotid swelling or other complication appears as the cause of death, the fatal result may be postponed to the thirtieth day, or even later, but it is not then due to the direct influence of the Typhus poison.

Termination and Sequelæ.—The *termination* of Typhus in recovery occurs often with great rapidity; the patient who the day before lay prostrate, stupid, and wandering, with only a slight fall in pulse and fever-heat to hint at the commencement of change, becoming conscious, looking comparatively intelligent, changing his position (though now feeling for the first time his utter weakness), and almost suddenly regaining his appetite. This improvement is more usually spread over two or three days, the lividity of the face gradually getting less, the tongue cleaning, the thirst disappearing, the pulse falling ten or twelve beats a day. The fall of temperature takes place pretty rapidly in almost all cases; and if it be retarded, while the patient appears to improve in other respects, the accession of some complication is commonly imminent. Occasionally the pulse does not fall below 90–100 for many days after manifest improvement in other respects. This is commonly due to some thickening of the lung remaining behind, and probably in other cases to the weakness of the heart being extreme. On the other hand, a fall in the pulse to a point much below the healthy standard is not infrequent in the early days of convalescence; the natural frequency being resumed in a short time as the patient gains strength.

From the time when the patient begins to mend, he commonly goes on getting appetite and strength from day to day. Any lung symptoms improve at the same time and rapidly disappear. Emaciation, which is seen towards the end of the acute stage, often continues to increase for some little time after the cessation of the febrile condition, but soon the convalescent regains what he has lost and returns to a state of unimpaired health. Frequently he finds himself after a month or two stronger and better than before his illness.

In cases of Typhus terminating fatally through the intensity of the disease itself, prostration, subsultus, and carphology increase, and low delirium passes into complete coma. The pulse and temperature

continue to rise; stools and urine are passed involuntarily; food is often obstinately refused, and there is much difficulty in swallowing for a day or so before death. When there is much bronchitis or congestion of the lung, increased lividity of face with quickened breathing and flapping nostrils are observed, but these conditions are often less noticed through the great prominence of the nervous symptoms.

A patient ill with Typhus does not always convalesce immediately upon the cessation of the specific fever. The *sequelæ* of the fever are indeed few and rare, especially when it is compared with typhoid or scarlet fever, and very seldom does it leave behind it any permanent impairment of health. These sequelæ consist either in the persistence of some of the local complications that have been enumerated in the description of the disease, or in the advent about the period of convalescence of certain conditions of an erysipelatous nature.

The complications that occasionally continue are consolidation of the lung, which occasionally goes on to gangrene, but generally mends in the course of a week or two; weakness of the heart, leaving the pulse feeble, with a tendency to palpitation for some short time: bedsores and gangrene of the toes of course have also to be repaired, or may cause death at a late period; and occasionally a patient dies shortly after the twenty-first day from the kidney disease that has been set up in the course of the fever.

When erysipelatous affections make their appearance, it is usually somewhat late in the fever, convalescence being retarded by their approach. But they may also occur during the earlier stages of the disease; and, although for practical purposes they may be regarded as sequelæ, it is probable they have an intimate connection in nature with the specific disease. Erysipelas itself, following the usual course of idiopathic erysipelas, occasionally occurs about the time of convalescence. It begins at the root of the nose, the fauces being at the same time red, and may spread over the face and head more or less widely, often causing suppuration in the eyelids. In three cases lately observed at the London Fever Hospital, rapidly developed œdema of the glottis had supervened in patients suffering from erysipelas after Typhus. Swelling of the extremities—sometimes consisting in inflammatory exudation into the cellular tissue, at other times connected with phlebitis—and pyæmic affections of the joints, always ending fatally, are other rare conditions of the same sort.

Of this group of sequelæ, the commonest is swelling, or bubo, of the salivary glands, which are liable to become affected at any period of Typhus, but especially in the third week; at any age, but mostly in the adult. In a few hours a swelling forms that is extremely tender, increases rapidly, and, in the majority of cases, goes on to suppuration. The parotid is the gland most commonly affected, and it is not uncommon for both sides to suffer. Next, but at a long interval in frequency, the submaxillary gland is liable to be affected, while the sublingual gland is very rarely attacked, in the only two cases seen by the writer, becoming involved after others of the salivary glands had swollen. Sometimes, more often in the child than the adult, parotid buboes subside without suppuration, but, usually, they go on increasing; in three or four days fluctuation is detected, and if the swelling be not evacuated artificially, it bursts in the mouth, or the meatus of the ear, or upon the external surface. These swellings appear to occur more in some epidemics than in others, and they add much to the fatality of the disease. If they are at all capable of spreading from one patient to another, it is to a very slight degree that this occurs, at any rate in hospital practice. Upon examination of all the evidence bearing on their nature, Murchison regards these buboes as forming a connecting link between Typhus and Oriental plague.

Inflammatory swellings and abscesses in other parts of the body are occasionally observed after Typhus. Commonly these accompany Typhus only as they accompany smallpox, or other acute specific diseases, but there have been some epidemics in which the lymphatic glands of the groin and axillæ have been observed to swell, again appearing to show an affinity of the disease with plague.

Diagnosis.—The diseases from which it is most often required to distinguish Typhus, are measles and typhoid fever, pneumonia, and certain brain diseases.

The eruption of Typhus is sometimes, though not commonly, a good deal like that of measles, and it appears about the same day after invasion. If it should happen that a child is attacked, and the source of contagion is unknown, there may be real difficulty in distinguishing the two diseases. Coryza, when present and distinct, points to measles. The eruption of Typhus is of a smaller pattern than in measles and scarcely ever has any crescentic shape. The occurrence of spots on the face by no means excludes Typhus. Much elevation of the rash is in favor of measles. If the diagnosis have remained difficult up to the sixth day, it may then usually be made with certainty, by noting the sudden fall of temperature that then characterizes measles, and that does not

occur so early or so completely in Typhus. Still even this means is only available for cases uncomplicated with pneumonia.

From typhoid fever, Typhus is usually pretty easy of diagnosis. Minor elements of distinction are, the nature of the fever prevailing in the same house and neighborhood, and the comparative immunity of old people from typhoid. In typhoid, the invasion symptoms are far more insidious than in Typhus; rigor and headache are less marked. In the early stages of typhoid, epistaxis is sometimes observed, a symptom very rare in Typhus, complicated with scurvy. [The same may be said of bronchitis.—H.] The eruption of typhoid appears later than that of Typhus, rarely being met with before the seventh day in typhoid, while in Typhus its appearance is very seldom postponed so late as this. The mottled, dull-red eruption of Typhus, with its irregular, non-elevated (at least after the first day), and often persistent spots, is not often closely simulated by the scantier eruption of lenticular, rose-colored spots of typhoid. But the most essential distinction between the two eruptions is, that of Typhus comes out in one single crop, while in typhoid fresh sets of spots appear day after day, and each spot lasts only three to four days. Diarrhœa is a much more frequent symptom in typhoid than in Typhus; but in the particular case, the presence or absence of diarrhœa is not much to be relied on for distinguishing the two fevers. The character of the stools is more distinctive; in Typhus they are natural or dark in color, and, if loose, of muddy consistence; while in typhoid they are yellow, and consist of powdery-looking matter suspended in liquid. In typhoid, the stools are alkaline, and contain crystals of triple phosphate; but the stools of Typhus come to resemble them in these respects when there is diarrhœa. The tongue does not give much help in diagnosis; for, though the typical tongue of Typhus—hard, thick, and with much dry brown fur,—resembles little the typical tongue of typhoid, flat, red, dry, and cracked, with little or thin fur; yet either of these descriptions of tongue, and every variety of them, may occasionally be found in either disease. That the tongue should be persistently moist, is a circumstance pointing much to typhoid. Considerable fluctuations of the pulse, and great evening rise of temperature, are points in which typhoid differs from Typhus, but to which appeal can rarely be wanted to guide diagnosis. The duration of the two fevers will commonly afford a point of distinction, if a case should by possibility have remained doubtful throughout. Typhus does not last more than three weeks, and in the attacks with obscure rash (where confusion with Typhoid is most possible), generally a fortnight only; while, in typhoid, the fever, as evidenced by the pyrexia and the eruption, goes on to a fourth week, and may go on to the thirtieth day, er even later. In referring to duration, as distinguishing the two diseases, it is, of course, necessary to exclude complications that may be keeping the patient ill, after the specific fever has left him.

With idiopathic pneumonia, it will easily be understood that Typhus may be confused, since a species of pneumonia is one of the commonest conditions complicating Typhus. The compressible pulse, the great prostration, and the brown tongue of Typhus are simulated by certain forms of pneumonia, in which, moreover, the signs that point to the chest may be no more prominent than they are in the lung-consolidation of Typhus. The presence of a Typhus rash is the essential means of separating the fever from the idiopathic local disease, and, without it, the diagnosis cannot certainly be made.

In the same way, the existence of Typhus eruption is the only way in which cases of this fever, complicated with other local lesions, can be distinguished from those local lesions occurring idiopathically—from uræmia or erysipelas, for instance.

In many cases of Typhus, especially when occurring among drunkards, the patient, without much apparent prostration, has active, suspecting delirium, there is total sleeplessness, the muscles tremble, and there is considerable resemblance to delirium tremens. But the moist tongue and skin, and the absence of eruption, usually separate this disease from Typhus, from which it also differs in the manner of its commencement.

We have often to make a diagnosis between Typhus and acute idiopathic or tubercular meningitis. Headache is of a sharper character in meningitis, making the patient cry out with pain, and it persists after delirium has set in, which it never does in Typhus. Instead of the senses being obtuse, as in Typhus, they are usually painfully acute in meningitis, and the countenance has not the look of intense prostration that it commonly has in Typhus. Unilateral symptoms, such as inequality of the pupils, or ptosis, may be seen in meningitis. And in this diagnosis, again, we are guided by the eruption, if it be present, more surely than by any other consideration.

Pathology.—It has been stated that Typhus is the result of a specific poison having the power of reproducing itself in a healthy person submitted to its influence in sufficient quantity and for a sufficient time. An account of the pathology of the disease should explain what this poison is, how it enters the body, and is given off to infect other persons, and how

it operates upon each organ and system to produce the clinical results that have been described. Our knowledge goes but a very little way towards such an explanation. The views of Virchow, Parkes, and Richardson upon the subject afford the most suggestive data, and may be combined into some such account as the following:—

The Typhus poison is a complex organic substance, probably itself in process of decomposition, and capable of producing chemical changes in the albuminous tissues and fluids of the body. Upon these changes the symptoms of the disease depend, and in the course of them a fresh amount of the specific poison is produced. The nature of these chemical changes is not known, but the evidence of their occurrence in the albuminous tissues and fluids comes from the changes observed in the blood and urine, and the alterations seen in the structure of the muscles in fatal cases.

The immediate effect of the mechanical change that is brought about by the poison of Typhus appears to consist in an alteration of the osmotic properties of the blood. Through this alteration many of the phenomena of fever are evolved. The interchange of material between the blood and the alimentary canal is interfered with—an interchange which in health is represented by several pints of fluid daily, and which is of as much consequence to the maintenance of the body as the interchange of gases in the lungs. From this interference arise the dryness of the mucous membrane, the arrested secretion of saliva and gastric fluid, the diminished secretion of chlorine from the body, and probably the febrile phenomena.

Another consequence of the chemical change in the albuminous substances of the body is altered metamorphosis of tissue, firstly, in the way of increase, as we see in the great excretion of urea; and secondly, in point of quality, the alteration of the albuminous substances giving rise to new products in the secretions. In the urine Frerichs shows two abnormal albuminous products. In the skin and mucous membrane the peculiar odor of Typhus, in the absence of chemical proof, serves as evidence of some similar change. And the self-producing poison of Typhus may itself be one of these abnormal elements of secretion.

It has been suggested that ammonia, or a compound related to it, is the actual poison of Typhus. To the writer it appears probable that the matter of this poison is of an organic nature less advanced in decomposition than the stage of ammonia, and that the evidence adduced to support the ammonia theory (even if the fallacy of decomposing matter about the mouth have been sufficiently excluded in the experiments upon which this theory is chiefly based) points rather to the production of ammonia as one of the subsequent changes, excretory in nature, of the altered albuminous compounds.

Morbid Anatomy.—The anatomical changes that can be appreciated in Typhus are few, and the only one that is quite constant is a change in the blood. But changes from the healthy standard are seen in the muscles, in the mucous membranes and glands, in the kidneys, in the lungs, and in the brain, and in other organs.

The body of a Typhus patient is not much emaciated, if the patient die at the time usually fatal, at the end of the second or in the third week. The Typhus maculæ, but not the subcuticular mottling, often persist on the skin after death. Decomposition is generally rapid.

The blood in Typhus is particularly liquid. Drawn from the veins during life it coagulates rapidly, but very imperfectly, the coagulum being large, dark, and soft. Under the microscope it is stated that the corpuscles are crenate and misshapen, and do not adhere into rolls, but run into amorphous heaps. After death the same appearances are observed in the blood. It is either not coagulated at all, or forms in the heart and great vessels large, very soft clots, and rapidly becomes putrid. Chemical analysis of the blood is as yet extremely imperfect. The proportion of fibrine is stated to be diminished and that of the red corpuscles to be increased, while urea and ammonia are said to have been found in the blood. Lehmann's account is that the fibrine corpuscles and albumen are all in excess at first, but that the amount of corpuscles diminishes in the latter stages, causing the blood to have a lower specific gravity. As to the abnormal elements of the blood and the chlorides in it, no sufficient observations have yet been made.

Morbid changes in the muscles of Typhus have been long observed in the heart's tissue. The organ is soft and flabby, and under the microscope the fibres are seen in a state of fatty degeneration, probably identical with that observed in other striated muscles.

Although softening of the voluntary muscles was long ago pointed out by Laennec, and they are known to be often darker and softer than natural, researches into their pathological changes have not been followed out. The elaborate researches of Zenker on the muscles in typhoid were not extended by him to Typhus Fever; but in cases examined by the writer the characteristic changes which were described by Zenker have been several times observed; the granular and

waxen degeneration having been well marked in fibres taken from the rectus abdominis and adductor magnus femoris muscles. When the cases examined had been rapidly fatal, the changes were not seen; but when death had occurred in the third week, or later than this, from some complication, the degeneration was well marked. Several instances are on record of hemorrhages into the voluntary muscles, an occurrence which further points to a muscular change in Typhus similar to that demonstrated by Zenker in typhoid.

The mucous membrane of the stomach is occasionally injected and softened. That of the intestines, particularly of the colon, is not uncommonly inflamed, its vessels being intensely engorged, and soft lymph being sometimes exuded on its surface. This condition is met with when during life there has been much diarrhœa. The agminated glands, and the solitary glands of both large and small intestines, may be found enlarged, especially in children who have them naturally very visible. But under no circumstances is there any deposit in these glands, nor does the ulceration of them which is so constant in typhoid ever happen in Typhus Fever. In those epidemics where dysentery has complicated the fever, the characteristic lesions of that disease have been observed.

Of the glands, the liver and spleen are frequently hyperæmic, large, and softened. The spleen is sometimes pulpy in consistence and enlarged to twice its natural volume; this is seen more when the disease has been fatal at a late period. When the salivary glands have been swollen in Typhus, inflammation and softening of the gland tissue itself is the lesion usually observed; and if the disease is advanced, suppuration and sloughing of the gland substance and the interstitial areolar tissue. Some authors have insisted that the pathological changes in these glands begin in the areolar tissue between the lobules, but in the writer's experience this is not the rule. Under the microscope abundance of oil-globules and of granules, and of pus cells are found, and the gland cells are full of oil-globules. The pancreas is frequently injected, but is not known to suffer any change resembling that of the salivary glands.

The kidneys are not much or often affected by Typhus. Their commonest deviation from health is congestion, the organs being large and somewhat friable. In the rare cases that prove fatal by convulsions, the kidneys are either found the seat of old disease, or they are in some stage of recent engorgement up to actual acute nephritis, or there may be no lesion whatever detected in them.

The bronchitis that is so very common a condition in fatal Typhus offers no appearances after death requiring special comment. The consolidation of the lungs, often met with in most fatal cases, consists either of true pneumonia or (more usually) of hypostatic congestion. In the latter the posterior parts of the lung are dark, non-aërated, friable, with a section that is not granular, as in pneumonia, and from which much dark serosity exudes.

The nervous system commonly shows after death no lesion whatever to account even for intense head symptoms. The utmost change that is usually observed is some fulness of the sinuses, coarse injection of the meninges, and increased vascularity of the brain substance, and none of these conditions reach any intense degree. Occasionally a film of hemorrhage is seen in the cavity of the arachnoid, and the amount of serum in the sulci and ventricles is greater than usual, but neither of these conditions appears to have any connection with the brain symptoms during life. Actual meningitis has, however, been observed in Typhus, in such a way as suggests its occurrence more in some epidemics than in others. It is described by the physicians of the London Fever Hospital in their works, published in 1830, as of no infrequent occurrence; but in the course of the last ten years, meningitis has certainly been very seldom seen there as a post-mortem appearance in Typhus. But in the last few years, a form of fever, akin to Typhus, if not actually identical with it, has been observed in America to be often complicated with meningitis of the brain and spinal cord. And in the present year (1865) a Russian physician, Dr. Kremiansky, describes a hemorrhagic inflammation of the dura mater as a frequent occurrence in persons dying of Typhus in St. Petersburg.

Prognosis and Mortality.—In Typhus, these are affected by nothing so much as by age; but to some degree by temperament and habit, by social position and nature of previous occupation, and also by the characters of the prevailing epidemic.

The rate of mortality from Typhus in a community attacked by it is usually stated much too high, the experience of hospitals, into which few children are received, being taken as a basis of calculation. When every attack, in persons of all ages, is included, the mortality of Typhus is about 10 per cent. But when such cases only as are ordinarily sent to hospitals are considered, the mortality is about 20 per cent. When age is compared strictly with age, however, this difference, either wholly or for the most part, disappears. Between one hospital and another, or between hospitals and cases treated at their homes, there are indeed some apparent differences of mor-

tality, but the causes of such differences (when real and not dependent merely upon age or accident) are not of a nature that any general statement of them can be made.

The death-rate of children under ten years of age, attacked by Typhus, is about 5 per cent.; that of persons over sixty years is 66 per cent. or upwards, of those seized by the disease. Between the two there is a regular gradation of fatality. For example, at Greenock, the death-rate, at seven periods of life, was recently found to be, under ten years, 5 per cent.; between ten and twenty, 8·6 per cent.; between twenty and thirty, 15·6 per cent.; between thirty and forty, 21·5 per cent.; between forty and fifty, 42 per cent.; and over fifty years, 66·6 per cent.; the mortality increasing with each decade of age that the patient had reached. The death-rates of the London Fever Hospital, analyzed in detail by Murchison, gave figures corresponding in the main with these, but all of them slightly higher through the severity of the cases that are presented to that institution. The difference in mortality, according to age, is so great and so universal, that the caution may well be given that no comparison between different methods of managing Typhus can have the slightest value which does not accurately allow for this overshadowing influence.

Bulky, lymphatic, and fat people are more likely to die than those of a different conformation when they are attacked with Typhus. Negroes, treated in the London Fever Hospital, have been observed to have the fever more severely than whites. People of a better class of life, though seldomer attacked, are believed to experience a larger mortality than the poor. Habits of intemperance very seriously add to the unfavorable prognosis.

Occupational differences only affect the prognosis of Typhus in so far as they have involved extreme exhaustion and fatigue, persons who are attacked under circumstances of that kind usually having a high mortality. Overworked soldiers, doctors and nurses, for example, get the disease with peculiar severity. And if the patient try to keep about, going on with his work until he takes to his bed from sheer inability to stand, he materially diminishes his chance of recovery.

The conditions occurring in an attack of Typhus, which gives especial gravity of prognosis, are as follow:—Very abundant rash, with spots scarcely affected by pressure; considerable duskiness of surface; a high maximum temperature, as 107° or above; continuous rise of temperature up to the end of the first week, a sudden great rise of temperature in the third week, this being, it is stated, of fatal significance; very weak pulse, with inaudible first sound of the heart; very rapid pulse, death being almost certain if in the adult the pulse exceed 150; lung complications of all kinds make the prognosis bad according to their amount; early delirium; severe and active delirium, with complete sleeplessness; profound coma, and especially coma vigil; intense prostration and subsultus; convulsions, which are almost certainly fatal; albumen in the urine; obstinate refusal of food; vomiting; uncontrollable diarrhœa. Any of the erysipelatous conditions noted as occurring towards the end of the disease contribute to reduce the patient's chances of recovery.

THERAPEUTICS.—Typhus Fever, like other diseases of its class, cannot be cured nor its duration shortened by any means at present known to medical science. Its symptoms may be combated, and its complications may be treated, while the patient's strength is supported through the time of the fever, but we know of no way of encountering the specific disease. Upon a full recognition of this truth, the treatment of a case of Typhus will be most satisfactorily based. If we propose to ourselves to give the patient the best possible opportunities of recovery, our treatment will be more successful, than if we direct our efforts to cutting the disease short by any supposed methods of cure.

In a disease which lasts two or three weeks, in which the metamorphosis of tissue is increased, and in which ordinary food cannot be taken, the patient must be kept up by nourishment appropriate to his new condition, or he will die, as a healthy person deprived of food for the same length of time would die. The essential part of the management of Typhus consists in giving this appropriate nourishment, and in preventing the patient dying from the want of it while the curative processes of nature are going on.

The character of the nourishment to be given requires some detailed consideration. In the early stages of the fever, if the patient have appetite, he may be allowed anything not positively noxious that he has a fancy for. As his dislike for food increases, he will still consent to take liquids and sick-room delicacies. But soon there comes a time, in every severe case of fever, when everything but cold water is distasteful, and when food has to be administered like so much medicine to the unwilling patient. At this time, the digestive functions are in more or less complete abeyance, and the nutriment given must be such as requires the simplest processes for its assimilation. Foremost among nutriments of this kind, experience has put beef-tea and broths, milk,

eggs, and alcoholic drinks.[1] Bread, arrowroot, jellies, are other suitable articles; and the nurse who can manage a good deal of variety in the choice and combination of such things as these, does much for her patient's chance of recovery. Samples of such variety are Gillon's meat-juice; vermicelli in beef-tea; chicken or veal broth; mutton broth with rice or bits of toast; eggs in custards or beaten up with milk or with wine; blancmange of isinglass or ground rice; syllabubs or wine-whey; barley-water or thin arrowroot with milk; weak tea or coffee with milk.

For drinks, lemonade, soda-water, currant-water, cold weak tea without sugar or milk, or any of these iced, may be allowed at the patient's choice. Often food is taken cold when hot is refused. But even of this light diet a little only can be taken at once, and it therefore becomes desirable that it should be given frequently. Every two or three hours the patient should be fed, and if he be in a drowsy state he should even be roused up to take his nourishment.

But of these means of giving support, there is none more important than alcoholic drinks judiciously used. It is not every patient that requires alcohol; children rarely do, and about half the adult cases admitted into hospital may be treated without any. But it is especially in two classes of patients that we need to give stimulants: those who cannot take a sufficient quantity of other kinds of nourishment, and those who are in health habituated to the use of strong drinks. Besides the use of alcohol as an aliment, it has also a medicinal effect upon the nervous and circulatory systems, and its full employment will much depend upon whether this effect is desirable. The cases in which alcoholic stimulants are most serviceable are (1), in old people almost universally; (2), cases of great prostration, with low delirium and coma; (3), cases where the pulse is very compressible and the first sound of the heart feeble, also when the pulse intermits and usually when it exceeds 120 in frequency; (4), cases where the extremities are cold and the surface is livid; (5), where there is much congestion of the lungs; (6), where there is any erysipelatous complication. In a great many cases of Typhus alcohol is unnecessary, and appears to do actual harm when there is violent maniacal excitement at an early stage, and also with young people in whom, without notable depression, there is much bronchitis, or in whom true pneumonia can be diagnosed. Alcohol is rarely wanted before the appearance of the eruption, and is most needful in the second or third week, as the patient is approaching the crisis of his disease. For ordinary cases requiring alcohol, the strong wines are best adapted, while lighter wines with water form excellent drinks. Beer is a very good form of giving alcohol with other nourishing principles, and it is often craved for by the patient. Severe cases, particularly in old persons and in drunkards, require spirits, which may be given mixed with beef-tea, with milk, or with eggs. A moderate allowance to an adult, suffering under pretty severe Typhus, with dry tongue, moderate delirium, and weak pulse of 120, would be a bottle of claret or half a bottle of sherry daily. A bad case, with livid features, tremulous muscles, much low delirium, with coma, and a very weak pulse of 140 or 150, may often have 12–20 ounces of brandy or whisky daily distributed in hourly doses. It often happens that in such cases a patient for whom there might otherwise be a chance, obstinately clenches his jaws against his nourishment, or is made sick by it; it is then sometimes possible to tide him over the time of crisis by frequent enemata of beef-tea and brandy, which are usually well retained, even if there have been some tendency to diarrhœa.

[The *manner of administration* of food and stimulants in typhus is very important. After the first few days, if not from the start, milk, beef-tea, or some other concentrated nutriment, should be given in small quantities every two or three hours; in cases of extreme prostration, every hour, day and night. Having had experience of an attack of typhus, when resident in a Hospital, I very well remember the distressing sense of sinking felt on awakening after a couple of hours of sleep; relieved for the time by a tablespoonful or two of concentrated liquid food. But for watchfulness with this kind of care of the patient (especially through the weakest time of the twenty-four hours, between midnight and morning), not a few will, as it were, slip through the fingers.

[1] The writer has no intention to side in the controversy concerning the food character of alcohol. He accepts the evidence that much ingested alcohol is got rid of by the excretory organs, or is retained for some time in the tissues after the manner of many medicines. But with food in its widest sense, as what keeps up the vital functions, the physician will have little hesitation in classing alcohol, who has observed the common case of an habitual tippler maintaining for years a fair standard of bodily health upon a quantity of other nutriment wholly insufficient by itself to maintain such health. And to such a case a fever patient offers some resemblance. He, too, may not be able to take enough of other food to maintain him, but alcoholic drinks will help him not to starve. And thus the writer judges them to have a food value apart from their medicinal action.

When alcohol is needed, it does the most good given in milk; say one tablespoonful of whisky to two of milk, or, in very feeble cases, half and half.—H.]

But though what has been said is, in the writer's experience, the essential part of the treatment of fever, there are many symptoms and complications that can be met by medicinal agents. The thirst is best relieved by acidulous draughts, and there is no better medicine for slight cases of fever than twenty drops of dilute hydrochloric acid in an ounce of water. The headache, sleeplessness, and delirium are very frequently lessened, even though there should be a good deal of suffusion of the eyes, by opium; the writer has constantly given five minims of laudanum every four hours, or else a night dose of fifteen minims, with considerable advantage to these symptoms. He has avoided opium when the pupils are very small, when there is coma, and when there are serious lung complications; but with these exceptions, he finds no contra-indications to the use of the drug. If opium be given with the object of soothing violent delirium, it should be in full doses at night, and not in small frequent doses: but if possible it is better not then to use opium. Combined with a small quantity of tartar emetic, opium has an increased power of relieving headache and of inducing sleep. When it is desired to use a sedative, but to avoid opium, good results have often followed from a grain or more of extract of cannabis indica given at night. Strong coffee is said to have relieved headache; the writer has seen no effect from it, either upon headache or coma, in the few cases where he has employed it. Cold lotions to the shaven head and blisters to the forehead are each of use in many cases of severe headache, and they are means that are especially applicable in the cases just mentioned where opium must not be used. In early furious delirium, two or three ounces of blood have been taken from the temples with good results; doubtless if meningitis were diagnosed, this would be right practice then also. When the patient's delirium causes him to leave the bed, there is no means of restraining him equal to the care of an experienced nurse, but there is no objection to mechanical restraint, for if it be effectually applied the patient often ceases to struggle and so saves his strength. From deep coma patients may sometimes be roused by blisters to the forehead and nape or to the shaven scalp. Subsultus and tremors are said to be peculiarly controlled by camphor and musk. Among stimulating remedies that may be used, along with much alcohol in cases of great prostration, spirit of chloroform and turpentine are the most valuable.

The bowels had best be kept open once a day, but slightly confined rather than purged. Any but very gentle laxatives are apt to cause weakening diarrhœa. Should this from any cause be present, draughts of sulphuric acid or of chalk and catechu may be given; and when diarrhœa is severe or obstinate, acetate of lead in draughts; or sulphate of copper in pill, are most useful, along with small doses of opium, if there be no reason against it. Starch and opium (fifteen minims of laudanum) injections are also of great use. Vomiting is best checked by ice, lime water, or soda water, and by bismuth; sometimes by a sinapism to the epigastrium; if it persist in spite of these remedies, it is well to let the stomach have complete rest for a while, supporting the patient by nutritive injections.

For lung complications of all kinds, one of our most valuable remedies in Typhus is carbonate of ammonia. The bicarbonate (formed by exposing powdered sesquicarbonate to the air till its pungency is gone) is less irritating to the parched mouth, and can be given in larger doses than the monocarbonate. Senega assists the expectorant action of ammonia, and may be given, except for its nastiness, in all cases complicated with bronchitis or with consolidation of the lungs. Another extremely useful remedy for congestion of the lungs, especially in old people, is turpentine, fifteen drops in mucilage. And with these internal remedies it is always right to use counter-irritants; mustard poultices, often repeated, to the back and sides of the chest, being the best form of them.

As for the urinary organs, it is important to be on the watch against retention of urine, and to relieve the bladder duly by catheter. Slight albuminuria itself calls for no special treatment, beyond contra-indicating opium, and being, it is thought by some, a reason for not giving alcohol very freely. When there is much albumen in the urine, or when convulsions have occurred, it is right to give gentle saline purgatives that may act also as diuretics, to use mustard or dry cups to the loins, and to get the skin to act by means of the hot-air bath.

Where swelling of the salivary glands occurs, the chance of their resolving without suppuration has seemed to be increased by blisters over them at an early stage. Cotton-wool may be applied over the swelling, and when the formation of pus cannot be avoided the abscess should be poulticed continuously and opened as early as ever fluctuation can be detected in it. It is of great consequence to give extra food to the patient as soon as a salivary gland is observed to swell. Eggs, fish, panada, and more stimulants should be given without any limit but his ability to swallow them. Erysipelas supervening

on Typhus is best treated by extra food and stimulants, by the tincture of iron internally, and by wrapping the affected parts in cotton-wool.

In the management of a case of Typhus it is of great consequence to place the patient in the best hygienic conditions, and if he has been attacked in the close crowded rooms where the disease mostly occurs, his removal to a properly constructed hospital should be insisted on. An ample supply of fresh air of even temperature, of clean linen, of soft but cool bedding, and the services of an experienced nurse, are parts of the treatment as essential as the prescriptions of the doctor. Of hygienic treatment, quiet, cleanliness, frequent sponging, and occasional changes of posture to avoid lung congestion and bed-sores, are the most worthy of mention.

As soon as the patient passes the crisis of the fever, and regains his appetite, he may be allowed any article of food that is good for him in health, and may eat and drink liberally. It often happens that, with a tongue still dry and brown, and only just moistening at the edges, the patient asks for meat, and if it is given him finds no ill effects from it. But as a rule it is certainly safer to keep him on light puddings and fish until his tongue has got pretty clean and moist. Beer is generally relished in convalescence more than wine, of which the palate is tired, and good ale or stout may be substituted for a great part or for the whole of the other stimulants. The amount of stimulants must be brought to the standard required in health by degrees, and not suddenly. When during convalescence the patient remains childish in intellect, or wandering in his mind, it is a reason for giving plenty of food and wine, not for diminishing his allowance.

Of the prophylactic measures to be used against Typhus little need be said, as they consist almost entirely in an avoidance of the predisposing causes that have been enumerated. Persons in attendance on the sick should not be overworked, or deprived too much of their natural rest and exercise, and they may be further guarded against the reception of the fever by the use of disinfectants, of which fresh air and cleanliness are incomparably the most important. Lime whiting and repapering (after lime whiting) of infected rooms, stoving the bed and bedding, boiling the patient's linen, or soaking it in water impregnated with chloride of lime, and the use of this substance or carbolic acid in the water employed for sponging his body, are other means that should be employed for avoiding contagion.

Varieties.—The varieties of Typhus are few, and consist chiefly in different degrees of severity. One epidemic may differ from another in its liability to special complications, to dysentery, or to cerebral inflammation, for example; or in intensity, in this respect being affected by the average age of the community in which it occurs, and by the degree to which the predisposing causes of the diseases are in operation. But of such varieties no further consideration is here required.

RELAPSING FEVER.

By J. Warburton Begbie, M.D.

This, its familiar name, has been applied to one of the forms of continued fever, on account of its most characteristic and peculiar feature.

The disease may be defined as follows:—

Definition.—A contagious[1] disease, rarely appearing, except as an epidemic; marked by its sudden invasion, the pyrectic symptoms continuing till about the fifth or seventh day, when, after the occurrence of a critical evacuation, their abrupt cessation occurs. There succeeds an interval of complete freedom from fever, followed by sudden relapse on the fourteenth day from the commencement of the original attack. The condition of pyrexia is again terminated by a crisis on or about the third day of the relapse, and for the most part convalescence ensues. Not very infrequently a second, with increasing rarity a third, fourth, and even a fifth relapse, has been noticed.

History, Nomenclature, and Bibliography.—In 1843 an epidemic of fe-

[1 The application of this term is questioned by some, at least, of those who have studied the disease.—H.]

ver appeared in Edinburgh, Glasgow, and other of the larger towns of Scotland, which, although at first believed to present characters previously undescribed, was soon recognized as being similar to the fever which had prevailed in the former city during the years 1817–18, and likewise to the fever which during these years and the one subsequent, 1819, as well as many previous years, had occurred in Ireland. Carefully observed in 1843, and very ably described by several Scotch physicians, this same fever during a later, though by no means so extensive prevalence, in 1847–48, attracted the attention of other accurate observers, both in Scotland and England. Since the disappearance of the last-named epidemic the disease has been very little seen. In 1851 Dr. Murchison informs us that in London, as well as in Glasgow, there was a considerable increase of Relapsing Fever; but since 1855, this excellent writer on fever remarks, he has reason to believe that not a single case of Relapsing Fever has been observed in either of these cities.[1] As regards Edinburgh, a very competent authority, Dr. W. T. Gairdner, has stated that he has not seen a single case distinctly referrible to this type since 1855.[2] The writer is able to offer an abundant confirmation of the latter statement, for, during his ten years' service as physician in the Royal Infirmary, dating from May 1855, while having at all times charge of fever-patients, he has never once encountered a case bearing any resemblance whatever to the Relapsing Fever.

Several of the physicians who have enjoyed the most extensive opportunities of observing the more recent epidemics of Relapsing Fever, have occupied themselves with an inquiry into its history; it may therefore be expedient here, before entering on the consideration of the phenomena presented by the disease, to make a few observations on the former subject, while indicating at the same time the different names by which it has been described, as well as the sources from which the most reliable information regarding Relapsing Fever is to be drawn. In one of the important discussions which took place in the Medico-Chirurgical Society of Edinburgh during the prevalence of fever in 1844, the late Dr. Robert Spittal called attention to the interesting fact "that the present epidemic seems to be exactly the same in all its important features as an epidemic described by Hippocrates as having occurred in the island of Thasus, off the coast of Thrace." The chief features of resemblance between the ancient and the modern epidemics are the invariable occurrence of relapses, the marked character of the crisis, and the frequent association with the more ordinary events in the disease, of copious perspirations,[1] hemorrhages, particularly epistaxis,[a] jaundice,[b] splenic enlargements,[c] and in women the tendency to miscarry.[d] A simple mention of the occurrence of one or more relapses in the progress of continued fever has been made by many writers in their descriptions of different epidemics. This circumstance alone, it is scarcely necessary to observe, does not admit of such cases—which were merely exceptional in these visitations of fever—being considered examples of the form of fever now under consideration. Thus, Dr. Srother, in his account of a fatal fever which prevailed for two years in London, mentions the occurrence of relapses as frequent. "Perhaps," he remarks, "we may find reason to lay some blame on the air for the frequent relapses."[2] Dr. Lind,[3] also, in treating of the contagious Typhus of the fleet, alludes to the same. "Many," he says, "relapsed." Joseph Frank, in his learned account of adynamic fevers, refers to the occurrence of relapses, but that these were not often witnessed, may be gathered from his words, "raro recidiva morbi timenda."[4] These quotations will suffice to establish, firstly, the circumstance that occasional relapses in cases of continued fever had long been observed and described; and secondly, that the fevers thus indicated were certainly not the disease we are now discussing, for in it relapses are not merely occasional, nor even frequent, but invariable. We come then to the well-known work of Dr. Rutty,[5] and in it there is

[1] A Treatise on the Continued Fevers of Great Britain, by Charles Murchison, M.D. London, 1862. P. 298.

[2] Clinical Medicine, by W. T. Gairdner. Edinburgh, 1862. P. 158.

[1] The London and Edinburgh Monthly Journal of Medical Science, vol. iv. for 1844, p. 177.

Καὶ ἐφίδρουν. [a] Ἔστι δ' ὅτι ἐκ ῥινῶν ἡμορράγησε. [b] Ἔστι δ' οἷσι ἴκτεροι ἐχταίοισι. [c] Αὐτίκα δὲ σπλὴν ἐπήνθη.

[d] Ὅσι δὲ ξυνεχύρησε ἐν γαστρὶ ἐχούσῃσι νοσῆσαι, πᾶσαι ἀπέφθειραν ἅς καὶ ἐγὼ οἶδα.—Epidemiorum Hippocratis, Liber Primus, Sectio Secunda, Status Tertius.

[2] Practical Observations on the Epidemical Fever which hath reigned so violently for these Two Years past, and still rages at this present time, by Edward Srother, M.D. London, 1792. P. 121.

[3] An Essay on the most effectual Means of preserving the Health of Seamen in the Royal Navy, and a Dissertation on Fevers and Infection, by James Lind, M.D. London, 1779. P. 190.

[4] Praxeos Medicæ Universæ Præcepta, Auctore Josepho Frank. De Febribus Typhodibus, Partis Primæ, Volumen Primum. P. 214.

[5] A Chronological History of the Weather and Seasons, and of the prevailing Diseases in Dublin, by John Rutty, M.D. London, 1770. Pp. 75, 90.

afforded ample proof of the existence of Relapsing Fever in an epidemic form in Dublin, ten years after the London epidemic described by Srother. The former writer, in giving an account of the summer of 1739, remarks, "The latter part of July, and the months of August, September, and October, were infected with a fever, which was very frequent during this period, not unlike that of the autumn of the preceding year, with which compare also the years 1741, 1745, and 1748. It was attended with an intense pain in the head. It terminated sometimes in four, for the most part in five or six days, sometimes in nine, and commonly in a critical sweat; it was far from being mortal. I was assured of seventy of the poorer sort, at the same time in this fever, abandoned to the use of whey, and God's good providence, who all recovered. The crisis, however, was very imperfect, for they were subject to relapses, even sometimes to the third time; nor did their urine come to a complete separation. Divers of them, as their fever declined, had a paroxysm in the evening, and in some there succeeded pains in the limbs." Again, in describing the summer of 1741, the same writer, Rutty, observes: "It seems also not unworthy of notice, that through the three summer months, there was frequent here and there a fever, altogether without the malignity attending the former (an adynamic fever which the author had already described), of six or seven days' duration, terminating in a critical sweat, as did the other also frequently; but in this the patients were subject to a relapse, even to a third or fourth time, and yet recovered." Huxham, in bearing a strong testimony to the value of bark in the advanced stage of "the slow nervous fevers," speaks of fevers which "are frequently attended with dangerous relapses."[1] But neither in the work from which this statement is quoted, nor in his other treatise on epidemics, referred to below, is there any account of Relapsing Fever. Dr. John Clark, in 1777, observed at Newcastle a fever "the duration of which was uncertain, and some relapsed into the fever." This disease about the eighth or tenth day presented a white or red miliary eruption, and sometimes a more universal red rash, resembling the measles; clearly it, too, was other than Relapsing Fever.[1] Drs. Barker and Cheyne, in the concluding paragraph of the first chapter of their interesting work on Epidemic Fevers of Ireland, indicate the occurrence in the very earliest years of the present century of a fever closely resembling, if not identical with, Relapsing Fever. "Certain it is," these authors remark, "that the fever of 1800 and 1801 very generally terminated on the fifth or seventh day by perspiration; that the disease was then very liable to recur; that the poor were the chief sufferers by it; and that it was much more fatal amongst the middle and upper classes in proportion to the number attacked."[2] The widespread epidemic fever of 1817, and the two succeeding years, was in all probability largely composed of Relapsing Fever. Our chief knowledge of the fever as it then occurred, is gathered from the works of Barker and Cheyne, Harty, Bateman, and Welsh. The progress and distribution of the epidemic has been ably sketched by Mr. Murchison,[3] who has in all probability correctly inferred that the proportion of typhus to the relapsing cases was greater towards the close of the epidemic from the circumstance that the rate of mortality increased at many places with the advance of the disease. Any accurate account of the distinctions between the two forms of fever composing the epidemic in question is not, however, to be expected, and indeed cannot be found. Dr. Christison, to whom, as Mr. R. Christison, Dr. Welsh in the preface to his work[4] refers, candidly admitted in 1844 that, "at the time when he had observed the same fever twenty years ago, it was the general impression that it could produce common typhus, and *vice versâ*." At the same time, Dr. Christison remarked that the Fever of 1843–44 was not a new one; it had been described by himself as "Synocha" in his article on fever in Dr. Tweedie's Library of Medicine.[5] Allusion has al-

[1] An Essay on Fevers and their various Kinds, as depending on different Constitutions of the Blood; with Dissertations on Slow Nervous Fevers, &c., by John Huxham, M.D. London, 1750. P. 87.

For a further notice of Epidemic Fevers, see the same author's work, entitled "Observationes de Ære et Morbis Epidemicis ab Anno 1728 ad Finem Anni 1737. Plymuthi factæ." London, 1752.

[1] Observations on Fevers, especially those of the Continued Type, &c., by John Clark, M.D., one of the Physicians to the Newcastle Dispensary. London, 1780. Pp. 131, 132.

[2] An Account of the Rise, Progress, and Decline of the Fever lately epidemical in Ireland, together with Communications from Physicians in the Provinces, and various Official Documents, by F. Barker, M.D., and J. Cheyne, M.D. In 2 vols. London and Dublin, 1821. Vol. i. p. 20.

[3] Loc. cit., p. 36.

[4] A Practical Treatise on the Efficacy of Blood-letting in the Epidemic Fever of Edinburgh, illustrated by numerous Cases and Tables, extracted from the Journals of the Queensberry House Fever Hospital, by Benjamin Welsh, M.D. Edinburgh, 1819.

[5] Discussion in Medico-Chirurgical Society of Edinburgh, January 3, 1844. Monthly Journal of Medical Science, p. 177.

ready been made to the circumstance of the fever of 1843–44 being at the first regarded as a new disease. The statement of Dr. Alison on this point is quite distinct. Writing even in 1847, that able and excellent man remarked, "The epidemic fever of 1843 is now generally admitted to have been a new pestilence, hardly anywhere seen in England, and not known in Scotland before that year, extending rapidly and generally in Scotland, but fortunately causing in itself no great mortality."[1] But although no correct distinction was drawn between the two forms of continued fever which undoubtedly composed the epidemic of 1817, 1818, and 1819, it was otherwise during the succeeding epidemic of 1826. Dr. O'Brien in particular, who published an account of the epidemic as witnessed in Dublin in 1826–27, wrote: "At the commencement of the epidemic two species of fever were distinguishable in the wards of this hospital, which, to use the words of Sydenham, we shall call the fever of the old and the fever of the new constitution. The first was the ordinary typhus of this country, marked by its usual protracted periods, running on to the eleventh, fourteenth, seventeenth, or twenty-first days. This species of fever was inferior in numerical amount to the other, but far more fatal. . . . . The other species of fever, or that of the new constitution, which constituted the bulk of this epidemic, was one of short periods, terminating in three, five, seven, or nine days, but the second of these periods was the most frequent. . . . . The patient was destined, perhaps, to be harassed by one, two, or three relapses, which prolonged the whole duration of his illness beyond that of the most protracted typhus—in fact, the liability to frequent relapses was one of the most striking characteristics by which this fever was distinguished from all previous epidemics, at least which happened in our time."[2] In respect to the statement of O'Brien on the mortality of the epidemic at different stages of its progress, Dr. Murchison, by a reference to statistical facts, has ably shown that in the latter part of its continuance the cases of Relapsing Fever had in all likelihood greatly diminished. This result is further corroborated by the statement of Dr. Alison regarding the Edinburgh fever of the years 1826–27; the mortality in the latter year, he has stated, exceeded that of the former.

The epidemic of 1826–27 ended, Relapsing Fever was probably absent from Great Britain till the year 1843, or the very close of the preceding year. At that time there appeared in Scotland an epidemic, of which excellent accounts were published by Drs. Alison, Craigie, Halliday Douglas, and Henderson; while the fulness and accuracy of the treatises written about the same time by Drs. Cormack[1] and Wardell[2] justly entitle them to be styled admirable histories of that epidemic. Three complete years separated the epidemic just referred to and its successor; although, during that period, embracing the years 1844–46, "a few cases of Relapsing Fever continued to be observed, both in Ireland and Britain."[3] In 1847–48, and this is the last occasion in these Islands, Relapsing Fever was again very prevalent, the particular epidemic being, as aforetime, constituted partly of typhus and partly of the shorter fever—cases of the latter, about the commencement of the unusual prevalence of fever, being in considerably greater proportion than those of the typhus. This epidemic of fever prevailed in England, as well as in Ireland and Scotland. In the former, it is probable that throughout its entire progress, cases of typhus were greatly more frequent than in Ireland, and decidedly more so than in Scotland. A very careful observer and instructive writer on the Irish fever has indeed stated, that "cases of genuine typhus were, through the whole epidemic, very rare. Occasional cases did occur, and these became more numerous with the advance of the epidemic." It is right to note here that Dr. Henry Kennedy's opinion, just quoted, is not acquiesced in by all authorities; for example, Dr. Lyons, in his valuable work on fever, makes the following observation: "While I admit the frequent occurrence of Relapsing Fever in Ireland, I must be allowed to record here my protest against the statements recently circulated on very insufficient data, that Relapsing Fever constituted the large majority of the cases of the famine fevers of Ireland. The contrary of this I believe to be the case; and having had large and extended experience in the last great famine visitations of Ireland, 1846, 1847, 1848, I can certify that the maculated

[1] Observations on the Famine of 1846–47, in the Highlands of Scotland, and in Ireland, by William Pulteney Alison, M.D. Edinburgh, 1847, p. 9.

[2] Medical Report of the House of Recovery and Fever Hospital, Cork Street, Dublin, for the Year ending 4th of January, 1827, by John O'Brien, M.D., in Transactions of the Association of Fellows and Licentiates of the King's and Queen's College of Physicians in Ireland. Dublin, 1828. Vol. v. pp. 526, 209.

[1] Natural History, Pathology, and Treatment of the Epidemic Fever at present prevailing in Edinburgh and other Towns, &c. London, 1843.

[2] The Scotch Epidemic Fever of 1843–44. London Medical Gazette, 1846–47.

[3] Murchison, loc. cit. p. 295.

typhus was the disease which chiefly prevailed; while the Relapsing Fever presented itself only at the close of the great typhus visitation."[1] Of the epidemic fever of 1847–48, many valuable accounts have been written, including those of Dr. Robert Paterson,[2] of Leith, and Dr. William Robertson.[3]

The works and papers which have been already referred to, are among those which contain the most satisfactory information regarding the Relapsing or Short Fever.[4] Other sources will be indicated in the sequel; it may however be stated, once for all, that in the exhaustive treatise of Dr. Murchison, the fullest, as well as the most reliable information respecting this and the other forms of continued fever which occur in Great Britain is to be found. This division of our subject may be closed with a notice of the different names under which the Relapsing Fever has been described.

The *Synonyms* are, indeed, numerous, and have been suggested by a consideration of the various particulars in regard to the disease itself, or its special epidemic prevalence—Short Fever; Fever of Short Periods; Five or Seven Days' Fever; a Five Days' Fever, with Relapses; Remittent Fever; Febris Becurrens; Das recurirende Fieber; Fièvre à Rechute; Synocha; Relapsing Synocha; Inflammatory Fever; Mild Yellow Fever, Remittent Icteric Fever, Bilious Relapsing Fever; Gastro-hepatic Fever; Famine Fever; Die Hungerpest; Fever of the New Constitution; Miliary Fever; Typhinia; Epidemic Fever of Scotland, or of Ireland; and according to the particular years in which it prevailed, as of 1843–44, 1847–48.

Geographical Distribution.—From the foregoing historical statement, it will have been gathered that in Ireland and Great Britain, the former more especially, while in Scotland more than in England, epidemics of Relapsing Fever have chiefly occurred. But not only has this form of fever been more prevalent in Ireland than elsewhere, but it has been clearly shown by Drs. R. Paterson,[5] Wardell,[6] Ormerod,[1] and more recently, and still more decidedly, by Dr. Murchison,[2] that the Irish resident in Great Britain suffers in greater proportion than either English or Scotch from Relapsing Fever. Notwithstanding the statement now made, it is to be borne in mind that the epidemic of 1843 was essentially a Scotch fever, originating in Scotland, and showing little tendency to extend to any great distance. Beyond Great Britain and Ireland, Relapsing Fever has been seen in the Silesian provinces of Prussia and Austria; while an able reviewer, in noticing the account of the Silesian fever of 1847, by Virchow—deputed by the Prussian Government to investigate it—Bärensprung, Dümmler, and Suchanek, has drawn a most interesting parallel between the Irish and the inhabitants of Upper or Prussian Silesia.[3] "During the summer months of 1855," writes Dr. Lyons, "this form of disease was pretty common amongst the British troops in the Crimea."[4] It was not fatal, for the same author observes, "No fatal cases came under our observation in the Crimea." Of its existence in Russia we have recently become aware, for the late exaggerated reports regarding the prevalence of a deadly pestilence in that country and Siberia, have now been shown to point to the occurrence of the Febris Recurrens, or Relapsing Fever of Ireland and Great Britain, associated, as heretofore in our home experience of it, with Typhus, probably, also, with Enteric Fever. Finally, as regards America, it is evident from the analysis of fifteen cases of Continued Fever, characterized by Relapses, by Dr. Austin Flint, that Relapsing Fever has been observed in the Western hemisphere, but the information supplied by that able writer does not permit us to conclude that this form of fever has ever originated in America; the cases detailed by him may have owed their occurrence, as the much larger numbers occurring in Canada undoubtedly did, to the Irish immigration. Dr. Flint's own statement is as follows: "The conclusion seems unavoidable, that the cases of fever, characterized by relapses, among those which came under my observation in 1850–51, presented the distinctive traits attributed to Relapsing Fever, sufficiently marked to entitle them

[1] A Treatise on Fever, or Selections from a Course of Lectures on Fever, by Robert D. Lyons, M.B., T.C.D., &c. London, 1861. P. 103.

[2] Account of the Epidemic Fever of 1847–48 in Edinburgh. Edinburgh Medical and Surgical Journal, No. 177, 1848.

[3] Notes on the Epidemic Fever of 1847–48. Edinburgh Monthly Journal of Medical Science, vol. ix., 1848.

[4] The disease is well described in Dr. Tweedie's Lectures on Fevers. London, 1862.

[5] Loc. cit.

[6] Loc. cit.

[1] Clinical Observation on the Pathology and Treatment of Continued Fever, by E. L. Ormerod, M.B. London, 1848.

[2] Loc. cit.

[3] The British and Foreign Medico-Chirurgical Review, vol. viii. London, 1851. Article, Diagnosis of Fevers, p. 29.

[4] Lyons, op. cit. p. 106, also p. 108. See also Relapsing Fever, in Science and Practice of Medicine, by William Aitken, M.D., vol. ii. London, 1864.

to be ranked in the class of cases which have been described by different observers as a peculiar form of continued fever."[1] [Dr. Meredith Clymer recognized Relapsing Fever in some Irish emigrants brought to the Philadelphia Hospital in 1844.[2] Dr. Dubois reported its occurrence, also mostly among emigrants, in New York, in 1847–48. During the summer of 1870, several hundred well-marked cases of it were observed, in localities presenting unsanitary conditions, in New York and Philadelphia.[3] Between April and November of that year, 517 cases were admitted into the Philadelphia Hospital; of which 89 were mortal. The whole number of deaths reported to the Board of Health in Philadelphia was 162; of which 107 were of the colored race. 26 per cent. of the latter, affected with this disease, died; while among white patients, but 5 per cent. of those attacked died.—H.]

Etiology.—Relapsing Fever affects persons of both sexes and all ages; the statistics of the London Fever Hospital, quoted by Dr. Murchison, making it probable "that the proportion of the young to the aged is greater than in the case of typhus."[4] The special season of the year has little, if any, influence on the prevalence of Relapsing Fever, neither is there proof of any particular occupation or employment predisposing to the disease. This much, however, has long been known; and the observation of Relapsing Fever when more recently epidemic in England and Scotland, as well as in Ireland, has strengthened the belief that this disease is peculiarly the fever of the vagrant and the unemployed. The contagious nature of Relapsing Fever scarcely admits of doubt. Two eminent authorities, and these only, have expressed the opinion that this form of fever is non-contagious. One of them, Dr. Cragie, has, indeed, almost admitted the contagious nature of Relapsing Fever: "This," however, he adds, "is rather a presumption than a well-founded inference." The other non-contagionist is Virchow; but, as Dr. Murchison has shown, the importance justly attached to the opinion of this eminent observer is necessarily diminished from the consideration that his experience of the disease was limited to a single fortnight; he left Berlin on the 20th February, 1848, and returned to it on the 10th of March. An examination thus conducted must have been cursory and incomplete. The opinion, moreover, expressed by Virchow, was not shared in by the other medical men of Silesia; all engaged in practice there believed the epidemic malady to be contagious. In all the epidemic visitations of Relapsing Fever, to which reference has already been made, but more especially in those of 1817, 1818, and 1819, of 1843–44, and 1847–48, precisely the same facts which have been held as sufficient to establish the contagious nature of such diseases as Typhus, Scarlatina, and Morbilli, were observed. Physicians engaged in the daily observation of the epidemic fever for many months together, unanimously formed the opinion that the Relapsing Fever propagated itself by contagion. Concerning the earliest mentioned of these epidemics, we find Dr. Welsh writing as follows: "When acting as clerk to Dr. Hamilton in the Royal Infirmary, in the course of four months, my three colleagues, two of the young men in the apothecary shop, two housemaids, and thirteen or fourteen nurses, caught the disease, and the matron and one of the dressers died of it. Since I left the infirmary, three more of the gentlemen acting as clerks, one of the young men in the shop, and many more of the nurses, have caught the infection, but the number I do not know. In this hospital (Queensberry House), since it was opened on the 23d February, 1818, my friends, Messrs. Stephenson and Christison, the matron, two apothecaries in succession, the shop-boy, washerwoman, and thirty-eight nurses have been infected; four of the nurses have died. With the exception of two or three nurses who have been but a short time in the hospital, I am now the only person in this house who has not caught the disease, either here or at the infirmary, within the last eight or ten months. Several students, whom curiosity led too near the persons of the patients, might be adduced as additional evidence. When it begins in a family, we always expect more than one of them to be affected; I could mention instances of four, five, six, and seven, being sent to the hospital out of one family; eight, nine, and ten, out of one room; twenty and thirty out of one stair; and thirty and forty out of one close; and this all in the course of a few months."[1] The contagious nature of the epidemic fever of 1843–44 is thus insisted on by Dr. Wardell: "Most of the medical officers connected with the Edinburgh Royal Infirmary and additional fever hospitals were seized with it; eight of the resident and clinical clerks in quick succession became affected, and, out of that

[1] Clinical Reports on Continued Fever, based on an Analysis of One Hundred and Sixty-four Cases, &c. &c., by Austin Flint, M.D. Philadelphia, 1855. P. 374.

[2] Fevers, &c., by M. Clymer, M.D., Philadelphia, 1846, p. 99.

[3] Philada. Med. Times, March, 1871.]

[4] Loc. cit. p. 303.

[1] Welsh, loc. cit. p. 45.

number, no less than six were yellow cases, and thus, obviously, in danger of their lives. The majority of the nurses and domestics took the disease, and of the former at one time no less than nineteen were laboring under it. Some of the dispensing physicians and other practitioners took the disorder, as also several of the clergy, and visitors of the sick, whose duties brought them to the bedsides of the patients. The few cases occurring amongst the higher classes, resident in the new town, were generally to be traced to the influence of contagion, the parties affected having had either immediate or indirect communication with those suffering under the disease."[1] And no less decided is the testimony borne by Dr. Cormack: "The disease," he remarks, "is contagious. Of this we have sufficient evidence in the fact that almost all the clerks and others exposed to the contagion have been seized. Dr. Heude and his successor, Mr. Reid, in the new Fever Hospital; Dr. Bennett, my successor there; Mr. Cameron and his successor; Mr. Balfour, in the adjoining fever house; as well as most of the resident and clinical clerks in the Royal Infirmary, have gone through severe attacks during the last summer and autumn. Hardly any of the nurses, laundry-women, or others, coming in contact either with the patients or their clothes, have escaped; at one time there were eighteen nurses off duty from the fever; and of those who have recently been engaged for the first time, or of those who have hitherto escaped, one and another is, from time to time, being laid up."[2] It is in language closely resembling that employed in the sentence now quoted, that Drs. Paterson, W. Robertson, and other physicians have expressed their belief in the contagious property of the Relapsing Fever of 1847–48; and the writer, whose position as resident medical officer in the Fever Hospital while under the care of Dr. Robertson, in the spring months of 1847, afforded him the best opportunity for studying the nature of the epidemic, arrived at the conclusion that the Relapsing Fever, like typhus, is capable of communication from the sick to the healthy; that, for this purpose, actual contact with the sick is not necessary, the subtle poison of this form of continued fever, equally with that of typhus, being readily conveyed through the air surrounding the latter; and, lastly, that, by means of fomites or clothes, the disease may readily be propagated. [In the Philadelphia Hospital, with 517 cases of Relapsing Fever admitted in eight months, no instance of its communication to other inmates of the Hospital occurred.—H.[1]] It appears sufficiently remarkable that, as specially noted by Dr. Cormack in 1843–44, laundry-women engaged in washing the clothes of the sick, though never brought into direct communication with patients themselves, suffered frequently from the disease; but this, too, was noticed in regard to epidemic cholera; and it was an experience of precisely the same nature in regard to that disease, acquired in the same building, the New Fever Hospital of 1843–44, being in 1853–54 used as a cholera hospital, that, more than anything else, convinced the writer of the contagious nature of epidemic cholera.[2] Resembling typhus in the mode of its propagation by contagion, there is one particular in which these forms of continued fever remarkably contrast. An attack of typhus, for the most part, secures the individual who has thus suffered from subsequent attacks. It is otherwise with Relapsing Fever; no such immunity is by it secured. Welsh noticed, in regard to the epidemic of 1817–18, that "being once affected with the disease seems to afford little, if any, protection against a second, or even a third attack, and that, too, in the space of a few months. I have seen many instances of a second attack within the last twelvemonth."[3] It is well known to the many personal friends, as well as the professional brethren of Dr. Christison, that he has frequently suffered from attacks of continued fever, and we have his own authority for stating that, during the epidemic described by Welsh, he experienced three separate attacks within a period of fifteen months. Dr. Wardell, Sir W. Jenner and many other writers on Relapsing Fever have noticed the like circumstance. It requires little acquaintance, however, with Relapsing Fever, and but slight familiarity with the remarkable epidemics to which reference has been made, more especially the last two, those of 1843–44 and 1847–48, to feel assured that contagion, while undoubtedly explaining in part the progress of the disease, does not do so wholly, and stops far short of satisfactorily accounting for any of the phenomena which were observed, and this more especially at the very commencement of the different outbreaks. As assisting the better understanding of a subject still encompassed with difficulty, the generation of fever, there is, in regard to Relapsing Fevers at all events, one consideration of

[1] Wardell, loc. cit.
[2] Cormack, loc. cit. p. 115.

[1] Philada. Med. Times, March, 1871.—H.]
[2] Short account of the Cases treated in the Cholera Hospital, Surgeon's Square, during the late Epidemic, by J. Warburton Begbie, M.D. Edinburgh Medical and Surgical Journal, 1855, p. 253.
[3] Welsh, loc. cit. p. 46.

very considerable importance. It cannot, we think, be denied that an intimate connection subsists between this form of fever and destitution. Dr. Alison—who, in all his many writings on fever, as consistently held as he ably supported the doctrine that intercourse with persons already sick of the disease is the only exciting cause of continued fever, of the efficacy of which we are certain—was evidently greatly impressed by what he and many other observant physicians had noticed of the connection between destitution, or famine, and fever, in the epidemic of 1846–47 more especially; and he has left it on record as his deliberate opinion regarding the prevalence of the fever at that time, that "although burdening the infirmary more than any other which I recollect, it has not for many months spread to any considerable extent among the working classes of the city, but is to be regarded as merely the effect of the unavoidable connection of this country with the destitution of Ireland."[1] Dr. Murchison has adopted an excellent method of demonstrating the intimacy of the relationship between Relapsing Fever and destitution; he has examined the records of the London Fever Hospital and shown that since 1847, 430, or 97·5 per cent. of the patients were paid for by the parochial authorities, and totally destitute. Nine of the remaining patients were admitted free, and were also destitute. Not a single patient had been a servant in a private family, and in only one instance was a fee for admission paid by the patient's friends. A large proportion of the patients for some time previous to their attack had been literally starving. Irish writers, with few exceptions, have insisted on the intimate connection which exists between fever and famine. Stokes described the fever of 1826 as "famine fever," and the well-known pamphlet of Dr. Corrigan, concerning the fever of 1847, had for its title, "On Famine and Fever, as Cause and Effect in Ireland."[2] These physicians, in their respective accounts, referred to epidemics largely composed of Relapsing Fever. Again, the expressions, "Die Hungerpest" and "Famine Fever," clearly indicate that by German observers, as well as by physicians among ourselves, this relationship has been noticed. The whole subject of the etiology of Relapsing Fever has been ably treated by Dr. Murchison, and the opinion which he has expressed of Relapsing Fever being the result of destitution, while typhus is produced by overcrowding and destitution combined, will, we are disposed to believe, stand the test of further observation and renewed careful investigation.

Reference has already been made to the circumstance that at the commencement of the Epidemic Fever of 1843, the disease was speedily recognized as presenting remarkable characters.

"The present epidemic," wrote Dr. Henderson, to whom is justly due the merit of having first expressed the opinion that Relapsing Fever is a separate and distinct disease from other forms of continued fever, "began to prevail in February last, and the very first cases which fell under my notice I distinguished at once as widely different from every fever that I had formerly seen."[1] About the same time other observers, and more particularly Dr. Cormack, from their separate and independent observation, were led to a similar conclusion; and it is indeed impossible for any one to read attentively the descriptions given by Henderson, Cormack, Wardell, Halliday Douglas, William Robertson, Paterson, Mackenzie, Reid, and more recently, but more particularly, Sir W. Jenner, in which the whole proof is admirably handled and exposed—without arriving at the conclusion that Relapsing Fever is a form of continued fever, wholly different from typhus fever, with which it had formerly been confounded. These observers — and all who have had the opportunity of carefully studying the two fevers, must admit the accuracy of their statements—pointed out that the one fever under no circumstances gave rise by communication to the other, and that an attack of typhus never conferred immunity from Relapsing Fever, any more than the latter afforded protection from typhus. It cannot be considered as offering any serious objection to the view which has now been expressed, that, as in the experience of Dr. Henry Kennedy of Dublin, in 1847–48, cases of Relapsing Fever and typhus have been occasionally met with among the members of the same family, and in individuals occupying at the time of the occurrence the same apartment. Dr. Murchison, indeed, alludes to such, as an occasional experience in the London Fever Hospital, since the well-known observations of Sir W. Jenner were made. The circumstance of the existing epidemic being composed of both forms of fever, and the further circumstance that both fevers are of an infectious nature, satisfactorily explain these coincidences. And, upon re-

[1] Observations on the Famine of 1846–47.

[2] Dr. Henry Kennedy's Observations on the Connection between Famine and Fever in Ireland and elsewhere (Dublin, 1847), contain much important information and ingenious argumentation, but, notwithstanding this admission, we adhere to the statements made in the text.

[1] On some of the Characters which distinguish the Fever at present epidemic from Typhus Fever, by W. Henderson, M.D. Edinburgh Medical and Surgical Journal, 1844.

flection, we feel constrained to acknowledge that were the one form of fever capable of producing the other, or, in the view of the spontaneous origin of fever, were typhus capable of being originated in the same way, or under precisely the same circumstances as Relapsing Fever; then the association of the two fevers in the one family, and in the one room among the poor, would have been of infinitely more frequent occurrence than it has ever been proved to be.

Symptomatology. — The suddenness of its invasion is characteristic of Relapsing Fever. The patient is seized with coldness and rigors, accompanied by headache, pain in the back, and loss of strength. The muscular feebleness and general prostration, however, are not at the first great; for, as Dr. Cormack has observed, "many walk long distances from the country to the hospital, especially during the first days of the disease; and a still greater number of the destitute town patients lounge about the streets after the seizure, and come in to us on their legs."[1] The feverishness gradually increases, while the muscular and articular pains and headache become more severe. By the third day, there is usually some amount of epigastric uneasiness, and not unfrequently vomiting. No general abdominal tenderness, however, presents itself, and diarrhœa is of rare occurrence. A perspiration, marked in character, and general over the body, occurs sometimes very early in the disease, on the second or third day, bringing with it little or no relief to the headache and other symptoms. It is from this circumstance, but particularly from the still better marked though more rarely-occurring alternation of rigors and sweating in the earlier days of the illness, that the resemblance to an intermittent fever of irregular character has been remarked by various physicians. On the third or fourth day the fever is at its height, and the case is for the most part characterized as follows: By the greatly augmented temperature of the surface (noted at 102° by Halliday Douglas; as high, and this is very high, as 107° by Wardell); a very quick pulse (this it was which first struck Dr. Henderson as remarkable, unlike what he had witnessed in typhus), very rarely below 100, often 120 (125 on the fifth day, being the average frequency in thirty-eight cases noted by Henderson); it has, however, been noted at 140, and even 160. It was soon determined that, unlike what holds true of typhus, this rapidity of the pulse did not indicate the existence of danger.

[In Philadelphia, in 1870, the temperature was found to rise generally to 104° or 105° on the second day, and to reach its maximum on the day before the critical defervescence. On the occurrence of the relapse, about the fourteenth day, it again rose to 104°, 105°, or even higher; declining again with convalescence.—H.]

With these, the true symptoms of pyrexia, are at the same time associated very slight disturbance about the head, headache frequently, rarely delirium, hepatic and splenic tenderness, with vomiting, great restlessness, thirst, and a white condition of the tongue. In a considerable proportion of the cases a peculiar yellowness of the skin becomes noticeable, best marked in the face, styled by Cormack "facial bronzing," and to this a distinct jaundice, with urgent vomiting, sometimes succeeds. To these symptoms there occurs, usually on the fifth or seventh day, an abrupt cessation. Nothing can be more remarkable than the sudden change—usually ushered in by a profuse perspiration, less frequently by an epistaxis, or other hemorrhage, or by diarrhœa — effected in the condition of the patient. The frequent pulse and hot skin have in a few hours vanished, there is a normal appearance presented by the tongue, and, as Cormack has described it, "one day we hear the patient moaning and groaning in pain, and on the next he is at ease and cheerful, his only complaint being of hunger and weakness." The condition of apyrexia established, the patient continues to improve; he gains strength, often rapidly, and convalescence appears to be altogether satisfactory, except that the pulse sometimes continues remarkably slow. On or about the fourteenth day from the commencement of the original attack the relapse takes place: there occurs a second paroxysm of fever in all points similar to the first; it may, however, be more severe, or on the contrary less severe in its symptoms. The duration of the relapse is usually three days; it may extend to five days, or even longer, and, when usually mild, it may terminate before the third. A second relapse, usually occurring about the twenty-first day, is far from uncommon. "Not less than five of these accessions or 'relapses' have been known to occur."[1]

To some of the more remarkable phenomena now briefly alluded to, and to a few other features in the symptomatology, it is proper to direct attention a little further in detail. Relapsing Fever, properly speaking, is undistinguished by cutaneous eruption. The most careful observers have failed to notice in it the measly rash so characteristic of typhus, no one has described the rose-colored spots (*taches roses lenticulaires* of Louis) now regarded

[1] Loc. cit., p. 3.

[1] British and Foreign Medico-Chirurgical Review, vol. viii. p. 8.

as equally characteristic of enteric or typhoid fever. The "measly-looking efflorescence," noted by Welsh in 1819, as occasionally present, may fairly be considered to have occurred alone in the cases of true typhus which constituted a portion of the epidemic he observed. Petechiæ, hemorrhagic spots, and vibices have all been described as of occasional occurrence, while Dr. Ormerod found a miliary eruption (sudamina) so common in the Relapsing Fever of 1847, that in his description he gave to the disease the name of "Miliary Fever."[1] Dr. Halliday Douglas found sudamina very rarely in the fever of 1843-44, and the writer can answer for their presence in that of 1847 being likewise quite exceptional. But another cutaneous appearance, although variable, is of decided importance—although it is probable that its gravity as a symptom has been unduly estimated by some physicians—namely, a yellowness, or jaundiced hue. Welsh also noticed this: "a yellowish, dusky state of the skin was not unfrequently observed." In connection with it he likewise noticed "that the patient's urine distinctly tinged linen cloth or similar substances immersed in it."[2] In the epidemics of 1843 and 1847 jaundice was observed, in the former specially by Cormack, Wardell, and Douglass; in the latter by Jenner, in London; also, but with greater rarity than during the previous epidemic, in Edinburgh, by William Robertson and R. Paterson.

Nausea and vomiting have been described as common symptoms in Relapsing Fever. The matters vomited have usually been found to consist of the ingesta, frequently tinged with bile. Occasionally an appearance resembling the black vomit of yellow fever was noted, as by Cormack and Wardell, who regarded it as a peculiarly unfavorable sign, and by Dr. Arnott, of Dundee, who, looking upon black vomit as quite common in its occurrence, did not find it by any means a fatal indication. Peculiarities, as regards the appearance presented by the tongue, have been noted. It is usually from the commencement coated with a white or yellowish fur, while a small triangular space towards the point of the tongue, as well as its edges, is clean, and often redder than natural. In mild cases the tongue continues moist throughout the attack; but in the more severe, dryness, blackness, and incrustation with sordes, occur. The appetite suffers in Relapsing Fever for the most part, as in other febrile disorders, but many observers have stated that an unusual and sometimes altogether inordinate desire for food has distinguished particular cases. The urine in Relapsing Fever was specially examined by Dr. Henderson and Mr. (shortly afterwards Dr.) Michael Taylor. Occasionally the quantity is reduced, or there may even be suppression of the secretion, while the amount of urea is greatly diminished; in connection with these changes the occurrence of serious cerebral symptoms is to be apprehended. Here our knowledge of a very important topic ceases. Dr. Parkes has truly observed that scarcely anything definite is known on the subject.[1] Dr. Henderson had his attention early directed to the condition of the kidneys, and satisfied himself that the occurrence of convulsions, coma, and less serious symptoms of a nervous nature, were to be ascribed to interference with the proper function of these organs. In connection with a diminished amount of urea in the urine, Dr. Douglass Maclagan determined, by investigations undertaken at Dr. Henderson's request, the existence of an increased amount of urea in the blood.

There are certain complications and sequelæ of Relapsing Fever. The inflammatory affections within the chest which are known to occur in the course of the other forms of continued fever—at times seriously influencing the mortality which these occasion—are met with also in Relapsing Fever, bronchitis, broncho-pneumonia, pneumonia and pleurisy. Laryngitis, requiring tracheotomy, occurred in one case in the experience of Dr. Paterson. The writer remembers that the presence of a similar inflammation necessitating the same operation in at least one other case, was ascribed, at the time of its occurrence in 1848, to the patient having been peculiarly exposed to cold while under treatment in one of the temporary sheds erected for the accommodation of the fever patients admitted to the Royal Infirmary of Edinburgh. Hemorrhages of various kinds have been noted to occur, for the most part, about the period of crisis. Paralysis of a local nature—of the deltoid muscles, as observed by Cormack—and much more frequently severe muscular and articular pains, are among the number of the nervous complications which have been observed. Parotitis, which is familiar to us in typhus and enteric fever, and other glandular enlargements and suppurations, were certainly of uncommon occurrence in the epidemics of Relapsing Fever witnessed in this country. In the recent Russian epidemic it would

[1] Clinical Observations on the Pathology and Treatment of Continued Fever, from Cases occurring in the Medical Practice of St. Bartholomew's Hospital, by Edward Latham Ormerod, M.B. London, 1848. P. 216.

[2] Loc. cit. p. 21.

[1] The Composition of the Urine in Health and Disease, and under the Action of Remedies, by Edmund A. Parkes, M.D. London, 1860. P. 260.

appear that these buboes are more frequent; and Mr. Simon has no doubt correctly inferred that "from this circumstance there arose the rumor of plague."[1] Diarrhœa, sometimes taking the place of perspiration, has been described by many observers as the critical evacuation in Relapsing Fever; with a greater degree of frequency it occurred during the relapse, or after recovery from the relapse. That diarrhœa raised the mortality considerably, is evidenced by the statements of various of the Scotch physicians.

There are few more interesting circumstances known in regard to Relapsing Fever than the frequency with which pregnant women abort or miscarry. By some this accident has indeed been described as invariable; it is not so, but the exceptions are infrequent. In relation to this peculiarity, Dr. Murchison has truly observed that, on the supposition that Relapsing Fever is but a mild variety of typhus, it would be very remarkable that, in the former, abortion is almost invariable, and the fœtus dies; whereas, in the latter, abortion is the exception, and when it occurs, the child, if near the full time, usually lives.[2] A sudden, wholly unlooked-for, and at times fatal, syncope has distinguished some cases of Relapsing Fever. Dr. Halliday Douglas mentions one such in which the patient was found dead about the period of the first crisis, and only half an hour after she had expressed herself as feeling easy. An occurrence of this kind, though fortunately not common, may well be considered important in a prognostic point of view. Lastly, an interesting form of ophthalmia presenting two distinct stages, the amaurotic and the inflammatory, has been met with and specially described by Dr. Mackenzie, of Glasgow, as post-febrile ophthalmia.[3] In that city so frequent was the eye affection in 1843, that Dr. Andrew Anderson speaks of multitudes of cases having been treated at one Eye Infirmary.[4]

When the frequency of the occurrence, either of more than one relapse, or one or other of the complications or sequelæ which have been attended to—others less frequent and of less severity it has been thought unnecessary to mention—is considered, it will be understood that Relapsing Fever, though happily occasioning a mortality greatly inferior to typhus, is a fever determining—in not a few of the sufferers from it—long-continued bodily weakness; while in a still larger number convalescence is greatly protracted.

The account which has been given of the symptoms and course of Relapsing Fever is sufficient, we think, to establish its separate and distinct nature, and to allow its being readily distinguished from typhus, enteric fever, febricula, remittent, or yellow fever, diseases, with the majority of which it has been at one time or other confounded. The relapse, which is the distinguishing feature of this pyrexia, is, properly speaking, unknown in any other form of fever; and Sir W. Jenner's careful observations, confirmed by the experience of many competent authorities, have proved that Relapsing Fever—a contagious disease, as we have already seen—is capable only of giving rise to a similar disease; it can engender no other form of fever, and no other form of fever can engender it. What there is in the morbid anatomy of Relapsing Fever, and in the mortality it occasions, to sustain and confirm this opinion, we shall now inquire. As to the former, unlike what holds true of pythogenic or enteric fever, there is then no constant or invariable morbid appearance to be detected. Nevertheless, there are a few thoroughly ascertained facts in regard to this subject, which, in the not unlikely event of another occurrence of Relapsing Fever, should form the groundwork for renewed and still more extended investigation. The spleen is almost always found altered; enlargement and softening, nearly in some instances to the extent of diffluence, are the most frequent changes, but increased firmness in its structure, and fibrinous deposits in the splenic substance, have likewise been observed. Enlargement and engorgement of the liver, without any structural change—even as has specially been stated by Cormack and others—in the best marked "yellow cases," have been generally found. The blood, when subjected to microscopic examination, has revealed the existence of an increased number of white corpuscles, similar to what occurs in the now better understood conditions of leukæmia.

[Dr. Hand, of Philadelphia, reported the observation of a granulated and crenated appearance of the red corpuscles. Obermeier, of Berlin, discovered minute moving filiform organisms, called *spirilla*, in the blood in Relapsing Fever.—H.]

The mortality occasioned by Relapsing Fever is usually not great, being far inferior to what is commonly observed in

[1] See his Letter to the Lord President of the Council, dated Whitehall, April 19, 1865. It may be right to mention here, that in the experience of the writer, parotitis, hitherto a very rarely observed phenomenon by him in fever, has during the last twelve months been seen frequently, both in typhus and enteric fever.

[2] See a confirmation of the last observation in an account published by Dr. Matthews Duncan, of a case which recently fell under the writer's notice. Edinburgh Medical Journal, September, 1863.

[3] See Mackenzie, in Medical Gazette, 1843.

[4] Lectures on Fever, p. 135.

either typhus or enteric fever. In the recent Russian epidemic, Relapsing Fever is, according to Mr. Simon, "causing more than its usual proportion of deaths;" but that usual proportion does not exceed 4·75 per cent., or one death in every twenty-one persons attacked. Dr. Murchison, on placing together the results noted at the London Fever Hospital, with those detailed by various physicians during the Scotch and Irish epidemics, has given as the total 14,119 cases, with 672 deaths—yielding the rate which has just been quoted. Age and habits influence the mortality in a manner closely resembling that which is witnessed in typhus and adynamic fevers generally. The mode of fatal termination is not always alike, the occurrence of a sudden and fatal syncope has already been noticed; and, as originally indicated by Dr. Henderson, the impaired action of the kidneys, leading to the imperfect elimination of urea, causes death by coma. An asthenic termination, too, may occur in those instances of the disease in which one or other of the complications already adverted to, have manifested themselves.

Therapeutics.—If, as we believe, there is an intimate connection between famine—by which is understood poverty and destitution—and Relapsing Fever, then it will readily be conceded that such attention to the wants of the poor, particularly as regards due nourishment, as ameliorates their condition, will tend directly to prevent the origin and to arrest the spread of this disease. It is likewise contagious, and therefore isolation of the sick should, as much as possible, be secured. Dr. Welsh, to whose account of the epidemic fever of 1817 and 1818 we have frequently had occasion to refer, conceived that blood-letting was the great remedy. Little reliance, however, can be placed upon the therapeutic observations of Welsh, seeing that neither he nor other physicians of his time had distinguished between typhus and the fever which showed the tendency to relapse. Further, it will, we think, appear to any attentive reader of Welsh's interesting work, that the changes in the condition of the patient, particularly the reduction of the pulse, the diminution of the fever heat, the occurrence of sweating—ascribed to the beneficial operation of the blood-letting—were in reality merely those changes which a subsequent better knowledge of the disease has led us to recognize as the essential phenomena of the fever itself, invariably occurring in its course, and uninfluenced by any treatment. All attempts to ward off the relapse, and for the most part attempts to postpone it, have signally failed. Those antiperiodic remedies in the use of which we place reliance, and the virtues of which in the ordinary intermittent and remittent fevers have been incontestably established—such remedies as quinine, arsenic, bebeerine, salicine—have all been faithfully tried, and all have failed.

Emetics of ipecacuanha, or of ipecacuanha with antimony and mild laxatives, exhibited early in the disease, have appeared to be useful in the hands of various physicians in relieving portal congestion, and producing freedom from the oftentimes distressing pain, or at least uneasiness experienced in the region of liver, stomach, and spleen. Diuretic remedies—and particularly the salts of potash—were serviceable in relieving the tendency to head symptoms, by which, as we have already stated, some cases of Relapsing Fever were distinguished.

Those considerations for the proper employment of food, and the administration of stimulants, which should guide the physician in the treatment of the other forms of continued fever, are available likewise in the instances of Relapsing Fever. It seems unnecessary to enter on an analysis of these here; and while the complications and sequelæ of Relapsing Fever are, as we have seen, sufficiently remarkable, there is only one, the ophthalmic affection, the treatment of which seems to require a brief description; this may be given in the words of Dr. Anderson: "We learned very important lessons from the treatment of this ophthalmia—lessons which tell against some of the theories which are fashionable at the present day. The previous fever and the actual debility of the patients made us at first eschew anything like depletion; but we found on the failure of other means that bleeding was the most effectual—the only effectual—mode of cutting short this dangerous ophthalmia."[1] After the abstraction of blood, calomel and opium were administered, until there appeared evidence of the system being slightly affected by the mercury. These remedies may be prudently combined with quinine and a generous diet, while it is almost unnecessary to add that in the treatment of the purely and simply amaurotic affection, altogether unconnected with hyperæmia, which occurs as a sequela of Relapsing Fever, only tonic remedies and an invigorating diet are required.

[1] Anderson, op. cit. p. 135.

YELLOW FEVER.

By John Denis Macdonald, M.D., F.R.S.

Definition.—Infectious continued fever, ushered in with languor, chilliness, and more or less severe lumbar pain and frontal headache; countenance flushed; eyes at first humid, then suffused, and ultimately ferrety; skin imparting a tingling heat to the touch, and as the second stage advances, gradually acquiring a lemon or greenish-yellow tinge; mind usually disturbed with hallucinations, or more or less violent delirium; restless watchfulness, or, possibly, drowsiness, even to extreme coma; epigastric uneasiness; spontaneous vomiting without effort, first of a clear glairy fluid, but subsequently with "coffee-ground" flocculi, or blood itself, often, towards the close, with irrepressible hiccough, and wild shrieking or melancholy wailing; tendency fatal, but the disease generally confers an immunity from subsequent attacks.

Synonyms.—Yellow Fever, Bulam Fever, Hæmagastric Pestilence, Black Vomit. *Latin*—Febris Flava, Synochus vel Typhus Icterodes, Synochus Atrabiliosa. *Spanish*—Fiebre ò Calentura Amarilla Vomitonegro ò Prieto. *French*—Fièvre ou Typhus jaune, Fièvre Mattilote, Mal de Siam. *Italian*—Febbre Gialla.

History.—From the year 1647, when the first recorded outbreak of Yellow Fever in the West Indies[1] occurred, to the present time, this disease has been recurring at irregular intervals in the epidemic form, and gradually extending its range. It has, moreover, appeared, in many instances, to borrow new vigor by its importation from one place to another, and though it may be said to be permanently present in some localities, *e. g.*, the islands of St. Thomas and St. Domingo, there is no proof whatever of its spontaneous development anywhere. Every epidemic seems to have some assignable source, and even where this is not very obvious, there are *à priori* reasons enough to trust that it would be discoverable, were investigation properly instituted.

[The earliest recorded visitation of Yellow Fever in the United States occurred at New York, in 1668. It appeared destructively in that city also in 1702, 1743, 1795, 1798, 1803, and 1822. Philadelphia was first visited by it in 1695; also, in 1793, 1797–98–99, 1805; and, with less severity, several times in later years. Mobile was the seat of an epidemic of it in 1705; New Orleans, for the first time, in 1769. The years of the most serious epidemics in the last-named city have been, 1819, 1847, 1853, 1854, 1855, 1858, 1867, and 1878. The last epidemic extended to many localities on both sides of the Mississippi River. Several of the Atlantic cities of the United States have suffered severely from it in different years.—H.]

The symptoms of Yellow Fever manifestly result from the more or less potent operation of some subtle organic poison upon the system through the medium of the blood; and the very fatal tendency of the malady is probably linked with nature's efforts to eliminate the poison by the gastro-intestinal mucous membrane, when both liver and kidney have been rendered ineffective in bringing about this result. The fact may want confirmation, but it has always occurred to the writer, that the liver is especially active in persons perpetually exposed to the specific infection of the disease, without, however, yielding to its influence; as though the elimination of the poison had been effected, in their case, without developing the train of symptoms proper to the disease.

Whatever physical conditions—such as increase of temperature, moisture, and subsequent evaporation, and the like—may be favorable to, or merely coincident with, the development of Yellow Fever on shore, when once communicated to a ship, and isolated by far removal from all local influences, its phenomena are very striking and suggestive. Under such circumstances it is difficult to witness the spread of the disease from one individual to another, and its virulence becoming more intensified by the unavoidable crowding of the sick, without recognizing the important part which the emanations and excretions of the human body itself must take in the matter. It may be objected that all the most potent of the terrestrial or atmospheric conditions alluded to are fulfilled in the bilge effluvia; but as these, *per se*, have never been known to originate the disease, we are driven to one or other of two positions, either that they

[1] Ligon's History of Barbadoes.

have nothing to do with it, being simply coincident like other things that might be named, or that they form the nidus for its further development and spread subordinate to a specific cause. It must, however, be apparent to a close observer, that the human element far outweighs all other suppositions, and, in this point of view, the refinement and subtlety of the poison may be more easily conceived. If Yellow Fever be referrible to the zymotic class, in support of which idea several cogent arguments might be adduced, its mode of origin cannot be materially different; and few nowadays would attempt to trace the whole of the specific virus of rubeola, scarlatina, or other true exanthemata, to any of the common decompositions of cesspools or the effluvia evolved in bad drainage, and such, otherwise, certainly unwholesome conditions.

We are in want of proof of the spontaneous development of Yellow Fever independently of infected places or persons, while a knowledge of the precise nature of its specific cause is still a desideratum to medical science. But that it is endemic in certain localities, though varying much as to its intensity, and the periods of its manifestation, cannot be doubted; for under similar circumstances, *i. e.* of latitude, or climate, &c., certain other localities have never been visited by the disease.

What has just been said in regard to Yellow Fever very strikingly points to analogous facts in the geographical distribution of plants and animals, and the curious laws that regulate both their propagation and decline. But it may be considered probable, however much the human system may modify their composition and manifestation, that the primary zymotic poisons owe their origin to the development of the humbler and more minute, and therefore more subtle, forms of animal and vegetable life; such being always coincident with a corresponding amount of decomposition, and the evolution of new or simply liberated compounds in a gaseous or diffusible form.

The presence of offal and filth, or stagnant water, with infused animal and vegetable matter, may be regarded as affording something more than predisposing conditions; and such a state of things is often, though certainly not always, discoverable where Yellow Fever is rife, but more particularly in sultry weather, after heavy rains.

The periodical occurrence of Yellow Fever, with intervals of immunity, has its parallel in a fact well known to the students of the diatomaceæ and dermidiaceæ —namely, that particular species, which are known to exist in a definite pond or pool one season, may be at another replaced by forms never before detected in the same spot; while, again, the original species, under favorable and often unaccountable circumstances, reappear after the lapse of a certain time.[1]

It is not our intention to make even brief reference to all the views that have been put forward as to the nature of the specific cause of Yellow Fever; but it may be remarked that even if they were carefully detailed, our conclusions would probably be the same; for upon this head very little more than what has been above stated is critically known.

The infectious nature of Yellow Fever is now not only generally admitted, but it forms one of the most distinctive features of the disease, at once marking it off from those fevers which in nearly every other particular simulate it.

It is scarcely necessary to multiply or repeat the "strong proofs" in this place, after the satisfactory evidence lately brought before the Epidemiological Society of London, in the papers of Dr. Bryson, and the important verbal support of the late lamented Dr. M'William. The tenets of the writer, derived from actual facts, are the following, acknowledging a genealogy to the widely-spread family of Yellow Fever.

1. That the first place or the first person, or both, must have become infected, somewhere or somehow.

2. That by veritable, but unknown, or rather untraced, links with this source, places, having become infected, may infect persons.

3. That persons infected may infect other persons and places previously presumed to be healthy.

4. That the clothing of infected persons, or of healthy persons having communicated with infected places or persons, may impart infection to other places or persons.

5. That if places were movable, like persons (which is literally true of ships), on being infected, they would impart the virus to other places in sufficiently close proximity.

6. Finally, from the investigation of the history of particular cases, it has been satisfactorily shown that the period of incubation, or latency, in this disease, *i. e.* from the imbibition of the poison to the first appearance of symptoms, ranges from one to fourteen or fifteen days.

[Of the above account, that which is most evidently beyond controversy is the statement that Yellow Fever is "endemic in certain localities," while, "under similar circumstances, of latitude, climate, &c., certain other localities have never been visited by the disease." This existence of a "true Yellow Fever Zone"[2] is,

[1] Med. Journal of H.M.S. *Icarus* for 1860, N. A. and W. I. Station.

[2] See *Diagnosis*, p. 286.]

indeed, the cardinal fact in the natural history of the disease. Its geographical "habitat" is as well marked as that of almost any plant, which is capable of transplantation, but difficult of permanent naturalization.

Dr. R. La Roche, in his exhaustive treatise on Yellow Fever,[1] summing up the results of all the history and literature of the subject, confirmed by personal observation, asserts strongly the non-contagion, as well as the locally infectious character of this disease. Of ships, he says[2] that "by them the disease, or its cause, may be, and has been, introduced into healthy places, and communicated to those who have gone on board, or approached sufficiently near to be placed under the influence of the effluvia issuing from them. But such an introduction differs materially in its consequences from that of a contagious poison."

While controversy is endless, from the nature of the case, between opposing interpretations of the same facts in regard to the extension of disease from place to place, it is important to bear in mind how often the most careful and elaborate investigation, by competent inquirers, has resulted in the conclusion just cited. Thus, the London General Board of Health, the Superior Council of Health at Paris, the Sanitary Commission of New Orleans, and other such bodies, have from time to time judicially pronounced in favor of the discrimination between contagion of *persons* and infection of *places* (including ships) in the causation of Yellow Fever. It is true that, in some of the same places, a similar official expression in a contrary sense has afterwards sometimes been obtained, by the urgency of a few, under a "wave" of alarm in regard to contagion and quarantine. But a survey of the whole history of the disease leaves it still to be concluded, that, at the most, contagion is to be admitted as a merely possible supposition, extremely hard even for its advocates to trace with certainty in any definite instances; while the geographical relations of the disease, and its promotion by local unsanitary conditions, are palpable and overwhelmingly important.

No more deliberate and competent consideration of this subject was ever given forth than that of the Third National Quarantine and Sanitary Convention, which assembled in New York in 1859. As the result of a full discussion, participated in by nearly all those who by experience and information were entitled to judge of the question as one of American sanitation, the following resolution was adopted, by a vote of 85 yeas to 6 nays:—

"Resolved, that in the absence of any evidence establishing the conclusion that Yellow Fever has ever been conveyed by one person to another, it is the opinion of this Convention that the personal quarantine of cases of Yellow Fever may be safely abolished, provided that *fomites* of every kind be rigidly restricted."

The majority of this Convention attached little importance to the clause in regard to *fomites*, except as a part of the general system of local sanitation, which has been shown to be as indispensable and efficacious in the prevention of Yellow Fever as in that of any other disease whatever.[1]

The *geography* of Yellow Fever is certainly remarkable. It is comprised within the borders of the Atlantic Ocean, and the waters (Gulf of Mexico, Western Mediterranean, and the rivers emptying into these) communicating with it, in the zone between 42° north and 35° south latitudes. Especially are the West India Islands subject to it. Next after these, in regard to frequency and severity of visitation, have been New Orleans and some other cities of the Southern United States.

Rarely does it prevail many miles from the sea or from a considerable river. None of the far interior cities of either continent have ever been visited by it. Never, except as, in a very few instances, transiently conveyed by infected ships, has it been seen upon the coasts of the Pacific Ocean. The oriental tropical homes of cholera and plague have never known it. How very different is all this from the history of an indefinitely portable, truly contagious disease!

It has been a prominent fact in repeated epidemics in different American cities, that the infection of Yellow Fever is, from time to time, *localized in certain limited spaces*, mostly measurable in fractions of a mile. Many times the removal of the inhabitants of an infected city, in large numbers, has brought them security, while the scattering of those who thus migrated, some of them with the disease in their systems, has not extended it elsewhere. Thus, from Barcelona, in 1821, 80,000 of the inhabitants escaped by flight; while of those who remained in the city, one-seventh, 10,000, died.[2] During the

[1 In two volumes, Philada., 1855.]
[2 Op. citat., vol. ii. p. 545.]

[1 At the meeting of the American Public Health Association, at Richmond, Va., in 1878, resolutions were passed pointing in a different direction from the above. But it was evident that this meeting, called under the pressure of public alarm on account of the extended and destructive epidemic of that year in the Southwestern States, had not time nor material for doing justice to its subject. This was clearly indicated in a circular issued by the Executive Committee of the Association, shortly after its adjournment.—H.]
[2 Second Report on Quarantine, 1852.]

war between the United States and Mexico, in 1846–7, Dr. Bennett Dowler reported that many thousand instances of such removal of individuals proved experimentally its advantage to those who have left infected localities, and its safety to others amongst whom they have gone.

Practically, we deduce from these facts the conclusion, that three things are unquestionably needful in the *prophylaxis* of Yellow Fever: 1. Complete and persistent *local sanitation* in all places within the "Yellow Fever Zone;" 2. Vigilant inspection, at sea and river ports, of all vessels, so as to detain those found to be unclean, at a distance from city wharves. until they have been thoroughly purified and disinfected; 3. Removal of all inhabitants from every spot ascertained to be infected, to some open and salubrious locality.[1] If but a small fraction of the large expenditure provided, by aid of the benevolence of the people of the Northern States, in the summer of 1878, for the care of the sick and dying in the stricken cities of the South, had been applied to their early removal, house by house, and ward by ward, as soon as local infection was ascertained, hundreds, perhaps thousands, of lives might have been saved, and much commercial distress averted. Of course part of such a plan must be, the choice of healthy places for accommodation of those removed, and the conservation among them of good sanitary conditions.—H.]

Altitudinal and Horizontal Ranges.—It may be very well to assign an altitudinal limit to the spread of Yellow Fever—and, roughly speaking, this may be estimated at between 2000 and 3000 feet above the level of the sea—but the local conditions of every country seem to determine a range peculiar to itself. Thus, the disease has been known at Newcastle, Jamaica, at an elevation of 4000 feet; while in the Valley of the Mississippi its highest recorded range is about 600 feet (admitting the Fever of Gallipolis to be of the genuine type). Humboldt alludes to the Farm of Encero, in Mexico, at an elevation of 3243 feet, as the altitudinal limit of Black Vomit. At St. Domingo, the mountain encampments of the French in 1792 and of the English in 1796, enjoyed an immunity from the disease, while it was spread far and wide amongst the troops in the low country.

Though the West Indian Islands, and the neighboring coasts of North and South America, may be looked upon as the focal area of Yellow Fever, yet taking the outlying points at which its occurrence in the epidemic form has been recorded, its geographical range must be regarded as very considerable indeed, *i. e.* between 97° west and 2° east longitude, and between 48° north and 35° south latitude.

At least for the space of a century and a half, up to the year 1850, the river Amazon, dividing the Brazils from Guiana, limited the extension of Yellow Fever south of the line; and while the disease was raging at Rio and Bahia at the close of that epoch, the Montevideans flattered themselves that they were without the geographical limit of the pestilence, until it fell to their turn to sustain its visitation several years later, when the illusion was dispelled. Similar facts may be adduced with regard to the extension of the disease along the shores of the Pacific; so that, however well we may be acquainted with its present range, making all due allowance for temperature, we cannot tell what the future may bring forth. In this connection it may be mentioned that a temperature of at least 72° is assumed to be essential to the development of Yellow Fever, though cases exceptional to this rule also may now and then happen.

Symptoms.—With or without such premonitory symptoms as loss of appetite, costiveness, flatulence, sense of debility, and the eyes humid and bright, the disease frequently makes its invasion with chills; but this will greatly depend upon the existing temperature or climate. Thus Jackson maintains that they seldom occur within the tropics, while they are quite usual in more temperate climates. The chills alternate with flushes of heat, and the latter gradually settle down into regular fever, which is often observed to become more severe towards evening, with something approaching a remission in the morning. The amount of fever, moreover, bears relation to the severity of the chills preceding it. Frontal headache is also an early symptom, with shooting pains through the orbits and temples; but distressing as these may be, they are usually trivial in comparison with the agony of the lumbar pains which frequently seize the patient at this period, and fell him to the ground in a writhing and convulsive state. In some severe cases, however, this symptom is nearly entirely absent.

From the very commencement the patient may be troubled with nausea and epigastric tenderness, or they may be developed as the reactive stage advances.

The pulse exhibits great diversity of character, being much accelerated, full, and strong, in keeping with the force of the paroxysm or even soft and weak where the febrile reaction is deficient in severe cases—the beats ranging between 90 and 120 in a minute.

[1 What to Do Against Yellow Fever: by H. Hartshorne, M.D. Reports and Papers of American Public Health Association, vol. i., 1873.]

Also varying with the nature of the paroxysm, the skin may be hot and pungent, dry or perspiring, livid, flabby, and even cold.

The tongue exhibits a creamy-white coat on the dorsum, with red tip and edges, and injected papillæ, with or without soreness of the throat.

As the second stage advances irritability of stomach is added to the nausea, and the epigastric pain and tenderness become more distressing. The patient craves for cold drinks, which are immediately rejected, first with some retching and pain, but subsequently without effort. The matters vomited usually have a suspicious appearance: thus, they are sometimes imbued with bile, lightly streaked with blood, or quite serous with small chocolate-colored flocculi, discovering the tendency to hemorrhagic oozing from the lining of the stomach.

The urine is scanty, high-colored, and probably albuminous: the stools become gradually more and more deficient of bile; and the bowels are often obstinately constipated.

The patient begins to be restless and vigilant, and disposed to leave his bed, go into another, or walk about naked if he be permitted. He exhibits an evident derangement of intellect, though he may answer questions coherently. In other instances, — with a suffused ferrety eye and a drunken expression of countenance, —wild hallucinations, similar in every respect to those of delirium tremens, afflict the victim's mind, and may deceive the practitioner most seriously in sporadic cases.

Febrile reaction may continue for an indefinite period between a few hours and two or three days, and its duration is said to be in the inverse ratio of the violence of the attack.

"Having run this course," says Dr. La Roche, "the fever subsides, never more, or very seldom, to return—the disease being one of a single paroxysm—and is followed by a state of remission or metaphosis." The nature of this remission is all important, as regards the fate of the patient. Should all the symptoms be alleviated, the pulse becoming less frequent, or even normal, the delirium subsiding, and, above all, if there is no more irritability of stomach; active diaphoresis, epistaxis, or a critical discharge of bile from the bowels, may place him on the highroad to recovery. Should the skin have assumed its lemon-yellow tint, it will remain all through the convalescence, towards the close of which the writer has noticed a desquamation of the cuticle much resembling that of the ordinary exanthemata.

If, on the other hand, the ferrety eye whitens, the cheek grows pale, and the lips are blanched, while the pulse is weak and compressible, and the delirium is persistent with irritable stomach, the apparent remission is delusive, and a fatal issue is pending. The patient refuses all medicine and food, lies down very much against his own inclination, cramps gather in the calves of his legs, and while they are being rubbed by the attendants, his delirium becomes frantic, and is probably retrospective of former impressions. His utterance is supernaturally rapid, keeping pace with a panoramic sequence of idea in which the mind is absorbed, the pulse is imperceptible at the wrist, and just when physical exhaustion has merged into death a final automatic discharge of black vomit closes the tragedy. It is only at this period, in many cases, the lemon-chrome tint of the skin makes its appearance. When it happens in the course of the disease, *i. e.*, third stage, it is observed to spread from the forehead downwards to the face, neck, and chest, and then it becomes general.

In another class of cases, the pulse gradually moderates, but thirst increases, and epigastric heat and pain are persistent, with irrepressible vomiting and hiccough, but the mind is calm and coherent to the last, though quite conscious of its tendency to wander. Indeed, the effort of the intellect to correct vagrant ideas, and give them a rational form, is often affectingly observable, more particularly in the case of educated persons.

Contrasting remarkably with the class of cases just described, some persons exhibit a tendency to drowsiness at a very early period of the disease, and finally settle down into a placid state of coma, not unlike that of severe concussion of the brain, without pressure or organic lesion.

This state of things, no doubt, results from uræmic poisoning in connection with the suppression of the urinary secretions.

It is a fact, worthy of special note, that the heart's action may continue long after the pulse at the wrist has become imperceptible, and when all respiratory movements have ceased.

Death may happen in the course or at the close of any of the three stages of the disease, namely: 1st, the accessionary; 2d, the reactive; or 3d, the remissional: and this will of course be in accordance with the type or variety assumed by the malady.

Further remarks on the symptomatology, rendering the ideas here given of it more complete, will be found in the section on the classification and varieties of the disease; as it is of importance to avoid unnecessary repetition.

DIAGNOSIS.—Though a very good general sketch of Yellow Fever may be given

by any one who has witnessed an epidemic of the malady, it is not quite so easy to isolate the symptoms that may be fairly assumed to be pathognomonic. Indeed, the whole aspect of the disease is often so diversified, or distinguished by the absence of this or that symptom where the collateral evidence of its identity is indubitable, that it is difficult to say which feature is of most diagnostic importance. Add to this the actual occurrence of the several symptoms of Yellow Fever in some part of the course of other febrile disorders which, after due consideration, have been declared to be essentially distinct, and the difficulty will be still more apparent.

Inasmuch as yellowness of the skin and conjunctivæ, not merely from the effusion of the hæmatine of the blood itself, as in Yellow Fever, sometimes occurs in the paludal remittent fevers of various countries, that character, singly, cannot be pathognomonic of Yellow Fever. Of black vomit also the same thing may be affirmed, so that two of the most important features of the malady are scarcely available for a satisfactory diagnosis. Much stress has been laid upon the very constant symptom of frontal headache, in connection with the watery and suffused eye, the white, creamy, or cottony coat of the tongue, and its red tip and edges; and in particular the early appearance of albumen in the urine; but none of these characters, nor even all together, can be more definite than the following positions:—

1st. Now that there can be no doubt of the infectious nature of the disease, it may thus at once be distinguished from those disorders with which it is likely to be confounded, for this property is sure to be developed in every epidemic of specific Yellow Fever.

2d. As Yellow Fever is one of a "single paroxysm," of longer or shorter duration, and divisible into three stages, the disease is continuous in its type, or it runs a definite course without such remission and exacerbation as are seen in the paludal fever.

3d. If Yellow Fever shall have passed through all its stages without destroying life, it in general confers immunity from a second attack.[1]

The first appearance of Yellow Fever not infrequently presents no other symptoms than those of an ordinary ephemeral fever; for which the writer very naturally mistook the first case of Yellow Fever that ever fell under his observation. The disease, however, declared itself on the second day, and there was no further doubt as to its real nature. There is a still greater possibility of confounding malarial remittents with specific Yellow Fever, and even good observers have regarded them as identical. In this connection, however, we may appropriately quote Dr. Maclean's bold diagnostics, as given by Dr. Aitken.[1] "I am now myself a firm convert to the doctrine that Yellow Fever is specially distinct from remittent. To this opinion I have come with a full knowledge of the fact that some cases of remittent fever in India closely resemble some of the forms of Yellow Fever. But of this I am now certain, that the Yellow Fever of the true yellow fever zone is unknown in India, where true malarial fevers abound. There is in true Yellow Fever, for the most part, an absence of that periodicity which is an unfailing characteristic of true malarial fevers. Then there is the difference, so well insisted upon by Blair, in true malarial fevers. Men do not pass from recovery to health, as is the case in such a marked degree in Yellow Fever, after which there is no, or very little, evidence of the existence of any cachexy. Malarial fevers exist and are destructive at a temperature at which Yellow Fever is at once destroyed. Albuminous urine is almost invariable in Yellow Fever—only occasional in remittent. There is in Yellow Fever an unexampled range of hemorrhages; in remittent fever these hemorrhages are often, indeed generally, absent. Quinine has a power over malarial fevers that is beyond the reach of doubt or cavil; the same is not true of Yellow Fever. Men suffer from malarial fevers again and again; second attacks of Yellow Fever are, to say the least, rare."

[There is reason to believe that what the late Dr. S. Dickson (formerly of South Carolina) called a "blending of types" sometimes occurs, between Yellow Fever and remittent fever. This will account for the very few instances in which true black vomit is said to have been witnessed in connection with remittent. If it ever is seen in an uncomplicated case of malarial fever ("country fever"), it must be one of the rarest of symptoms.—H.]

PATHOLOGY.—*Temperature of the Body.*—The writer has observed that when patients previously treated in the open air on board ship were transferred to hospital, the body exhibited a marked increase of temperature, and the febrile symptoms became more active. Moreover, any part of the body exposed more than another soon evidenced a diminution of animal

[1] In H.M.S. *Icarus*, in 1860, only five persons out of about 130 escaped an attack of Yellow Fever; yet not a single case of relapse occurred during the whole course of the epidemic.

[1] Science and Practice of Medicine, vol. i. pp. 479–80.

heat. In the axilla the temperature may range from blood heat to 107° Fahr., the maximum observed by Dr. Blair. In the "sthenic form," in comparison with others, Dr. Lyons noticed a general elevation of temperature ranging between 3° and 7° Fahr.

Coloration of the Skin.—On the first accession of the disease the skin becomes pale and, perhaps, shrivelled to a greater or less extent; but, as the reactive stage sets in, it warms up and grows red, the face in particular appearing animated and flushed. The depth of this redness, of course, bears relation to the intensity of the febrile symptoms, and it is looked upon as pathognomonic from its very constant occurrence, whenever reaction is developed at all; but should the latter be defective, the countenance may be pale, livid, or sallow.

The so-called jaundicing of the conjunctivæ and skin is by no means a constant symptom, but may be more or less characteristic of particular epidemics or of different stages of the same epidemic.

It is highly probable that the greenish-yellow hue is often due to the presence of bile. But, as I believe was first suggested by Warren,[1] and subsequently by Sir G. Blane and others, the lemon-yellow and orange tints are unquestionably owing to the solution and effusion of the coloring matter of the blood. M. Guyon regarded it as nothing more than the tinge of contusion. They should not be confounded with the much darker and more greenish hue of Yellow Remittent Fever, depending altogether upon jaundice, and therefore of a very different nature.

Though yellowness of the skin may set in at any time from the first to the fifteenth day, or even exhibit itself after death, yet from Dr. Blair's observations, it occurs most frequently on the fourth and fifth days of the disease. It is important to note, however, that it makes its appearance coincidently with the black vomit in the generality of cases.

The Tongue.—It is only when the febrile reaction is taking place, or even some time after this has begun, that the tongue in general assumes its characteristic white coating, with red tip and edges. Previously to this it may be quite normal in appearance, only perhaps bearing the impressions of the teeth. Indeed, instances occur in which it remains without marked change, even up to the close of the malady, either in recovery or death. The characters of the tongue, therefore, cannot be always said to go hand in hand with the increased heat of skin and acceleration of pulse.

After having first become coated with a white creamy substance this condenses into a thick cottony fur, and the marginal papillæ become enlarged. The coating becomes thicker towards the base of the tongue, and more discolored; moreover, one, two, or more yellowish, brown, or black longitudinal bands run down its middle. When hemorrhage arises from the parts about the mouth, the epithelium of the tongue and fauces is soon stripped off, leaving the surface glazed with half-dried blood and sordes.

Under such circumstances, the tongue is more pointed and smaller than usual, of a mahogany-red color, and more or less fissured. As might be expected, the denuded throat is sore and requires special treatment.

The accompanying table, as quoted by Dr. La Roche, from the records of Roper Hospital, Charleston, during the sickly season of 1854, gives a good idea of the difference of character presented by the tongue in the three stages of Yellow Fever.

[1] "This yellowishness, I am persuaded, chiefly arises from a more complete colliquation or dissolution of the red globules of the blood into a yellowish serum, which will naturally soon give that tincture to the whole skin. The same is also observable on human bodies soon after bites of some poisonous serpents, or other venomous animals; and, in such cases, it cannot with any reason be supposed to proceed from a suffusion of bile, but rather from a colliquation, and perhaps a gangrenous diathesis of the sanguineous mass, occasioned by the force of the deleterious venom that had been infused into it. What is observed every day in all common bruises of the flesh may serve somewhat further to elucidate the matter; for here, when the texture of the extravasated blood begins to loosen and dissolve into a liquid serous consistence, a very visible yellowness appears in and about the part; but this soon goes off again, when the matter is fully absorbed back into the vessels, where it commits no hurt, but is readily overcome by the force of nature, as the quantity of such dissolved blood is small, and at the same time very innocuous. I do not, however, deny but that, through a great propensity and straining to vomit, some quantity of the bile may be thrown into the blood; but then I must observe, that the yellowness of this distemper I am speaking of very frequently shows itself when there has been no vomiting or retching at all, or scarce any sensible sickness of the stomach; for the truth of which I can appeal to many."—Treatise on the Malignant Fever of Barbadoes, p. 11, as quoted by Dr. La Roche.

Condition of the tongue.	1st stage.	2d stage.	3d stage.	Total.
Swollen	4	29	31	64
Dry	52	23	14	89
Bloody	—	3	31	34
Whitish	44	33	18	95
Brownish	94	53	39	186
Moist	109	109	110	328
Red	43	26	33	102
Velvety and white	23	2	3	28
Black	1	1	19	21
Natural	26	10	7	43
Glazed	4	2	—	6
Cracked	—	3	—	3
Total number of observations				999

The Pulse.—If the tongue has been observed to be variable and inconstant in its character, the same may be said of the pulse, and trusting to it alone a very incorrect prognosis may be formed. Thus, it has been known to preserve an apparently normal state, even coincidently with the most portentous symptoms; and it has been previously noticed that the pulsation of the heart itself may continue some time after all respiratory movements have ceased. It is easy to imagine the evil results that might follow active depletory measures in the reactive stage, when the force of the pulse usually gives so false an idea of the stamina of the system (for, naturally, without such means, the pulse diminishes in force and frequency as the third stage sets in); but their employment in accordance with *primâ facie* indications may render the ebb fatally low.

The pulse in Yellow Fever is usually full; but it is assumed to be less tense and hard than it may be in other fevers at a corresponding stage, and from a frequency of 100–110 it will rapidly fall to the healthy standard, or even below it, when the period of excitement closes.

The Blood.—Accurate observations on the physical characters and chemical properties of the blood in Yellow Fever are much wanting to improve our knowledge of the pathology of the disease.

The following facts have been observed from time to time by various authorities:—

1. Blood of a bright scarlet color has sometimes been drawn at the very onset of the malady.

2. It gradually acquires a darker hue as the disease advances.

3. As observed on both sides of the heart, arterial and venous blood exhibit no appreciable difference.

4. It may present the appearance of being composed of two differently colored fluids.

5. It may assume a brighter color as it flows from the arm, or on exposure to the air.

6. It may be of the consistence of molasses, or a thin fluid from the commencement, though this is more commonly observed in the third stage, or only after death.

7. In many instances the blood remains without coagulation, or is very slow in the process.

8. The crassamentum appears as if it were undergoing solution at the base.

9. The serum varies in color from a whitish appearance, through yellow and orange, to a red, which has been compared to the tint of water in which meat had been washed.

10. The amount of serum with respect to the clot is smaller than in other cases, and at the commencement than at the close of the malady.

11. In all cases in which yellowness of the skin presents itself, the serum is also found to be yellow from solution of the coloring matters, and the blood globules, broken up from their nummular arrangement, are precipitated to the bottom.

12. The morbid discolorations of the blood, of even healthy persons residing within the range of infection, shows the agreement of Yellow Fever in this particular with what has been observed in the case of other zymotic diseases.

13. Dr. Blair has shown that the dark grumous character of the blood in hemorrhages supports no necessary assumption that the blood within the vessels is of the same nature.

14. Persons who have had much to do with bleeding in Yellow Fever affirm that the odor of the blood, like that of the skin,[1] is quite characteristic of the disease, differing appreciably from the odor emitted by the blood in other fevers.

15. Dr. Davy has demonstrated the acidity of the blood in Yellow Fever, while alkalinity of that fluid is known to exist in other zymotic diseases.

16. Chassaniol has detected a larger amount of urea in the blood, more particularly in that of the third stage, when the urinary secretion has been more or less suppressed. But Professor Rogers,

[1] See Barruel's remarkable experiments, in which even sex has been determined by the odor of the blood, so closely resembling that of the cutaneous secretions.

who proved an excess of salts, contrary to the views of Stevens, was unable to obtain urea in any of the stages. His specimens, however, were believed to have been in a semi-decomposed state. Chassaniol, with great reason, refers the more important symptoms of the adynamic stage to the presence of urea in the circulation.

17. As in other diseases arising from a specific poison, the proportion of fibrine in the blood is much below the normal standard in Yellow Fever; and it is probable that whenever buffing and cupping of the blood occurs in this disease the condition is due to the co-existence of some inflammatory complication.

18. The destruction of the cell wall of the blood corpuscles, said to have been observed by M. de Bienperthuy, is, in the writer's opinion, a doubtful appearance, and certainly not borne out by the researches of Professor Leidy and Dr. Davy.

Tendency to Hemorrhage.—As a hemorrhagic tendency is often developed, more particularly in the third stage of Yellow Fever, it may be well to notice some of its leading features in this place.

Active congestion usually precedes the extravasation of blood in the parts affected, and discharges of this kind have happened from all the outlets of the body without exception. But the special seat of the hemorrhage, and the amount of it, will vary with the type or perhaps with the period or locality, of the epidemic.

Hemorrhagic oozing from the skin, without abrasion, is known at Martinique under the appellation of "sueur de sang." Epistaxis, however, is much more common, and usually happens in the third stage, though it has been known at the close of the first.

Blood may exude from the inner canthus of the eyes, and from the auditory passages, but these hemorrhages are of rare occurrence. Bleeding from the cavity of the mouth, tongue, gums, and lips is usual in every epidemic; and though the blood may be at first well-colored, when it mixes up with the secretions of the mouth its character becomes altered, and it assumes a dirty madder-brown hue, coating tongue and teeth.

The menstrual discharge, when not profuse at this period, appears to exert a salutary influence on the disease; but more active coincident hemorrhage, as in parturition or abortion, is eminently perilous.

Hæmatemesis scarcely ever happens without the passage of blood by stool, which is often critical in its nature.

Ecchymotic abscesses occurring in the neck produce distressing symptoms from their pressure on important parts; and if it be found necessary to relieve them, the writer would recommend a valvular opening. As the patients advance towards recovery, the bloody, ichorous discharge will gradually assume a more purulent character, until finally laudable pus is formed, and the cure goes on in the usual way.

White and Black Vomit.—Up to the commencement of the second stage of the disease, namely, on the second, third, fourth, or fifth day, the ejections from the stomach have been observed to be alkaline; subsequently to which they gradually give an acid reaction, and the discharge of a limpid, ropy, more or less transparent or opalescent, fluid, known as "white vomit," precedes the black, and passes insensibly into it.

Dr. Davy was of opinion, reasoning from analogy, that the white, or precursory, vomit would be found to be of a serous nature, and contain albumen; but if it "remains clear on the application of heat and nitric acid," as stated by Dr. Aitken, the presence of albumen is so far negatived.

As conciseness of matter must be our object in the present article, without entering minutely into the merits of the prolix reasoning of pathologists in favor of the bilious or of the sanguineous theory of black vomit, the following summary may be given as a fair conclusion from the facts adduced.

When hemorrhagic oozing begins to take place from the congested lining membrane of the stomach, the blood *ab initio* intermixes with the existing and concomitant acescent secretions of the organ. The blood globules aggregated in masses lose their colored contents by exosmosis, while they become distended with a thinner fluid, and blend together with a common connecting and finely granular substance, composed of coagulated albumen and the liberated hæmato-globulin. Dark madder-brown flocculi thus result, floating in a more or less homogeneously-tinted or colorless liquid, and this is probably the simplest definition of black vomit.

Free hydrochloric acid is the grand source of the acidity, tested with litmus and brown turmeric papers.

Nitrate of silver throws down a white precipitate, which is redissolved by ammonia, though not by nitric acid.

Liquor potassæ being added to black vomit, in larger quantity than that required to neutralize its acidity, the flocculent sediment becomes dissolved, with the disengagement of ammonia. It effervesces with alkaline carbonates, and Dr. Blair neutralized ℥xij with ℥j of carbonate of potash.

At the request of Dr. La Roche, Professor Rogers examined several specimens of black vomit, with a specific gravity ranging between 1·003 and 1·016, with the following results:—

Albumen
Sulphuric acid in a state of combination
Chlorine
Alkaline bases
Earthy phosphates
Iron
Hydrochloric acid in a free state.

In his communication to Dr. La Roche, he says: "These substances, although not the sole ingredients of the blood, are yet, all of them, with the exception of free hydrochloric acid, constituents of that liquid—a fact which, taken in connection with other characters, and especially the microscopic appearance of the liquids, gives strong evidence that they contain much altered blood; indeed, the presence of several of the substances enumerated, as albumen, iron, and sulphuric acid, seems not to admit of any other explanation, since it could scarcely be possible that in that stage of the disease they were the results of any food remaining in the stomach."

On submitting black vomit to microscopic examination all the changes above noticed with regard to the blood may be distinctly traced out, but no importance can be attached to the presence of torulæ or other humble vegetable organisms, which are by no means constant. The mention of fatty globules, epithelium (except perhaps that of the tubular glands), starch corpuscles, chicken muscle, and other matters introduced from without, only add scientific jargon to the plain facts of the case, and tend to becloud them.

Mr. Wharton Jones states, that in the contents of the hepatic ducts of man and the sheep, extracted by means of a forceps and without injuring the organ, hepatic cells may be detected. This fact is a difficult problem for the microscopic anatomist to solve, but it sinks into insignificance when he is told that Dr. Blair has not only found the glandular cells, but what he conceived to be "the radical secreting ducts of the liver," in black vomit.

The Urine.—The urinary secretion is generally diminished in quantity from the onset of the malady, and in many instances becomes almost or altogether suppressed, when the symptoms of uræmic poisoning may be developed. Dr. Blair, who was an excellent observer, affirms that it is always acid in the first stage of the disease, and gives an alkaline reaction during convalescence, or when bile is present in large quantity.

Albumen makes its appearance generally on the second or third day, and it may be detected in all severe cases by its appropriate tests.

The color of the urine, in passing from its natural standard, changes to a bright yellow, a dirty orange, a greenish brown, and olivaceous black, or to a more or less positive red, from the presence of blood.[1]

The ordinary post-mortem appearance of the kidney, to say nothing of its minute anatomy, precludes the idea of the physical detachment of its capillary vessels, or even the Malpighian tufts, and their escape through the tubular system, so as to be commonly witnessed in the urine, as stated by Dr. Blair. Dr. Aitken observes that this admits of doubt, and that it is not borne out by Blair's own specimens preserved in the cabinet of microscopic preparations of the Army Medical School at Netley. Tube casts, fatty cells, free fat, and blood disks, full and emptied of their contents, with such appearances as often characterize the urine in common jaundice, were the most invariable objects observed by the writer. There was perhaps a large amount of torulæ and allied forms; but, as in the case of the black vomit, notice of them is of little importance.

Morbid Anatomy.—*External Appearances.*—Should the individual die soon after the invasion of the complaint, the bulk of the body may be a little increased on account of a slight puffiness of the skin; but, drained by hemorrhages and with no repair of the vital fluid, emaciation must happen in more protracted cases.

The eye loses the suffused redness it previously exhibited, and the yellowness of the skin becomes more intense; indeed, it sometimes happens that the skin, which could scarcely be called yellow before, acquires that hue after death, and presents

1 "On chemical analysis," writes Dr. La Roche, "urine taken from the bladder after death has been found greatly deficient of urea. In one case 200 grammes (51 drachms) of the fluid contained but one gramme and $\frac{90}{100}$ (about 16 grains) of urea, no uric acid, and 0·45 per cent. of albumen. In another case, 15 grammes ($\frac{1}{2}$ oz.) of the fluid obtained in the same way, gave 0·08 of urea, 2·50 of albumen, and no uric acid. In several other experiments, conducted in the same way, the results were similar. Finally, in one case the urine was examined in the first stage of the disease, and a short time after the death of the patient. In the first the fluid was found to contain, in 100 parts:—

Water	76·08
Urea	2·64
Albumen	0·40
Uric acid	0·08
Earthy phosphates, sulphates, alkaline phosphates, and chlorates	0·80
	80·00

"After death, 20 grammes gave more traces of urea, 0·50 of albumen, and no uric acid."

a remarkable contrast with the rich purple blotches and marblings in the more depending parts of the body.

The extremities of the fingers and toes and the tips of the ears are also darkened with stagnant blood.

The tissues in general appear to become abnormally friable, even though examined very soon after death: this is particularly the case with the muscular system, including the heart itself. The flesh is also of a dingy color, watery, and sodden. It is, however, much paler and softer in those who have suffered much wasting. The connective tissue, moreover, is in general loose and sanguinolent—a condition also observable in malarious Yellow Fever, which is admitted to be a distinct type of disease. The areolar and adipose tissues often exhibit a yellowness similar to those of the skin.

Nervous System.—The brain has been very closely examined in numerous cases, with nearly the same result, namely, that no pathological condition in the slightest degree noteworthy has been detected, even including those cases in which cerebral symptoms, such as active delirium or profound coma, existed before death. (Gillkrest.)

The spinal marrow, on the contrary, usually exhibits a congested state of the vessels, more especially in the lumbar region, where also the arachnoid membrane has been supposed to be in a state of inflammation.

Effusion of blood has been found in the canal, but we cannot be certain of all that has been said about the existence of actual inflammation. The ganglionic system has also been examined, and with apparently still more definite results. Thus, the semi-lunar ganglia, solar, cæliac, hepatic, and neighboring plexuses, as well as the connecting tissue investing them, have been stated, on the authority of Dr. Cartwright and others, to be uniformly in a condition indicative of inflammation. Nevertheless, excellent observers are not wanting who have never been able to discover any decidedly abnormal state of this system.

Respiratory Organs.—The lungs have been carefully scrutinized in epidemics occurring in different countries within the range of Yellow Fever, and the only important particular, not due to other complications, appeared to be small sanguinolent effusions into the connective tissue of the organs themselves, and that immediately beneath the pleuræ.

Circulatory System.—Usually no very abnormal change is observable in the heart, if we except a dusky and flabby appearance which it often presents in common with the muscular system generally. Professor Riddell and others have laid much stress upon the pretty constant molecular degeneration occurring in the muscular fibres of the heart, with a more or less complete obliteration of their transverse striæ; but it is very probable that this condition, which is perhaps more usual than physicians in general imagine, would be detected in the same subjects had they died of any other malady.

In the pericardium, effusions take place, with or without marks of inflammation, and the fluid may be simply serous, purulent or sanguineous; and in one instance thus described by Dr. Bache, the pericardium contained four ounces of a very turbid greenish-brown fluid, resembling black vomit. On pouring this into a bottle and allowing it to stand a short time separated into two portions: that at the bottom was of a white or pale yellow color, while the rest remained without change. An examination by the microscope proved the brownish fluid to be composed of altered blood corpuscles, with less of the granular amorphous matter than is usually found in genuine black vomit. The whitish fluid was pus. The heart itself was stained of a dark color at its base, and the pericardium was minutely injected in points. (La Roche.)

Transparent amber-looking clots have been frequently detected in the cavities of the heart, particularly on the right side. In fifty-four post-mortems made by Dr. Pennell, at Rio Janciro, clots of the same kind were present.

The endocardium commonly presents no signs of inflammation.

The Stomach.—As might be expected, morbid appearances are more constant in the stomach than in any other organ, so intimately associated as it is with the most serious symptoms of the disease. The effusion of the coloring matter of the blood into the subserous connective tissue may give it a yellowish appearance, but, more unusually, no abnormal change presents itself externally.

A certain amount of the matter of black vomit is generally found in the cavity of the stomach, in some stages of conversion, from actual blood to a dark grumous fluid, like that ejected by the patient while yet alive. It would seem as though the mucous membrane of the stomach were called upon to compensate for the defective secreting and eliminating power of the kidneys; and in those cases where little or no black vomit was found in the stomach after death, the lining membrane presented a thickened appearance with a muco-sanguineous coating.

Sometimes the interior of the stomach presents little or no trace of congestion or inflammation where the mucous membrane has been cautiously washed; while at others the little orifices of the tubular glands are seen to be filled up with a dark brown matter that may be dislodged with

the point of a needle. This appearance has been ignorantly spoken of as the open orifices of the hypothetical vessels, known to older anatomists as the "exhalents," in the act of elimination. There appears to be no relation whatever between the amount of congestion observed in the mucous coat and the quantity of matter thrown off from it into the cavity of the stomach. Thus the hemorrhagic oozing may have emptied the capillaries, in some instances, while they still remained congested by the "vis a tergo" filling up the loss in others.

The mucous membrane often becomes mammillated, thickened, the longitudinal folds enlarged, and it is more easily stripped off than usual; but veritable proofs of inflammation, though decidedly present in some cases, are far from being universal in this disease. The tints of congestion, ranging from rose red to claret purple, and dingy gray passing into shades of green and greenish yellow, impart an iridescent appearance to the mucous membrane.

The Intestines present much the same external appearance as the stomach, but the glandular organs, Peyer's patches in particular, are not so uniformly in a morbid state as they are in other fevers. Indeed, the duodenum and upper part of jejunum are often more seriously affected than the ileum.

The Liver is said to become soft and friable in those who die within forty-eight hours, and a serous discharge may take the place of natural bile.

The gall-bladder is usually found empty, or with a small quantity of tarry-looking bile in its cavity.

The *capsule* and fat surrounding the *kidney* have a yellowish appearance, but the organ itself, though prone to fatty change, even during the short period of the disease, is yet not so decidedly involved in it as the liver. Indeed, under ordinary circumstances, an amount of congestion in the liver, only producing the slightest biliary derangement, would be a very serious occurrence in the kidney; and though the latter is a much smaller organ, it would appear to occupy a longer time in passing on to a state of fatty degeneration. Little livid spots and ecchymoses are sometimes found in the infundibulum and pelvis, and pus has been found in the pelvis and ureter. Dr. Pennel notices, besides the manifest congestion of the kidney, that the papillæ yielded upon pressure a glutinous tenacious exudation sufficient to interfere mechanically with the escape of the urine. This, however, the writer is disposed to think was in great part composed of the forcibly extruded epithelial lining of the little tubes in a manner well known to microscopic anatomists.

The *pancreas* and *spleen* present no constant pathological change worth noticing, and the same may be said of the *urinary bladder*, if we except the possible occurrence of a dark grumous fluid, closely resembling black vomit, recorded by one or two observers.

Prognosis.—It is a difficult matter to form a reliable prognosis in almost any stage or variety of Yellow Fever, for the very cases that would strike the physician as affording most promise of speedy recovery may prove to him, by a rapidly fatal issue, how uncertain his judgment must be. Still, there are symptoms or circumstances which experience has shown to be usually of good import, and others again which augur badly or point to an almost immediate dissolution.

Favorable Signs.—If the pulse approximates without reaching 110 in the febrile stage;[1] if it preserves an even and normal force and frequency when the third stage sets in; if the urine be in good quantity and exhibits no disorganization of the kidney, under the microscope—the presence of albumen in small amount is to be regarded more as a usual than an unfavorable symptom—if epigastric tenderness and irritability subside with the febrile state, a good hope of the case may be entertained.

Unfavorable Signs.—Dr. Jackson gives the following as indicative of danger, founded upon his experience of the disease, occurring on the south coast of Spain:—

1. A sudden invasion by the fever, with intense pain of the head and eyeballs, accompanied by sickness and vomiting.
2. The fever being ushered in by a fit, convulsions, or apoplectic stupor, or outrageous delirium.
3. A torpid, heavy, or statue-like aspect of countenance gave strong suspicion of danger.
4. A dry, rough, milk-white, or swollen and red tongue indicated danger.
5. Distress and anguish at stomach, with pain at the epigastrium, forcible eructations, or explosions of flatus from the stomach, gave impression of much danger.
6. Obscure hiccough marked danger.
7. A ghastly appearance, with a faint nauseous odor from the body, indicated extreme danger.
8. Yellowness of the skin, with turgid veins on the conjunctivæ in the latter stage, "always decisive of a fatal issue."
9. Torpor of the skin—to such an extent as to be insensible to the stimulation of blisters and sinapisms—is ranked among dangerous signs.

[1] The frequency of the pulse as a rule being less in Yellow Fever than in others.

10. Extreme dampness or extreme dryness of the skin indicates great danger.

11. Petechiæ are suspicious: streaks, or patches, of livid green, or violet color, are almost certain indications of approaching death.

12. Vomiting of black matter, like the grounds of coffee, is reported a sign of the highest danger; [but]

13. Vomiting of bitter bile, whether green or yellow, even with straining and severe retching, affords a sign of comparative safety.

14. Black watery stools, with shreds, "are of the worst prognostics" (as quoted by Martin).

To the above may be added:—

1. A very weak pulse on the invasion of the disease.

2. A pulse much exceeding 110 in the febrile stage.

3. Sudden and excessive lumbar pain, or rachialgia.

4. Countenance swollen, tense, and bloated, or on the contrary much pinched up, or terror-stricken.

5. Fiery redness, with prominence of the eyes, or on the other hand a pearly whiteness of the conjunctivæ.

6. Widely dilated pupils, indicative of cerebral complication.

7. Suppression of urine, or deposits indicating a serious condition of the kidney.

THERAPEUTICS. — *Treatment.* — There can be little doubt that whatever is to be done in Yellow Fever should be done quickly, and the earlier a clear diagnosis is formed the better, if any hope may be reposed in medicine.

We have it on the authority of physicians of experience, that the disease has been cut short by the timely administration of remedies that have been tried and found wanting at a later period. Dealing with the subject in a purely philosophical light, the evidence before us is not as conclusive as it could be wished; on this point, and "in the present state of our knowledge, we can only be guided by the common principles of medicine; for it is plain that a specific mode of cure cannot be suggested until the nature of the specific cause is known. We know also that the disease manifests itself with various degrees of intensity in different cases, and how much of any happy recovery depends upon the stamina of the system, or upon the means employed, is above all things difficult to determine—a fact which should never be forgotten by those who may be zealously inclined to advocate their own suggestions." Even a brief notice of the various modes of practice adopted by medical men from time to time, in the treatment of this disease, would occupy more space than would be of advantage in the present article; but we shall pass the more important remedial agents in review.

The *pediluvium* on the first invasion of the malady is now very largely employed by American practitioners, and the indication is further carried out by the use of warm drinks to excite diaphoresis and a genial warmth. This may be followed by a brisk purgative, composed of calomel, gr. vi–x, jalap gr. x, and ginger gr. iij, in the bolus form, which will be found the most convenient and certain mode of administering medicines of this kind on an extended scale.

[Bleeding was largely resorted to and favorably reported upon by Dr. Rush and others, a century ago.[1] Although this practice is by general consent now ruled out, it cannot be ignored that the relief sometimes following moderate spontaneous hemorrhages affords a natural suggestion in its favor. Leeches to the epigastrium, during the first day or two of the attack, have certainly sometimes done good.—H.]

A dose of calomel exceeding ten grains would appear to be rather experimental or empirical than based upon a sound physiology. No one has been able to advance a therapeutical principle to warrant it, in opposition to the evil effects so often known to follow in its wake. The same also may be said of large doses of quinine, which, in Yellow Fever at least only tends to impede secretion and derange the circulation within the head. The employment of this medicine at all is more suitable to the convalescent than to the patient more immediately under the influence of the disease. It is perhaps more useful in such febrile states as exhibit a periodicity in their recurrence. Should it be thought expedient to prescribe quinine in Yellow Fever, small doses frequently repeated are to be preferred, so that any resulting prejudicial effect may be observed in good time and the medicine simply omitted without doing much mischief.

As the bowels are commonly sluggish in Yellow Fever, an enema may be necessary after the first dose above suggested; in which case one of turpentine, as recommended by Drs. Copland and Smith, will

[1] Mathew Carey, in his "Short Account" of the Yellow Fever of 1793 in Philadelphia, says: "The efficacy of bleeding in all cases not attended with putridity was great. The quantity of blood taken was, in many cases, astonishing. Dr. Griffits was bled seven times in five days, and ascribes his recovery principally to that operation. Dr. Mease, in five days, lost seventy-two ounces of blood, by which he was recovered when at the lowest stage of the disorder. Many others were bled still more, and are now as well as ever they were."—H.]

prove to be the most beneficial, as it in general brings away feculent and normal-looking stools. Turpentine, in drachm doses, by the mouth, is advocated by the same authorities, but of its use in this way the writer has had no experience.

The most must be made of the time now remaining until retching and vomiting set in. Acetate of ammonia, nitrate of potash, nitrous ether, and the tincture of squills[1] and henbane, may be combined and administered in periodical doses, with lime juice for drink. In this way excessive febrile action will be moderated, the action of the kidney and the skin preserved, and even if the reaction is defective no further depressing influence can be exerted.

Of all the symptoms of Yellow Fever the most distressing, to both patient and physician, is irritability of the stomach. It is so constantly present, and so often uncontrollable, that the knowledge of every available means of checking it is of the greatest importance.

In the epidemic of Yellow Fever on board H.M.S. *Icarus*, in the West Indies, a few drops of chloroform prepared the stomach for the reception and retention of food, "but the dose should be repeated a short time before food is again taken, as the effect of chloroform is transitory."

Creosote and hydrocyanic acid have been used with the same intention, but the valuable suggestion of chlorodyne made by Dr. Aitken, would bid fair to supersede every other, if we may except that of lime-water, in this connection. Lime-water was used with great benefit on the *Icarus*, on the recommendation merely of its known therapeutic properties in the practice of medicine. But it had long previously been employed by the American physicians, and with a higher object, namely, the correction of acidity in the stomach, due to the presence of free hydrochloric acid. Dr. Hosack used lime-water most successfully in combination with milk, and with porter when the milk could not be retained. More stress ought to be laid upon the importance of this agent by English writers. A chalk mixture has also been resorted to with the same intention.

Professor Frost, of Charleston, strongly advocates the chlorate of potash; and it is richly worth extensive trial, from its known valuable properties in adynamic states generally, oxygenating the blood, and aiding in elimination by its action on the skin and kidneys. It is fair, however, to state that some cases, in which it was tried, were not very satisfactory.

Tannic acid, besides the astringency which it exerts upon the smaller vessels, is reputed to have the property of diminishing the irritability of the stomach.

Pepsine, in regulated doses, so as to assist an organ incapable of discharging its own functions efficiently, requires further trial. In connection with pepsine, and the internal use of ice in the intervals of its exhibition, the essence of beef should be unremittingly supplied and suited by dilution and seasoning to the nature of the case. This will be the time also for stimulants—our great object being to obviate the tendency to death, and to sustain the vital power in its struggle with the foe. Champagne, in particular, will be beneficial, when it can be obtained. The ward-room cook of H.M.S. *Icarus* had very nearly succumbed at this crisis, but he rallied immediately on the administration of a stout glass of rum and water, and recovered steadily.

The Yellow Fever poison is evidently of an intoxicating kind, and a marked difference is apparent between the mild and irritable delirium produced by it, when thrown out in contact with the brain substance and the comatose state brought about by those principles, urea in particular, that accumulate in the circulation in consequence of the ineffective action of the kidneys and liver. When the semi-comatose patient is aroused, he may be made to answer rationally; but in the delirium, which is characterized by vigilance and irritability, this can scarcely be expected, and even a coherent reply may be in league, as it were, with the mental derangement. Morphia certainly has the effect of allaying this excitable state and procuring sleep. Here, again, Dr. Aitken suggests chlorodyne, as the administration of "opium in any form," on good therapeutic grounds, is objectionable. [Ice, dissolved slowly in the mouth and swallowed, is very refreshing and suitable during the febrile paroxysm. So are effervescent drinks; probably none are better than carbonic acid water (soda water) cooled with ice. Spice poultices applied to the epigastrium may materially assist in relieving the vomiting and epigastric tenderness. Cold or cool sponging will be appropriate at an early stage; even hot bathing has been found serviceable in the later prostration.—H.]

VARIETIES, AND THEIR CLASSIFICATION.—Notwithstanding all that has been written on the subject of the Yellow Fever, the gist of nearly every attempted classification of its varieties amounts to little more than a grouping of them in accordance with the several degrees of comparison. Indeed, the phases presented by different epidemics, and by the individual cases in the same epidemic, with no possibility of the interpolation of other complaints to be confounded with it, would

[1] Many practitioners would prefer to omit the squills and nitrate of potassium.—H.]

give systematists more than they could easily accomplish to define and arrange intelligibly. The types, therefore, are exceedingly variable, and little practical benefit can accrue from their nice discrimination; but, when they do not transgress broad boundary lines, they may afford a more precise knowledge of the symptoms and pathology of the disease as a whole. It is notorious that a classification, founded upon the experience of one epidemic, may be quite inapplicable to another; consequently, whoever has to do with one, naturally makes a classification for himself. On this account, systems have become so numerous, that we can only give place to a notice of one of the best of them, namely, that put forward by Dr. La Roche of Philadelphia, whose masterly work on Yellow Fever, with a bibliography of sixty-one pages, has afforded the writer great assistance in the composition of this article.

According to Dr. La Roche, Yellow Fever (as a genus) is divisible into two species, viz., 1, the Inflammatory; and 2, the Congestive; though these are often connected by cases in every conceivable degree of transition from one to the other.

1. The inflammatory species appears under three grades, viz. (*a*) the Intense; (*b*) the Mild; and (*c*) the Ephemeral.

From all that has been previously said in the symptomatology, the character of these grades may be easily conceived. In the *first*, the fever is active and short, and death commonly happens before the accession of black vomit; in the *second*, the fever is more protracted, and may even exhibit partial remissions before the final stage sets in; while the *third*, as its name implies, is of short duration, and easily amenable to treatment.

2. The Congestive species is marked by the passage of the disease "as it were directly from the first sign of indisposition to the last stage, without going through that of reaction." Of this species there are four grades: viz. (*a*) the Aggravated; (*b*) the Adynamic or Typhoid; (*c*) the Walking; and (*d*) the Apoplectic.

(*a*) The Aggravated grade is attended from the commencement with considerable prostration, giddiness, stupor, and loss of memory, delirium, or coma. The tongue is natural, or with white patches and red tip and edges. The countenance is livid and apathetic, and the skin yellow or bronzed. Hemorrhage occurs from one or more of the natural outlets, and the patient keeps up a low monotonous wailing. In some cases the pulse is nearly natural, the tongue clean, and the stomach calm; but these are attended with excessive restlessless, anxiety, and distress, soon followed by black vomit and fatal collapse.

(*b*) The Adynamic or Typhoid grade occurs in persons deficient in vital power, ushered in with chills, followed by burning heat, partially distributed over the body, viz., principally on the under parts of the arm and inner surface of the thighs. The pulse is small and weak. The skin assumes an olive hue, and is covered with petechiæ or vibices. Hemorrhages are common from the natural outlets, or into the connective tissue beneath the skin, or amongst the muscles.

(*c*) The Walking grade.—Here the functions of organic life appear to be at first alone implicated, those of animal life remaining unaffected. The patient, though sometimes in bed, is found more frequently walking about his room. He only feels weak, but his eye is watery, and his countenance dull and listless; his pulse grows fainter and fainter, until at last he is overtaken by black vomit, and death speedily ensues.[1]

(*d*) The Apoplectic grade.—The patient is more or less suddenly struck down with stupor or coma, and death, preceded by convulsions, soon follows. The pulse is rather weak, and finally becomes faltering. The skin is cold and clammy, or sometimes dry and flabby. "In the mean time the patient lies as if stunned, with dilated pupils and an expression of gloom upon the countenance. From this unpromising state an effort at reaction occasionally takes place, but this scarcely ever leads to a successful result. More generally, the patient becomes perfectly comatose, the eyes assume a glassy appearance, the pulse fades away, involuntary discharges and profuse hemorrhages supervene, and death soon ensues."

Some few years ago[2] the writer surmised the possibility of the extension of the range of Yellow Fever to our own shores, all favorable conditions being fulfilled, and this has been realized in the late epidemic of Yellow Fever at Swansea, reported in a very able manner by Dr. Buchanan. The circumstances are briefly the following:—

On the 9th September, 1865, the *Hecla* barque, laden with copper ore, returned from Cuba to Swansea, with one case of Yellow Fever on board, three having proved fatal on the voyage home.

The remaining case (James Saunders) and two convalescents were sent on shore. Soon, also, the crew had left the ship and distributed themselves over the town, and two passengers with their luggage were landed. Moreover, a good many people boarded the vessel as she entered the dock.

An outbreak of Yellow Fever thus

[1] Several well-marked cases of this grade occurred on board H.M.S. *Icarus*.

[2] Health of the Navy for 1860.

originated ashore, and in a well drawn-up table of the cases Dr. Buchanan shows their invariable connection with the source of the malady, directly or indirectly, adding further proof of its infectious nature, should such be required.

Note.—As bearing upon the question of infection, the following important quotation is from the remarks of Deputy Inspector-General R. D. Mason of Port Royal Hospital, recorded in the Statistical Report of the Health of the Navy for 1866 :—

"During the Christmas quarter nine cases of Yellow Fever have been received into hospital, of which eight terminated fatally. The first case received was that of the commanding officer of the gunboat *Nettle*. The *Nettle* had been employed between the 1st of October and 25th of November at Morant Bay and Port Morant, spending a week alternately at each place. At Morant Bay this officer resided on shore, passing scarcely any time on board. The servant died of Yellow Fever at Morant Bay on the 22d of November, after about four days' illness; and a staff assistant-surgeon, who lived in the same house and who attended him, was subsequently attacked with the same disease and died on the 10th of December.

"Up to the time that the *Nettle* left Morant Bay no other cases were known to have occurred, and there had been no unusual amount of sickness, nor any cases of Yellow Fever, as far as I have been able to ascertain.

"The surgeon of the *Cadmus* states, however, that as far back as the month of July, it was reported that a merchant-ship from St. Thomas, then lying at Morant Bay, or one of the eastern ports of the island, had cases of Yellow Fever on board: cause and effect are also shown in the fact that the chaplain of the *Cadmus* had on several occasions visited the commander of the *Nettle* during his illness, and that the surgeon was at his bedside about an hour before his death, and that the *Cadmus*, previously a healthy ship, had subsequently sent eleven cases of Yellow Fever to hospital."

Circumstances like these may appear very unimportant at the time of their occurrence, but it may be safely affirmed that such are always traceable in connection with the communication and spread of Yellow Fever.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

By J. Netten Radcliffe.

Definition.—An acute, epidemic disease, characterized by profound disturbance of the central nervous system; indicated, at the outset, chiefly by shivering, intense headache, or vertigo, or both, and persistent vomiting; subsequently by delirium, often violent, alternating with somnolence, or with a state of apathy or stupor; an acute painful condition with spasm—sometimes tetanoid—of certain groups of muscles, especially the posterior muscles of the neck, occasioning retraction of the head; and an increased sensitiveness of the surface of the body. Throughout the disease there is marked depression of the vital powers; not unfrequently collapse; and in its course an eruption of vesicles, petechiæ, or purpuric spots, or mottling of the skin, is apt to occur. If the disease tend to recovery, the symptoms gradually subside without any critical phenomena, and convalescence is protracted: if to a fatal termination, death is almost invariably preceded by coma. After death, the enveloping membranes of the brain and spinal cord are found in a morbid state, of which the most notable signs are engorgement of the bloodvessels, usually excessive, and an effusion of seropurulent matter into the meshes of the pia mater, and beneath the arachnoid.

Synonyms. — (a) *Technical*:—Cerebro-spinal fever[1] (*Royal College of Physicians*); cerebro-spinal arachnitis; typhus syncopalis; tifo apoplettico tetanico; typhus cérébro-spinal (*Boudin*); cerebral typhus; epi-

[1] Since the completion of this article the Royal College of Physicians, in its "Nomenclature of Disease," has adopted the following designation and definition of this malady: "*Cerebro-spinal Fever*. A malignant epidemic fever, attended by painful contraction of the muscles of the neck, and retraction of the head. In certain epidemics it is frequently accompanied by a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain and spinal cord and their membranes are found on dissection." It is, however, determined to retain the name by which this affection was described in the first edition of this System of Medicine, and for the simple reason that such name, while sufficient for the purpose of recognition, conveys no opinion as to the nature of the disease.—Editor.

demic meningitis (*Stillé*, U. S.); petechial fever (*G. B. Wood*, U. S.); fever with cerebro-spinal meningitis (*S. Gordon*); malignant purpuric fever (*W. Stokes*); malignant purple fever; nervo-purpuric fever (*Mapother*); malignant purpuræ (*M'Swinney*); pestilential purpuræ (*Banks*); febris nigra (*R. D. Lyons*).—(b) *Popular:*—Spotted fever (*New England*); cold plague (*Southern States*, U. S.); Kolik, Nackenstarre, Genickkrampf (*Germany*); Nacksjuka, Dragsjuka (*Sweden*).

DESCRIPTION OF THE DISEASE.—I. *General Symptoms:*—Epidemic cerebro-spinal meningitis is observed in three principal forms: A.—*Simple*, in which the symptoms indicative of disorder of the nervous centres predominate throughout the whole course of the disease; B.—*Fulminant*, in which the depressed state of the vital powers, with profound blood-change—as shown by hemorrhage of various forms into the cutis—characterizes the disease; and C.—*Purpuric*, in which the cerebro-spinal symptoms, and the symptoms which mark blood-change (*petechiæ*, *purpuræ*, *vibices*, &c.), and flagging of the vital powers, occur together. The proportion in which the three forms of the disease are manifest varies considerably in different epidemics. In every outbreak cases are observed which link, by insensible gradations, one form with another; while in other, and rarer cases, the characteristic symptoms of the three forms are merged together. Continental and American writers have described an *abortive* form of the disease, the term being given (*a*) to certain anomalous symptoms observed in communities among which the disease is active: and (*b*) to sundry symptoms characteristic of the malady, but of transitory duration: such as severe cephalalgia, a sense of dragging at the back of the neck, or actual slight retraction of the head; cardialgia, enteralgia;—these symptoms often ending contemporaneously with the appearance of profuse perspiration, or epistaxis.

(A.) *Simple Epidemic Cerebro-spinal Meningitis.*—In the majority of the cases before the onset of the disease the patient suffers from more or less indisposition. There are discomfort in the head, neuralgic pains in the back, the principal groups of muscles, and the abdomen; failure of the appetite, indifference to exertion, perhaps also slight shiverings, and a quasi-febrile state. These indications of disordered innervation may persist from three to seven days, or may be manifested only during a few hours, before the confirmed malady fully declares itself. But in numerous cases the onset of the disease is sudden and characteristic. In both classes the accession of the malady is declared by similar well-marked signs. Acute shivering is followed or accompanied by severe, commonly intolerable, headache, or vertigo, or both; and after a short interval, or contemporaneously, profuse and irrepressible vomiting takes place, rarely preceded by nausea. Or vomiting may be the initiatory symptom, the shivering, headache, or vertigo following quickly after. The intensity of the symptoms marking the onset of the disease is remarkable and characteristic. The sickness is often, and from the outset accompanied by severe abdominal pain, apparently neuralgic; and not unfrequently this pain precedes the disorder of the stomach, as the cephalalgia precedes mental confusion. In like manner, the shivering ushers in, or is accompanied by, an acutely painful state of the muscles, more or less general, the forerunner of spasm. Cephalalgia and delirium, abdominal neuralgia and vomiting, and myalgia and spasm are the principal morbid factors of simple epidemic cerebro-spinal meningitis. They distinguish the malady, and the varying prominence with which they occur in different outbreaks gives rise to many diversities in the grouping of symptoms during the progress of the disease. The onward course of the disorder is usually rapid. The headache continues, often without a lull; vertigo occurs frequently; and after the lapse of a very brief period, measured usually by a few hours, the mind becomes confused, and, in some cases, a state of restlessness supervenes not unlike that observed in delirium tremens. The mental confusion assumes the form of muttering delirium, with periods of somnolence, often interrupted by cries provoked by the intense cephalalgia, or by the neuralgic pain elsewhere; or the patient falls into a state of apathy or stupor, from which he may be partially roused, but into which he relapses when left undisturbed, the mind acting as in a dream; or there is acute and violent delirium. Contemporaneously with, or immediately prior to, the mental disturbance, the painful state of the muscles increases, certain groups being more manifestly affected than others, especially the posterior muscles of the neck, the muscles of the spinal column, and those of the lower extremities. The pain, often of an acutely neuralgic character, shoots along the spine and limbs, and across the walls of the abdomen. Partly as a voluntary action, partly as a consequence of spasm of the painful muscles, the head is drawn backwards. The retraction thus arising is one of the commonest and most characteristic symptoms of the disease. As the malady advances an actual or apparent tetanoid contraction of other groups of muscles may occur, the trunk most frequently being curved backwards, and the legs bent upon

the thighs. At the same time there may be fleeting spasmodic action of some of the muscles of the face, and occasionally of the eyeballs; or in some cases tonic contraction of these muscles, giving rise to the so-called sardonic laugh, or to persistent strabismus. In many cases cutaneous sensibility is much exaggerated, and very frequently a vesicular or roseolar eruption is developed, the former particularly about the lips. The aspect of the patient as the disease advances is dependent upon the degree of pain, the state of delirium or stupor, and extent of spasm which may be present. The countenance is rigid and contracted, the expression of face betokening acute pain; or it is dominated by the delirious fancies; or reflects the mental torpidity; or is distorted by spasm. There is frequently a slight suffusion of the eyes, altogether different from the dusky appearance of typhus; and the face is commonly pale and sunken, seldom and only transitorily flushed and swollen, except when affected more or less extensively by the vesicular eruption. The surface sometimes moist, sometimes dry, rarely gives to the hand a sensation of febrile heat, although the temperature of the body ranges above the normal standard. The pulse from the outset is wanting in firmness, and the indications of defective tone increase as the disease advances. The respiration exhibits no marked disturbance, excepting an increase of rapidity witnessed during accessions of pain and restlessness, and in the advanced stage of the malady the diminution dependent upon failing circulation and innervation. The alimentary canal, apart from the vomiting, which usually ceases as the disease becomes fully developed, presents little indication of disturbance. The tongue is as frequently clean and moist as dry, foul and discolored; and the bowels may be either costive or loose, the former, perhaps, more commonly than the latter. In some outbreaks, indeed, costiveness has been marked and almost general, but in others diarrhœa has been prevalent. The renal secretion is rarely much disturbed.

As the malady proceeds, if it tends towards a fatal termination, the spasmodic symptoms increase, the patient becomes comatose, and death may occur either from asphyxia or exhaustion in from ten or twelve hours to seven or eight days. If the disease be prolonged beyond this period, various secondary lesions are apt to occur, especially certain inflammatory states of the eyes and the ears, the mischief in the former organs being shown by ulceration of the cornea, iritis, and sometimes suppuration of the globe; in the latter by less obvious structural changes during life except as indicated by deafness. Or there may be paralysis affecting one-half of the body, or one side of the face, or one of the limbs, or an isolated group of muscles. Or there may be an inflammatory state, with sero-purulent effusion into one or more of the large joints. Or, finally, the patient may fall into a state of marasmus and nervous exhaustion, often protracted and not rarely fatal. If the malady proceed to a favorable termination without any of these sequences, health may be recovered in from three to four weeks. If the progress of the disorder, otherwise favorable, be interrupted by one other complication, the period of recovery is uncertain and often long postponed.

(B.) *Fulminant Epidemic Cerebro-spinal Meningitis.*—In the fulminant form of the malady the onset is without premonition. The patient suddenly falls into a state of collapse. The surface of the body has often a cyanotic aspect, and is cold and clammy to the touch, or covered with profuse perspiration, the face being not rarely shrunken and livid, and the eyes deep sunk as in the algide stage of cholera. There may be some shivering at intervals, more or less pain of the head, and occasional vomiting, sometimes of a grumous black or coffee-colored fluid. Drowsiness, if not present at the outset, rapidly supervenes, followed by or concurrently with delirium. Coma, rarely other than the precursor of death, quickly succeeds. In the mean time purpuric spots show themselves over the surface of the body generally, red or purple and circumscribed in the beginning, but rapidly becoming black, and often extending their margins so as to form irregular inky blotches, or streaks, or great patches; and not unfrequently several of the spots become gangrenous. Sometimes the purpuric spots appear contemporaneously with the collapse at the outset of the attack. The respiration is preternaturally slow, and the pulse (if it has not been absent at the wrist from the beginning) falls with the progress of the disease. The urine is loaded with albumen. Life may be extinguished in less than *five hours*, or it may be prolonged for two or three days. Recovery from this form of epidemic cerebro-spinal meningitis is not unknown, but it is an exceedingly rare event.

(C.) *Purpuric Epidemic Cerebro-spinal Meningitis.*—In the purpuric form of epidemic cerebro-spinal meningitis, the symptoms which distinguish the simple and fulminant forms of the disease occur combined together in various proportions, some cases approximating more or less closely to the latter, others, as is most common, to the former variety of the affection. Thus concurrently with shivering, intense headache, vomiting, rachialgia and retraction of the head, there may be depression of the vital powers approaching collapse, or collapse itself, with the

development of petechiæ or purpuræ, vibices, ecchymoses, hemorrhage from mucous tracts, delirium, coma, and rapid dissolution. In by far the greater number of cases, however, the disease follows the course of simple epidemic cerebro-spinal meningitis; but within twenty-four hours, or from this period to the fourth day, or still later in the progress of the malady petechiæ or purpuræ are developed more or less copiously, and occasionally hemorrhage occurs from the mucous tracts. This phase of epidemic cerebro-spinal meningitis does not appear to be more fatal than the simple form of the disease. It has been observed more commonly in the United States than on the continent of Europe, and it was the principal variety which occurred during the outbreak in Ireland in 1867.

II. *Special Symptoms.*—1. *The Nervous System.*—Headache is almost constant, and it is remarkable for its early and persistent severity. At the outset it is not localized in any particular part of the head. It may be referred to the forehead, the sides, the vertex, or the occiput; or it may be general. Later in the disease, the occiput is, perhaps, most commonly the seat of pain. The intensity of the headache is, as a rule, peculiar. The patients describe the pain as sharp, lancinating, stabbing, plunging, tensive, throbbing, boring, or crushing. It is so intolerable as to elicit groans and cries from the sufferer; often, even during delirium or stupor, the exclamations, the contraction of the forehead, and the manner in which the hands are moved towards the head, show that the pain continues. In young children this state closely resembles that which is so significant of tubercular meningitis. The headache may cease when the disease has become fully developed, or as is probably more common, it may persist throughout the whole course of the malady so long as consciousness remains. Occasionally, indeed, when recovery takes place, it will continue far into the period of convalescence.

Rachialgia is rarely absent. It is sometimes general throughout the spinal region, but more frequently it is limited to the loins, the dorsal region, or, as is most usual, to the posterior part of the neck. Occasionally the pain radiates from the neck to the extremities and walls of the abdominal and thoracic cavities. In rare cases the pain has commenced at some point of the peripheral nervous system, and spread thence to the back, occurring in paroxysms. This pain has the same character as the cephalalgia, and the words (intolerable, atrocious, tensive, &c.) used to indicate the nature of the latter may be employed also to describe the former. It is augmented by movements, and its chief seat is in the muscles of the spinal column.

The nuchal pain and its consequences constitute one of the most characteristic signs of the disease. Frequently, at the outset of the malady, this pain is preceded by a dragging sensation at the back of the head. As the pain increases in intensity, the head is voluntarily thrown back to relieve all strain upon the exquisitely sensitive muscles. Or, in conjunction with the pain, spasm of the affected muscles occurs, and the head is forcibly drawn backwards. Among the popular terms of the disease, those arising from this symptom (Nackenstarre, Genickkrampf, Nacksjuka, &c.) are very prominent. When the rachialgia is more diffused, and the pain extends also to the limbs, adapted or spasmodic contractions of the trunk and lower extremities are apt to occur. Rachialgia is not present in the fulminant and in severe cases of the purpuric forms of the affection. It is noteworthy that pressure on the spinous processes, during the most acute rachialgia, rarely causes pain.

Enteralgia and other Neuralgic Pains.—Abdominal pain, neuralgic in character, and more or less closely linked to the pain in the course of the spine, is not unfrequent, and it is often closely associated with uncontrollable vomiting. In some epidemics, as in that of 1865 on the Lower Vistula, enteralgia was so common among children seized with cerebro-spinal meningitis that it gave rise to the trivial designation "belly-ache," as one of the popular names of the disease. Neuralgic pains in the limbs, referred to in connection with rachialgia, are less common than like pains along the course of the spine and in the abdomen.

Increased Sensitiveness of the Surface of the Body has been described as frequent in several outbreaks. During the late prevalence of the disease in the United States (1861–67), cutaneous hyperæsthesia is said to have been a characteristic symptom of the malady in its fully developed state. During the outbreak on the Lower Vistula, an increase of cutaneous sensitiveness was also observed very commonly, but it was not regarded by Dr. Burdon Sanderson as a characteristic symptom, but "a mere consequence or interlude of pain:" being, in fact, an excessive tenderness experienced during intermissions, or after the cessation of pain.

Spasm.—Sufficient care has not always been taken to discriminate between apparent and actual spasm in this disease. Tourdes, in 1843, showed that the retraction of the head and curvature of the spine did not in all cases arise from a spasmodic contraction of the muscles, but that the position was not rarely voluntarily or instinctively assumed by the patient as most conducive to relief of the spinal pain. Dr. Burdon Sanderson con-

firmed this observation of Tourdes, so far as retraction of the head was concerned, in 1865. In the cases observed by him, in which the head was apparently drawn backwards, it was practicable to extend the seemingly contracted muscles, although the effort gave rise to exquisite pain and instinctive resistance. There was not any tension of the muscles except such as arose from this resistance; no tightness was felt so long as they were at rest. "It was not till the neck was completely extended that the muscles became hard, and even then the hardness was not for a moment comparable to that which is felt in tetanus." The position in bed of the patients observed by Dr. Burdon Sanderson was that which would produce the greatest relaxation of painful groups

[Fig. 13.

Cerebro-spinal Fever (J. Lewis Smith).]

of muscles. There can be no doubt, however, that spasm is a frequent accompaniment of epidemic cerebro-spinal meningitis. In the clonic form it is witnessed in some cases as transitory contractions of the facial muscles, cramps of the extremities, the convulsive agitation and trembling referred to in the general description as somewhat like what is observed in delirium tremens, very rarely in local convulsions of a single limb, and still more rarely in general convulsions. Tonic spasm of the muscles of the face, jaws, (trismus), and gullet, and of the limbs and trunk, may also occur, giving rise to true opisthotonos, emprosthotonos, or general tetanic rigidity of the trunk and limbs.

Paralysis is not of very common occurrence during the progress of epidemic cerebro-spinal meningitis. Hemiplegia has been occasionally noticed, and paralysis more or less complete of one or both extremities, upper and lower, of the muscles of deglutition, of articulation, and of certain other associated groups, the latter chiefly towards the close of the malady. The general paralysis noticed by some writers was usually significant of, and indeed a part of the phenomena of, approaching dissolution.

The *special senses* do not often manifest much change, except as a consequence of certain structural lesions. Increased, sometimes exquisite, sensitiveness of sight and hearing has occasionally been noticed, concurrently with augmented sensitiveness to other external impressions, especially towards the close of the malady, when complete consciousness returns. Amaurosis has also occurred, without apparent change in the ocular apparatus. It may be noted, moreover, of the eye and sight, that occasionally there are strabismus and double vision. The pupils may be normal in aspect and action, or they may present various changes. They may be dilated or contracted, or one dilated and the other contracted, or they may exhibit curious alternations of contraction and dilatation under the influence of the same degree of light. Both the *eyes* and the *ears* are liable to undergo certain structural lesions, consisting in well-marked inflammatory changes. These commence, in the former organs, sometimes in the cornea, sometimes in the deeper tissues. Most commonly keratitis is set up, ending in opacity or ulceration; and, in the latter case, the iris may become involved. Or, iritis may occur independently, with effusion of lymph or pus, and the consequences thereof (synechia posterior and distortion of the iris are particularly noted). Among more deeply-seated changes may be mentioned opacity of the lens or of the vitreous humor, separation of the retina from the choroid, purulent infiltration, or atrophy of the eyeball. The ear suffers, perhaps, more frequently than the eye. Deafness is probably more common than defects of vision, and it is largely dependent upon inflammatory changes set up in the organ, and particularly affecting the lining membrane at the vestibule and semicircular canals. Occasionally, the external meatus has been affected, and a profuse purulent discharge has flowed from it. These lesions of the organs of sight and hearing

may occur either early or late in the course of the disease. The sense of *smell* very rarely suffers. Its loss in one nostril has been recorded in a single case, and this was, perhaps, dependent upon inflammatory changes in the lining membrane of the nose; as purulent discharge from the nostrils has occasionally taken place in other instances.

Vertigo is sometimes observed as an initial symptom of the disease in conjunction with the cephalalgia. Instances are recorded in which the first accession of the disease was marked by severe giddiness, during which the patient either staggered about like a drunken man, or turned round several times, and then fell.

Delirium is rarely absent. It varies much in character, and may occur at any period of the seizure. It may be quiet or violent, transitory or more or less persistent. It sometimes, but rarely, forms one of the symptoms of invasion, when its access is sudden and its character acute. It may supervene with violence after the malady has continued from several hours to two or three days. In the acute form of delirium, the patient is very noisy, and often so violent as to require restraint. Sometimes it happens that paroxysms of furious excitement occur with intervals of placid delirium. Hence the necessity of great watchfulness in the care of these cases. Most commonly the delirium follows closely upon the initiatory symptoms, and is aggravated as the disease advances. At the beginning, the confusion of thought may not be so great but that the patient can be roused so as to answer questions intelligibly. Later, the incoherence becomes much greater, and is usually accompanied with considerable agitation. Much difference is observed, not only in the degree of impairment of the consciousness, but also in the periods of manifestation of the impairment. In some cases, the delirium occurs chiefly during the night; in others, and very commonly, it alternates with periods of somnolence or of quietude. In the more persistent cases there are usually exacerbations. If the disease tend to a fatal ending, the delirium is followed by coma; if to recovery, consciousness is, as a rule, gradually recovered: but, at times, a period of stupor intervenes between the subsidence of the delirium and returning perception. In the slightest cases of the malady the delirium may be transient only, taking place at intervals, and chiefly during the night. In the gravest cases, when death occurs in a few hours, delirium is most commonly present. The duration of the delirium depends entirely upon the nature and duration of the case. Instances are recorded in which furious delirium has occurred for three nights in succession. In other instances a delirious state has persisted more or less continuously for fifteen days.

Stupor and Coma.—In not a few protracted cases, delirium is followed by a prolonged state of stupor, the patient lying completely indifferent to external impressions. In six cases observed by Dr. Burdon Sanderson, in which there had been violent delirium at the outset, this state lasted from one week to five weeks, the mean duration of the several cases being nineteen days. The observer remarks, however, that as four of the cases "emerged from their stupor in a state of complete deafness, there was much difficulty in limiting accurately the period of unconsciousness." Sometimes the state of stupor supervenes without the intervention of violent delirium. *Coma* occurs in nearly all fatal cases, and is, indeed, generally the forerunner of death.

2. *The Digestive System.*—The uncontrollable *vomiting*, which is one of the characteristic initiatory symptoms of the disorder, is an effect of the cerebral mischief. Most frequent at the beginning of the malady, the vomiting diminishes as the disease advances, occasionally increasing during exacerbations. The matter evacuated, after the stomach has been emptied of food, is usually of a greenish or yellowish color and bitter taste, and is composed largely of bile; more rarely it is viscid and white. Occasionally, in the fulminant and purpuric forms of the malady, a grumous black or coffee-colored fluid is vomited. In several outbreaks, the vomiting of large quantities of *Ascarides lumbricoides* has been specially noted. The *buccal cavity* and *tongue* do not exhibit any particular signs, except in those rare cases in which there is hemorrhage from the gums. As a rule, the tongue is clean and natural at the outset, and its subsequent state depends upon the degree of febrile excitement which may be set up, or upon the development of a typhous state, when it may become foul with various well-known aspects, or with dry and black sordes accumulating on the teeth. From the beginning of the attack the appetite for food is destroyed, whatever the state of the buccal cavity; and sometimes there is much, at others, insatiable, thirst. The *bowels* are more commonly costive than the reverse. In some outbreaks costiveness has been of general occurrence. Diarrhœa, late in the disease, is not unfrequently to be attributed to the previous administration of purgatives, and involuntary stools are usually one of the accompaniments of complete nervous and vital prostration.

3. *The Urinary System.*—In the simple form of epidemic cerebro-spinal meningitis the urine does not exhibit any marked change. It may be more abundant, and slight deposits of lithic acid

may occur. In the fulminant, and in severe cases of the purpuric forms, it commonly (in the first-named form perhaps invariably) contains albumen, sometimes in large amount, and occasionally cylindrical casts and blood-corpuscles. Retention or incontinence of urine has occurred in the progress of the disease.

4. *The Respiratory System.*—In all the graver cases the respiration is more or less altered. It is sighing, labored, or interrupted. Dr. Burdon Sanderson writes of the outbreak on the Lower Vistula: "In all severe cases, whether of children or adults, the breathing was embarrassed in proportion to the general gravity of the symptoms. This embarrassment was marked by a slow labored inspiration, followed by quick respiration and a long pause,—that condition of breathing which is so frequently observed in continued fever (especially in typhoid), and is often called suspirous. In all the fatal cases which came under my notice, the most prominent symptoms which preceded death were those which indicate impairment and perversion of the respiratory function. As the breathing became more hurried and difficult, the general depression became more intense, the pulse became weaker and quicker, and the temperature of the skin more elevated." Dr. S. Gordon records a case, fatal in less than five hours, in which the respirations rapidly fell to nine per minute, the pulse at the time being 120.

5. *The Circulatory System.*—The cardinal point with respect to the circulation, as indicated by the radial *pulse*, is defect of arterial tension. This has been common to all epidemics, with hardly an exception; and the exceptional instances have probably been more apparent than real. The frequency of the pulse does not admit of general statement. It has a wide range. In the epidemic on the Lower Vistula, the pulse in six adult cases observed by Dr. Burdon Sanderson varied from 56 to 98, the average beats being 85. In several cases noted by the same observer, "its frequency varied considerably from day to day, without apparent relation to the condition of the patient in other respects." During the Philadelphia outbreak of 1866, in 98 cases observed by Dr. W. H. H. Githens, the pulse varied from the normal beat to 150 per minute in uncomplicated cases, and reached as high as 160 in two cases in puerperal women. "It was in all very weak, with a dichrotic tendency, sometimes entirely imperceptible in the radial artery, and always interrupted by slight pressure."

6. *The Cutaneous System.*—In respect of dryness or moisture or feeling to the touch, the *skin* presents no constant condition; but, in numerous cases, it is the seat of various forms of eruption of remarkable interest. The extent of prevalence or predominance of one or other of these different forms of eruption has varied considerably in the numerous recorded outbreaks. In the epidemics which have occurred in the United States, *petechiæ* have been so common as to have given rise to the popular name of the disease (*spotted fever*), and to have induced Dr. G. B. Wood, Professor of the Theory and Practice of Medicine in the University of Pennsylvania, to adopt as the technical designation of the disease the term *petechial fever*. During the recent outbreak in Ireland (1866–67), *purpura* was the predominant form of eruption, and Professor Stokes proposed to designate the malady *malignant purpuric fever;* other observers also suggested terms founded upon this character. In the outbreak on the Lower Vistula (1865), an herpetic eruption was most common. In all the greater outbreaks, each form of eruption mentioned in the definition of the disease has been observed; but the proportion of cases in which one or other form of eruption has prevailed has varied greatly in each outbreak. In some of the earlier outbreaks in the United States few cases occurred in which a petechial eruption was not noted. Of 98 cases admitted into the Philadelphia Hospital (Blockley) in 1866, 36 had petechiæ; 13 mixed petechiæ and erythema; 9 erythema and urticaria; 3 indistinct petechial mottling; and 37 no eruption at all (Githens). In the outbreak on the Lower Vistula the proportion of cases exhibiting an eruption was comparatively small; in the recent outbreak in Ireland, large. The forms of eruption observed are as follows: (*a*) *Vesicles.* A vesicular eruption (*eczema*, Hirsch), sometimes herpetic in character, chiefly appearing in the vicinity of the lips, but occasionally extending over the sides of the face, diffused more or less on the trunk, or showing itself in patches on the limbs. This symptom has occasionally taken the form of shingles. It is most commonly noticed in the simple form of the disease, but it may take place in either of the other forms, and when associated with purpura, the vesicles may be flattened and rest upon a livid base, presenting a horrible aspect. This form of eruption may appear as early as the second day. (*b*) *Purpura.* 1. True *petechiæ.* 2. *Purpuric spots*, varying in size from a split pea to half-a-crown, with more or less extensive effusions of blood, or of its coloring matter, into the cutis (*vibices*, *ecchymoses*). The spots have sometimes a regular, sometimes an irregular, even a ragged, outline. Their size may remain fixed from the time of their first appearance, or it may increase largely or rapidly. They may be of a

light or dark red color at the outset, subsequently becoming purple and black; or, as is most common, they may from the beginning be dark purple or black, their blackness being often fittingly likened to that of ink—the eruption resembling "spots" or "splashes" of that fluid. They may appear on the trunk or limbs only, or they may be scattered copiously over the whole surface of the body, including the face. The purpuric spots are frequently hard to the touch, the margin being defined, and giving the impression to the fingers of being raised above the surface; sometimes a vesicle forms above several of the spots, and gangrene of the adjacent tissue takes place. Dr. S. Gordon writes of the recent epidemic in Ireland: "Many cases are accompanied by a distinct eruption, which comes out with great rapidity; is found over all parts of the body, but chiefly on the lower extremities; is of a very dark color, sometimes a very deep brown, or purple, or even black. The spots are of various sizes and shapes, some small and round, others large and irregular; some appear like large spots of very black purpura, only more mottled and more irregular in color and shape; others are more confined, and raised above the level of the skin, consisting of an effusion into its substance: many patients die in this stage, but in some the disease progresses, and these spots are absorbed, leaving a yellowish mark under the cuticle; or they pass into superficial gangrene, which was spreading at the time of the patient's death, or is healed with loss of substance." Purpuric spots are sometimes, although rarely, one of the earliest signs of the fulminant and purpuric forms of the malady; or they may occur at any period during the more advanced stages. Usually they appear at some period during the first four days, chiefly perhaps during the first or second day. Sometimes, with or without the purpuric spots, there is a cyanosed aspect of the skin, or a peculiar livid mottling. During recovery the purpuric spots gradually lose their refined character and fade away, passing through the different stages of color which mark a healing bruise. (*c*) *Roseola, erythema, &c.* Rose-colored spots or patches are occasionally observed; also erythema, more or less diffused, a rubeoloid eruption, and urticaria.

7. *Temperature.* — The temperature of the body, as marked in the axilla, is heightened in every case; except, perhaps, those accompanied by profound collapse from the beginning. In many cases this heightened temperature is found contemporaneously with the invasion of the disease; in other cases there is no conspicuous increment until the second or third day. When the characteristic symptoms of the malady are developed, the temperature rarely falls below 100° Fahr., and, as the disease advances, it ranges in adults from 100° to 105°, in children sometimes even higher. There is no constant or conspicuous difference between the morning and the evening temperature, as in typhus and typhoid. A steady fall marks the decline of the disease and the approach of recovery; a rapid fall ushers in collapse or death.

Complications.— The course of the disease is liable to be modified by certain complications. Of these the chief are as follows: — (*a*) Thoracic inflammations: pleurisy, pneumonia, bronchitis, or pericarditis. Dr. S. Gordon describes œdema of the lung and diffuse pulmonary apoplexy. (*b*) Swelling or inflammation of the parotids. (*c*) Inflammation of the large joints, marked by swelling and pain, and sometimes ending in sero-purulent effusion. This complication, in its less aggravated form, has been described by some writers as rheumatic. (*d*) An inflammatory condition of the eyes and ears, as already noted. (*e*) Bed-sores. Large, deep, black sloughs occurred in four cases out of 161 treated in the Philadelphia Hospital in 1866. (*f*) The course of the disease has also been complicated by the supervention of other maladies, namely (1) *Intermittent fever*, or certain paroxysmal phenomena simulating malarious poisoning: a complication which has led to erroneous notions of the nature of the disease. In the outbreak on the Lower Vistula cases were observed in which regular or irregular intermissions took place that could not be assigned to a malarious origin. (2) *Typhoid fever*, the two diseases prevailing simultaneously in the same district. The symptoms of both diseases more or less modified, pursue their course together, and the characteristic lesions of typhoid fever as well as of epidemic cerebro-spinal meningitis are discovered after death. (3) Measles and scarlet fever. (4) Cholera (Levy).

Duration.— In the outbreak on the Lower Vistula, the most acute cases terminated fatally in from 12 to 72 hours. Cases of less intensity, but in which the patient eventually died in a typhous state, lasted from 8 to 14 days, the characteristic symptoms of the disease persisting to the end. In the more protracted, or complicated cases, from 5 to 8 weeks have passed before a patient entered upon convalescence, and death has taken place in the 6th or 7th week. Of the cases observed in the Philadelphia Hospital (1866), the duration of those which ended fatally was from 24 hours to 14 days; of those which recovered, from 20 to 30 days, the acute symptoms rarely exceeding a fortnight.

In the outbreak of 1866 in Ireland Dr. S. Gordon reported a well-marked case which ended fatally after less than *five hours'* duration. A large proportion of the fatal cases in that outbreak died in from 10 to 48 hours; in other cases the fatal ending did not occur until the close of the second and during the course of the third week of the disease. The duration of the disease, as shown by death, may be clearly stated; as marked by the beginning of convalescence, it does not admit of definite description. Moreover, convalescence is often very protracted. The course of the disease towards recovery is sometimes interrupted by *relapses*.

Termination. — The disease terminates, after a longer or shorter period of convalescence, in health; or it entails during convalescence a series of physical or mental ills; or it ends in death. The rate of *mortality* of the disease is the measure of probable recovery. It varies much in different outbreaks, but is at all times formidable. Among the cases observed in the Philadelphia Hospital in 1866 the mortality was 33 per cent.; in the Hardwicke Hospital, Dublin, the same year, the mortality was 80 per cent. Dr. Stillé remarks that, "while ten epidemics in various places, occurring between 1838 and 1848, presented an average mortality of 70 per cent., a similar number occurring during the decade from 1855 to 1865 gives an average mortality of about 30 per cent. This remarkable fact would seem to indicate a gradual decline of power in the epidemic." The minimum rate of mortality recorded is 20 per cent. The proportion of fatal cases is greatest, and the duration of these cases least, at the commencement of an outbreak. The *sequelæ* which interfere with the restoration of the patient to perfect health are: —Deafness; impaired vision from structural changes in one or both eyes; paralysis of one or more limbs or of certain groups of muscles; impaired memory; carbuncles, and boils. Dr. S. Gordon describes a case in which the patient "recovered from all the acute symptoms, but gradually passed into a state of almost organic life. He ate, drank, and slept well; he passed solid feces and urine without giving any notice, yet, evidently, not unconsciously; he was excessively emaciated, and there was a peculiar mouse-like smell from him; he seemed to understand what was said to him, but he could not answer; he never called for anything; his breathing was rather slow; his pulse, 120; his heart acting with a peculiar strong jerking motion; his eye was quite well, as also his knee (he had suffered from ulceration of the right cornea and immense effusion into the right knee-joint); he could draw his legs and arms up to him; but he could not use his hands at all." Such was the condition of the patient fifty-eight days after the invasion of the disease.

Mode of Death.—Death chiefly occurs from (*a*) asphyxia, caused by damage to the respiratory nerve-centres; (*b*) from asthenia; and (*c*) in some of the fulminant cases probably from necræmia, so profound are the changes observed in the blood.

Diagnosis.—In some instances the disease approximates in certain symptoms to *typhus* or *typhoid*, and it occasionally prevails contemporaneously with both maladies. But the history of the development and progress of the disease, with the absence of characteristic eruption, will usually clear up any doubt. From *sporadic spinal meningitis* the disease is distinguished by its epidemicity, the almost constant concurrence of cerebral disorder, the tendency to cutaneous eruptions, the great mortality, and the rareness of protracted or permanent paralysis or contraction of the lower limbs. The distinction between the disease and *cerebral meningitis* is less defined as to particular symptoms, especially in children, but the mode of development of the malady will rarely leave much room for doubt during an outbreak. *Tetanus* (so-called idiopathic), with which it is suggested that epidemic cerebro-spinal meningitis may, under certain states of spasm, be confounded, never manifests the early grave cerebral symptoms which occur in the latter disease. The tetanoid contraction also observed in epidemic cerebro-spinal meningitis, is rarely, if ever, as in tetanus, aggravated by sudden and painful spasms. The grouping of the symptoms in the two diseases is, moreover, altogether different. Dr. S. Gordon points out the possibility of confounding the purpuric form of epidemic cerebro-spinal meningitis with malignant measles, which malady has often prevailed at the same time. The last-named disease may resemble the fulminant form of the first-named in several respects, particularly the rapidity of development, the dark color of the eruption, and the rapid appearance of petechiæ; also in the sudden and often extreme collapse which accompanies the invasion of the affection. But the eruption of measles rarely loses its characteristic form, and the affection of the respiratory passages is commonly present, while purpuric spots and patches are seldom observed. Dr. S. Gordon also states that he has known several cases in which the earlier symptoms of epidemic cerebro-spinal meningitis in young excitable females have been mistaken for *hysteria*. Dr. Murchison records a case which presented the symptoms of cerebro-spinal

fever, including severe headache, moaning, retraction of the head, rigidity of arms, and vomiting; but in which, after death, the duration of the case having been ten days, "no appreciable lesion of the membranes of the brain or spinal cord could be discovered, and the cause of death was ascertained to have been uræmia from contracted kidneys, and recent pericarditis."[1]

Prognosis.—At the best, the prognosis of the disease is very grave. The mortality may be equally great in each of the three varieties, and petechiæ and purpura do not, as in other acute diseases, necessarily indicate an aggravated degree of danger. In 50 per cent. of the cases recorded by Dr. Githens, in one of the least fatal outbreaks known, petechiæ were present, and it is especially remarked that neither this nor any other form of eruption had "any reference to the prognosis." But when hemorrhage into the cutis is extensive, either from the number or the size of the spots, and is accompanied by marked signs of vital prostration, it indicates an extremity of danger, although not a certainty of death. The disease is more fatal among infants and young children than among youths and adults in the prime of life; but, in some outbreaks, the latter have suffered most. After thirty years of age it becomes more dangerous. Life is most endangered in the earlier days of the disease, particularly during the first five. But danger is present at all periods of the malady, and the convalescent is not entirely safe until health is fully restored. Of the special symptoms, whether of excitement or depression, the rules of prognosis hold good which apply to other highly fatal acute maladies.

Morbid Anatomy.—The essential anatomical characteristics of the disease, found after death, are hyperæmia, often intense, of the pia mater of the brain and spinal cord; with more or less copious subarachnoid and interstitial effusion into the meshes of the congested pia mater, either of serum, or of a transparent, gelatinous material, or of purulent matter: the latter more frequently than either of the two former. The purulent effusion is of greenish or yellowish color, and is sometimes flaky. It has been found in a case in which death took place in less than five hours from the invasion of the disease (S. Gordon). The extent to which these appearances are observed and the amount of effusion varies greatly in different cases. No part of the encephalic or spinal pia mater and arachnoid may be free, or certain portions alone may be affected; but effusion is limited to the sub-arachnoid space, and does not occur into the arachnoid cavity. Under the microscope, according to Dr. Burdon Sanderson, the gelatinous material is "always found to consist of cell-like bodies, either adhering to each other so closely that they could not be completely separated, or imbedded in a transparent interstitial substance; while the sero-purulent liquid which occupied the spinal sub-arachnoid space, and in some cases the ventricles, exhibited corpuscles and granules floating freely. The cell-like bodies, although in general resembling pus corpuscles, did not present that uniformity of size and character which is met with in normal pus. They were usually, but not always, of regular circular contour, and varied in diameter from $\frac{1}{3500}$th to $\frac{1}{1200}$th of an inch. Occasionally they exhibited the appearance of an external cell-membrane, but in most instances this could not be made out even in perfectly fresh exudations—as, *e. g.*, in those cases which were examined as early as eight hours after death. They invariably contained numerous granules, some of which were cleared away on the addition of acetic acid. Those which remained were highly refractive, but did not assume any special form of arrangement. The interstitial substance was beset with granules, some of which were albuminous, others fatty. It was most abundant and distinct on the surface of the spinal arachnoid, where it infiltrated the fine connecting tissue and minute bloodvessels of the pia mater."

For the rest, the nervous system of the brain and spinal cord is usually gorged with blood, unless death has taken place late in the course of the disease. The visceral arachnoid is frequently thickened and opaque. Dr. Klebs[1] has shown that often, where the eye detects opacity alone, the microscope reveals extensive cell formation, purulent in character. Softening of some portion of the spinal cord has sometimes been observed; and Mr. J. Simon thinks that, "for practical purposes, the state of the covering membranes of the nervous centres may be regarded as a mere index of changes more or less distinctive, which those centres in their own intimate composition have at the same time undergone; and hence it is that the essential phenomena of the disease during life consist in disturbances, more or less grave, of the functions of these all-important organs." Dr. Klebs describes œdema of the medullary substance and loosening

[1] Proceedings of the Pathological Society, vol. xviii.

[1] Zur Pathologie der Epidemischen Meningitis. Virchow's Archiv. Band xxxiv. British and Foreign Medico-Chirurgical Review, 1868.

of the nervous elements, and suggests that this may explain the extensive motor disturbances which have been observed in some cases, in which purulent effusion into the subarachnoid space has been slight. The same author has observed also purulent encephalitis.

In fatal cases of the simple and purpuric forms of epidemic cerebro-spinal meningitis the characteristic anatomical lesions are almost invariably found. In the fulminant form of the disease they are often absent. The cases in which there is no indication of morbid change in the nervous centres are exceedingly few. It has been suggested that in these cases death has occurred so rapidly that there was insufficient time for the formation of a structural lesion. In connection with this explanation the case recorded by Dr. S. Gordon must be borne in mind, in which purulent effusion was found, although the whole duration of the attack was under *five* hours. Practically the apparent absence of characteristic anatomical change in the nervous centres, in certain rare cases of epidemic cerebro-spinal meningitis, is a phenomenon analogous to that which sometimes occurs in rapidly fatal cases of malarious, variolous, and scarlatinous poisoning, in which the characteristic eruptions or lesions of the diseases have not been developed.

No lesions manifestly peculiar to epidemic cerebro-spinal meningitis have, as yet, been found in other organs of the body. Such lesions as occur elsewhere than in the coverings of the brain and spinal cord usually have a definite relation to the thoracic, abdominal, or genito-urinary complications which may have happened during the progress of the malady. Dr. Klebs, however, describes certain changes in the intimate structure of the kidneys and liver, which he believes to be characteristic of the disease. In the fatal cases of the purpuric form of the affection recorded by Dr. S. Gordon, and other writers, an excessive fluidity of the blood was noted.

History and Geographical Distribution.—The scientific history of epidemic cerebro-spinal meningitis dates only from the fourth decennium of the present century. At that period the disease was, for the first time, clearly distinguished as an independent malady; and with the light then obtained, outbreaks which had occurred earlier in the century, in various localities of both the Eastern and Western hemispheres, and had been recorded under other names, were recognized as of a similar character. It has been sought, indeed, to show that epidemic cerebro-spinal meningitis has probably existed from remote periods (Tourdes, Boudin). The probability may be admitted, for the first recognition of a malady as an independent affection does not necessarily imply that the malady is new.

In 1837 epidemic cerebro-spinal meningitis broke out in the southwest of France, and prevailed in various localities of the district intervening between Bayonne and La Rochelle, and along the whole line of the Pyrenean frontier. Dax, Bordeaux, Auch, Foix, Narbonne, and Perpignan suffered, as well as the two cities previously named. The disease, according to Boudin, at the commencement and during the continuance of this outbreak, chiefly showed itself among troops in garrison. During 1837 and 1838 the garrisons of Bayonne, Dax, Bordeaux, Rochefort, and La Rochelle suffered. From 1838 to 1841 the disease was prevalent among the garrisons of southeastern France, particularly those of the valley of the Rhone. Thus it broke out at Toulon, Marseilles, Aigues-Mortes, Nîsmes, Avignon, and Pont-Saint-Esprit. In the course of the four years 1839–40–41–42, the malady appeared in succession among the troops occupying the fortresses of Strasburg, Schelestadt, Calmar, Nancy, Metz, and Givet. From 1839 to 1842 it prevailed among the forces at Versailles, Saint-Cloud, Rambouillet, and Chartres. Those stationed along the coast of Brittany, at Brest, L'Orient, Nantes, and Ancenis, suffered in 1841; and during 1840 and 1841 the disease manifested itself among divers detachments of a regiment scattered at Laval, Le Mans, Château-Gontier, Tours, and Poitiers. It was during the outbreak—of which the most remarkable episode is thus sketched by Boudin—that a scientific knowledge of epidemic cerebro-spinal meningitis was first obtained. From 1837 to 1848 inclusive, forty-seven outbreaks of the malady were recorded in thirty-six of the eighty-six departments into which France was then divided. These outbreaks were distributed in the departments of the Loire, Rhône, Bouches-du-Rhône, Bas-Rhin, Seine, Seine-et-Oise, Landes, Basses-Pyrénées, Charente-Inférieure, Gard, Vaucluse, Var, Moselle, and Loiret. The three first-named departments suffered most. In 1840 the disease appeared in Naples and prevailed in the Papal States. The same year it broke out among the French garrison at Douera, Algeria, and during the next seven years it attacked numerous towns and localities of the province, affecting the civil population, both European and native, as well as the military. In 1844 an outbreak of the disease took place among the civil population of Gibraltar; and in 1846 the malady showed itself slightly in Ireland among the inmates of the Rathdown, South Dublin, and Belfast workhouses, and several

cases occurred among the population of Dublin. During 1849 and 1850 the disease was prevalent to some extent among the French troops in Italy, and in the last-named year several localities of France suffered from it. Epidemic cerebro-spinal meningitis appeared in Denmark in 1841, and prevailed in that country until 1848. The disease was first noticed in Sweden in 1854, this country again suffering from it in 1861. In Norway the malady broke out in 1859, and it prevailed in that country more or less until 1867, if not to a later period. During 1860 the disease was prevalent in Holland; and the same year it was widely spread in Portugal. In 1863, 1864, and 1865 an extensive outbreak occurred in North Germany; and in 1866 the malady broke out in Dublin and elsewhere in Ireland. Cases were recorded in St. Petersburg during 1866 and 1867.

In the United States (where the disease may be traced back to the commencement of the century), epidemic cerebro-spinal meningitis became prevalent about the same time that it exhibited great activity in Europe. From 1842 to 1850 inclusive, a series of outbreaks took place in the States of Kentucky, Tennessee, South Illinois, Mississippi, Arkansas, Alabama, Pennsylvania, Massachusetts, New York, and North Carolina. After this period there would appear to have been an interval of comparative inactivity. In 1861 the disease broke out in North and Central Missouri, and from that time to the present it has prevailed, more or less extensively, in almost all, if not all, the States of the Union, with the exception, perhaps, of the Pacific States. In 1862 outbreaks were recorded in Connecticut, Kentucky, Indiana, and Tennessee; in 1863, in Rhode Island; in 1864, in Pennsylvania, Ohio, Illinois, New York, Maryland, Massachusetts, and Vermont; and in 1865, in North Carolina and other Southern States. During 1867 and 1868 the disease was active in several States.

It must be borne in mind that these historical notes very imperfectly represent the probable prevalence and geographical distribution of the disease. They simply include a brief summary of outbreaks which have come under the notice of thoughtful observers who have published their observations. The history of the malady in the British Islands is, perhaps, less liable to error from this source. The earliest recorded outbreak of the disease occurred in Ireland during the early months of 1846. It broke out to a very limited extent among the boys living in the Rathdown Union, South Dublin, and Belfast workhouses; and two cases, both in females, one aged 17 years, the other 36 years, were admitted into the Hardwicke Hospital, Dublin.[1] Prior to this outbreak, there is not any trustworthy history of the presence of epidemic cerebro-spinal meningitis in the British Islands. It is not improbable, however, that the disease existed at Blackaton, in Devonshire, in 1807;[2] and at Sunderland in 1830.[3] Dr. Benjamin W. Richardson saw, he believes, a case at Mortlake, Surrey, in 1843.[4] From the time of the outbreak in 1846, cases of a similar malady were occasionally observed in Dublin, until the latter half of 1850, when they became more common.[5] There is no further notice of epidemic cerebro-spinal meningitis in Ireland until the year 1865, when cases began to be again observed in Dublin.[6] A case of cerebro-spinal meningitis was observed by Dr. Samuel Wilks, in each of the three years 1856, 1858, 1859, in the metropolis.[7] In October 1859, a fatal case of cerebro-spinal disorder with petechial eruption, came under the notice of Dr. Henry Day, in the vicinity of Stafford. In this case, hyperæmia of the meninges of the brain and spinal cord, and copious effusion of fluid at the base of the brain, were discovered after death. A similar but more rapidly fatal case was also observed by Dr. Day, in the Stafford General Infirmary in September, 1865.[8] The largest and most fatal outbreak of epidemic cerebro-spinal meningitis which has occurred within the limits of the United Kingdom began in Ireland in March 1866, and attained its chief development in the subsequent winter. Its effects were almost entirely limited to the sister island, and the brunt of the outbreak fell upon Dublin. Other localities affected, during the first year of prevalence, were Tullamore, Parsonstown, Mitchelstown, Thurles, Clondalkin, and the Curragh camp. Subsequently cases were recorded in the counties of Cork, Waterford, Clare, Galway, Meath, Down, &c. The cases were not very numerous in Dublin; and in the country towns they were comparatively few. It is noteworthy that, as in the earlier outbreaks in France, the military in Ireland, in proportion to their strength; suffered prominently from the disease. In some of the country districts

[1] Dr. Robt. Mayne, Dublin Quarterly Journal of Medical Science, 1846, vol. ii. p. 95.

[2] Mr. Henry Gervis, Medico-Chirurgical Society's Transactions, vol. ii.

[3] Dr. John Scott, Medical Times and Gazette, 1865, vol. i. p. 515.

[4] Social Science Review, May 1865, p. 398.

[5] Dr. McDowell, The London Journal of Medicine, 1851, vol. iii. p. 858.

[6] Dr. Kennedy, The Medical Press and Circular, June 12, 1861, p. 551.

[7] The Lancet, April 15, 1865, p. 389.

[8] Clinical Histories and Commentaries 1866, pp. 3–7.

cases were recorded among the troops alone, or among persons in immediate connection with them.[1] In January and February 1867, an outbreak of a disease characterized by severe rigors, tetanic convulsions, intense neuralgic pain in the head and upper part of the trunk, increased sensitiveness of the surface, obstinate vomiting, restlessness, and, in one instance at least, by a dark purple eruption, but of which not a single case died, took place at Bardney, in Lincolnshire, a village about ten miles east of Lincoln, on the verge of a fen country, and having a population of 1500, the bulk of whom are engaged in agricultural pursuits.[2] Two cases of epidemic cerebro-spinal meningitis were recorded in London in the summer of 1867. One, a case of the fulminant form of the malady, in which death occurred in twenty-seven hours, took place in June ;[3] the other, a case of the purpuric form, in which death occurred in seven days, took place in June.[4] Since the attention of English practitioners has been more fully directed to epidemic cerebro-spinal meningitis by the late outbreak in Ireland, so-called sporadic instances of the disease have been noted in various parts of the kingdom, under circumstances which lead to the surmise that the malady is not so rare among the population as had previously been supposed.

The peculiarity of distribution of the disease in the British Islands, its epidemic manifestations being limited to one portion of the kingdom, and chiefly, even in recurrent outbreaks, to a small section of the population of that portion, is not an isolated phenomenon. Notwithstanding the wide geographical prevalence of the malady as shown by the foregoing details, it must not be concluded that this prevalence represents a general diffusion of the disease among the different populations during the periods of its activity. The outbreaks of epidemic cebro-spinal meningitis, as a rule, are limited to small sections of a population, and its distribution is by a series of isolated outbreaks, rather than by extensive spreading. This was shown remarkably, as already described, during the outbreak in France in 1837 and following years, when the ravages of the malady were principally confined to certain garrisons, and even to small sections of a garrison, without affecting the surrounding population. A like limitation of the disease to certain detachments of troops was observed during the recent war in the United States; and the restriction of the malady to small portions of workhouse populations, as in the first outbreak in Ireland, is an analogous phenomenon. Perhaps the sole outbreak in which an extensive diffusion of the disease among a community has occurred was that in the province of Dantzic, in 1864-65. The tendency to reproduction in a locality, as in Dublin, was particularly observed during the great outbreak in France from 1838 to 1848, when the disease reappeared again and again among the forces in Bayonne, Versailles, and Avignon, notwithstanding changes of garrison. The freedom of England and Scotland from epidemic outbreaks of so widely spread a malady is very remarkable; particularly if the seeming occasional cases of the disease to which reference has been made are to be regarded as true examples.

Etiology.—(*a*) *Predisposing Causes.*—*Age.* The personal liability to the disease is not governed in any definite manner by age. In some epidemics children, in others young people, in others again adults of from thirty to fifty years, have suffered in greatest proportion. — *Sex.* Generally, and in some outbreaks very markedly, *males* are more liable to the disease than *females.*—*Profession.* During the outbreaks of the disease in France from 1837 to 1849, a peculiar proclivity to the disease was observed among soldiers. But in subsequent outbreaks in France, and wide-spread outbreaks elsewhere, no special liability to the disease was manifested among any vocation.—*Climate and Seasons.* In the Eastern hemisphere our knowledge of the disease is limited to Western and Central Europe and Algeria, the northern boundary of the district not passing beyond lat. 61° N., the southern not beyond lat. 35° S.—the one extreme closely approaching the arctic, the other the torrid zone. In the Western hemisphere the records of the malady are confined to the populous districts of the eastern division of the United States, from lat. 30° N. to lat. 48° N. It is noteworthy that the northern and southern limits of distribution in both hemispheres but slightly overlap the isothermal lines 5° and 20°. *Season* acts as an unquestionable and powerful predisposing cause of epidemic cerebro-spinal meningitis, which is especially a disease of the cold months. Of 216 local outbreaks in France and the United States, 166 prevailed between December 1st and May 31st; 50 in the other six months of the year. In Sweden, of 417 local outbreaks, 311 took

[1] Dr. E. D. Mapother, and Staff-surgeon Dr. Jeffrey A. Marston, The Lancet, July 6, and July 13, 1867; also Transactions of the Epidemiological Society, vol. iii. p. 118, and p. 129.

[2] G. M. Lowe, M.B., The Lancet, June 26, 1867, p. 790; Mr. Geo. Newnham Woolley, The Lancet, Aug. 3, 1867, p. 130.

[3] Dr. Edward Crisp, The Lancet, June 22, 1867, p. 773.

[4] Dr. Thomas Clark, The Lancet, July 13, 1867.

place in the former period of the year, 106 in the latter (Stillé). During the recent outbreak in Ireland, the brunt of the disease fell between January and July, 1867. Of 85 outbreaks in various parts of Europe and the United States, noted by Hirsch, 33 prevailed in winter, 24 in winter and spring, 11 in spring, 1 in spring and summer, 2 in summer, 1 in summer and autumn, 1 in autumn, 1 in autumn and winter, 3 in autumn, winter, and spring, and 6 prevailed throughout the whole year.—*Locality and soil* do not, so far as yet ascertained, exercise any manifest influence over the disease. It has been observed indifferently on low grounds, on highlands, and on soils of the most various character.—*Sanitary conditions.* No definite relation exists between the occurrence of the disease and the sanitary state of habitations or of individuals. It has prevailed in some epidemics alike among the affluent and the impoverished—among those who are well-fed, well-housed, and well-clothed, as among those who are ill-fed, ill-housed, and insufficiently clothed. In certain outbreaks, as in that on the Lower Vistula, the prosperous classes suffered to a much less extent from the malady than the poor and miserable, who were subjected to privation and to much foulness of persons, dwellings, and atmosphere.

(*b*) *Exciting Causes.*—Fatigue has been mentioned as an exciting cause. In some of the early outbreaks of the disease among French troops, France being at war at the time, fatigue apparently exercised a determining influence. Again, during the recent outbreak in Ireland, the malady appeared very early among a "flying column" of troops occupied in the suppression of the Fenian disturbance, and exposed to great fatigue and inclemency of weather. But fatigue has played little or no part in determining the disease among the civil population, especially among children and the inmates of workhouses and prisons. — *Cold.* The marked predominance of the disease in the winter and spring months has suggested a causal connection with cold. Hirsch has submitted the question to a detailed examination, and with this result: that, although we cannot exclude the suspicion that the temperature of winter and spring may have some direct effect upon the genesis of the disease, "the modifications in the mode of living incidental to these seasons exert, in a far higher degree, an influence favorable to the presence of this as of many other infectious maladies."[1] —*Certain Insanitary States.* There is not any constant or even common relationship between any insanitary state and the appearance of the disease. Neither foulness of house and its surroundings, nor of the atmosphere, whether from putrid emanations or from overcrowding, nor impurity of any other kind, has any determinate relation with epidemic cerebro-spinal meningitis. But Hirsch remarks[1] of the outbreak in the province of Dantzic in 1865, that "the disease prevailed exactly in that season of the year in which, on account of inclement weather, many individuals were crowded together into small and dirty rooms kept constantly closed by their occupants, and from which all ventilation was excluded, and in which the before-mentioned unfavorable hygienic conditions (dampness, great filth, and an atmosphere loaded with putrid emanations) were extremely perceptible." The causes here suggested have been held to be not altogether inoperative in other and more circumscribed outbreaks.—*Communication of the sick with the well.* The great majority of observers have come to the conclusion that the disease is incommunicable from the sick to the well. Among the minority who hesitate to accept this deduction without reservation are Professor Hirsch, Professor Stokes, and Mr. J. Simon. The facts which suggest the possibility of the active cause of the disease being portable in some way are of the following character:—(*a*) A child was seized with epidemic cerebro-spinal meningitis, and died. A second child of the same family was attacked with the malady a few days later. The day following the attack of this child, the mother, who slept in the same bed with it, sickened of the disease.[2] (*b*) 1. On the 8th of February, 1865, a youth, aged 20 years, was attacked with the characteristic symptoms of epidemic cerebro-spinal meningitis. He was nursed by a woman from another village. The youth died, and after his death the woman returned home. She soon sickened, and she died of the epidemic disease on the 26th February. There had been but one case previously in the village. To the interment of the woman came a family from another locality, the funeral obsequies, as customary in the district, being performed with the coffin opened. After the return home of this family, a child, three months old, sickened immediately of meningitis and died within twenty-four hours. Then a man who had accompanied the family to the interment was attacked with the disease, and died on the 2d of March. Lastly, a girl, in the same locality, who had also been at the funeral, was seized, and died on the 7th March. 2. At another village, two children of one family, aged three and a half and one and a half

[1] Transactions of the Epidemiological Society, vol. ii. p. 369.

[1] Ibid. vol. ii. p. 372.

[2] Professor Stokes, The Medical Press and Circular, June 19, 1867, p. 581.

years respectively, died of the epidemic, one on the 27th January, the other on the 7th February. The clothes of the deceased were taken to a neighboring village, and came into the possession of a girl aged five years. She soon sickened of the epidemic, and died on the 14th February.[1] (c) Boudin relates instances of the appearance of the disease in garrisons, and among the civil population of towns, after the introduction of detachments of troops among whom the disease had prevailed or was prevailing at the time.

[The occurrence of this affection in garrisons has been too frequent to be otherwise than important. During the civil war in the United States, many cases occurred amongst soldiers in camp, and at their homes in Northern cities upon their return from service. Two cases at least occurred in Philadelphia, the origination of which coincided with exposure to the effluvia from filthy clothing of returned private soldiers. But this is, most probably, evidence of the existence of a peculiar morbid poison generated by slow organic decay; not at all necessarily giving proof of somatic contagion. The idea of the personal communicability of cerebro-spinal fever has met with no support in the general experience of American practitioners.—H.]

The foregoing facts simply suggest the possibility of the active cause of epidemic cerebro-spinal meningitis being communicable by the sick to the well. This possibility, notwithstanding the apparent formidable array of facts to the contrary, is not to be lightly dealt with. The lesson taught by the difficulties and doubts which beset the discovery of the communicability of typhoid fever and cholera, will have been strangely misunderstood if it is necessary to urge upon observers, the importance of keeping the question of the possible communicability of epidemic cerebro-spinal meningitis constantly before the mind. In the consideration of this question, however, a caution is needed. The term "contagion" is used too indiscriminately. It has been so long employed to express the manner of transmission of disease which is witnessed in smallpox, scarlet fever, or typhus, that it is difficult to dissociate the idea of this manner from the word. It is almost impossible, in reading the opinions of those writers who have come to the conclusion that epidemic cerebro-spinal meningitis is not a "contagious" disease, to avoid the suspicion, from their use of the adjective, that they have looked upon the question too exclusively from the point of view suggested by the diseases named. It is obvious that contagiousness of a like character to that of smallpox, scarlet fever, or typhus, is not possessed by the malady under consideration. The question is: Does epidemic meningitis, like typhoid fever or cholera, possess a peculiar contagiousness of its own, a property of communicability peculiar to itself? This has yet to be solved. Another explanation of the facts which appear to indicate a possible communicability of the disease from the sick to the well is, however, open, and is set forth in the next paragraph.

Diseased grain.—Dr. B. W. Richardson has suggested that epidemic cerebro-spinal meningitis may possibly arise from the consumption of diseased grain after the manner of ergotism, and perhaps acrodynia. He thinks that the probabilities are altogether in favor of the suggestion, that "the cause in fact is a diseased grain, or fungus, contained in some kinds of flour out of which the breadstuffs are made. This fungus may not be present in large quantities, and many persons may eat of the food without getting a poisonous part; but one will get it out of a number, and this without any communication beyond the breaking of bread together: the disease may occur in one member of a family, leaving the rest free, and in this irregular way it may be distributed, in an epidemic form, over a large surface of country." He adds, "If my hypothesis, as regards cause, be correct, there is little danger of the disorder extending widely in this country; for of our cereals used as food, nearly the whole of the population now select wheat, and our wheat generally is selected for the market with great judgment and circumspection. Any cases, therefore, that might occur would be isolated, and would be easily traced out and prevented."[1] This suggestion opens out an altogether new field of inquiry respecting the origin of the disease, and it demands active and thoughtful consideration in subsequent outbreaks. Dr. H. Day, of Stafford, has endeavored, by experiments on the lower animals, to obtain some light on the subject. He fed three rabbits with unsound grain (wheat, oats, ergot of rye, and mouldy bread) with this result: In all the animals a spasmodic affection was produced, and in two inflammatory changes in the right eye, proceeding in one case to ulceration of the cornea, and evacuation of the contents of the globe. One of the rabbits died on the eighth day, the other two were killed on the twelfth day, and in all more or less congestion of the membranes of the spinal cord was found on dissection.[2]

The sum of our knowledge of the etiology of epidemic cerebro-spinal meningitis is

[1] Hirsch, Transactions of Epidemiological Society, vol. ii. p. 373.

[1] Social Science Review, May, 1865, p. 403.

[2] Clinical Histories and Comments, pp. 18—23.

this—that the clue to its explanation has not as yet been discovered.

NATURE.—1. *Is the disease malarious, as suggested by some writers?* The outbreaks in which the disease has occurred in malarious districts, or in which the malady has shown an intermittent character, are too few in number to admit of much, if any, doubt resting upon the answer. There is no sufficient ground for believing that the malady is of malarious origin. The numerous examples of prevalence of the disease in localities free from malaria set the question aside definitely. Even when intermissions or remissions have been observed in the progress of the malady, it must not be hastily assumed that they are consequent upon malarious poisoning. Hirsch has shown that certain cases of epidemic cerebro-spinal meningitis, distinguished by intermissions and remissions, which came under his own observation, took place in the course of an outbreak in a district free from malaria. Further, he states that this outbreak prevailed at a season (winter) and in a state of climate (intense cold) which notoriously exclude the prevalence of malarious disease, even where endemic; that the period of life (1–5 years) least liable to malarious disease furnished the largest contingent of victims, while the classes most advanced in life, and who are most liable, escaped the epidemic in a remarkable degree. Finally, the infallible test of malarious disease, quinine, by its inutility in cases of the epidemic which assumed an intermittent or remittent character, showed the non-malarious nature of the affection.[1] 2. *Is epidemic cerebro-spinal meningitis a form of, or allied to, typhus?* Epidemic cerebro-spinal meningitis differs from typhus in the aspect of the patient, progress of the disease, range and course of temperature, form of cerebral affection, character of eruption, sequelæ, rate of mortality, anatomical lesions, and manner of dissemination. Differing in all essential particulars, doubt can only arise when the two diseases prevail together. Under such circumstances, cases of the fulminant and purpuric forms of the one malady may be difficult to discriminate from the graver and more rapidly fatal forms of the other. Doubt also may arise when in the course of the former disease typhous or typhoid symptoms occur. But such a doubt applies equally to the discrimination of the disease from measles and typhoid fever, as from typhus. 3. *Is epidemic cerebro-spinal meningitis a true or a pseudo-epidemic disease?* Is this disease a true epidemic disease in the sense of its being due to a specific febrile poison (to which class of diseases the term epidemic is now well-nigh alone restricted)? Or is it a pseudo-epidemic malady, as being an exaggerated and more prevalent form, from certain climatic or other conditions, of an idiopathic inflammatory affection of the brain and spinal cord? No absolute distinction can be drawn between sporadic cerebro-spinal meningitis and the epidemic malady of the same name. But there are certain broad and well-defined differences. The conjoined inflammatory affection of the covering membranes of the brain and spinal cord, which is the rule in epidemic cerebro-spinal meningitis, is a rare exception in sporadic inflammation of the envelopes of the central nervous centres. Again, the indications of blood-change which are so common in the epidemic disease have been witnessed only in exceptional cases in the sporadic disease. The question arises whether the exceptional cases are instances of idiopathic cerebro-spinal meningitis or of the epidemic form of the malady. This cannot be determined arbitrarily. It is certain that cases of cerebro-spinal meningitis, indistinguishable from the epidemic disease, are observed, in the intervals of prevalence of the latter, even in this country (H. Day, Wilks, &c.). These cases are of much, although as yet of indeterminate, interest, in reference to the etiology of the malady. It has been suggested that the blood-change and herpetic and purpuric eruptions may be of nervous origin, and consequent upon the profound alteration in the nervous system.[1] It has been suggested, also, that the purpuric eruption of epidemic cerebro-spinal meningitis may be one of several signs of a general tendency to purpura in disease at the time of prevalence, and merely an incidental phenomenon of the epidemic malady. Thus in Dublin (1866–67), purpura has been observed in rheumatic fever, and there was an outbreak of purpura among swine,[2] contemporaneously with the epidemic. The first suggestion touches a very curious question, which as yet does not admit of solution. But it is worthy of remark that the form of eruption which of all others is peculiar to epidemic cerebro-spinal meningitis is the *herpetic*—a form which, in some of its manifestations at least, as in herpes labialis, and in shingles, has singular neurotic relations. Mr. Jonathan Hutchinson has propounded the riddle, *Is herpes zoster an exanthem or neurosis?*[3] This is certain, that it is a symptom which has some definite connection with lesions of nerve

[1] Transactions of the Epidemiological Society, vol. ii. p. 377.

[1] Dr. Banks, The Medical Press and Circular, June 19, 1867, p. 580.

[2] Dr. Mapother, The Lancet, July 13, 1867, p. 39.

[3] London Hosp. Reps., vol. iii. p. 70.

trunks, if not of nerve *centres*.[1] The facts upon which the second suggestion is based are of interest, but they form too narrow a basis for conclusions. 4. *Is epidemic cerebro-spinal meningitis a disease, sui generis?* The association of symptoms shows that it is an independent malady; the aptitude to blood-changes in the course of the disease, judged by analogy with like changes which occur in acute specific diseases, suggests the inference that it also is dependent upon a specific poison, from whatever source derived. This is the conclusion which appears to have the highest degree of probability in the present state of our knowledge.

An intercurrent question arises here—Is the fulminant form of epidemic cerebro-spinal meningitis really a variety of the disease, or a different malady altogether? Dr. R. D. Lyons maintains that during the prevalence of the epidemic in Dublin in 1866, two independent diseases existed. The one, characterized by collapse, profuse purpuric eruption, great rapidity of course, excessive fatality, and absence of anatomical lesion in the nervous centres after death, he designates *febris nigra;* the other was the disease commonly known as cerebro-spinal meningitis. But it is to be remarked that the two varieties of disease have never been observed except in the same epidemic; that they pass by insensible grades the one into the other; that the most highly developed symptoms of the so-called *febris nigra* sometimes occur together with the most marked symptoms of cerebro-spinal meningitis; and that it is more consistent with experience to consider the two series of symptoms as indications of one and the same malady, rather than two maladies going forward at the same time in the same patient. A second intercurrent question is, whether the purpuric form of the disease be of scorbutic origin? The question amounts to little more than a suggestion. There are no facts which support an affirmative answer; for, apart from other well-known signs, purpuric spots are not indications of a scorbutic taint.

TREATMENT. — *Prophylactic.* — Ignorance of the true etiology of the disease limits our preventive efforts to general sanitary measures, applicable to all epidemic diseases, for the purification of houses and localities. Mr. J. Simon, recording the conditions under which the disease has prevailed, writes: "I am strongly of opinion that the best sanitary precaution which in the present state of knowledge can be taken against the disease, must consist in care for the ventilation of dwellings." He adds, however, "that in some cases, according to local reports, the distribution of an epidemic has very decidedly not been governed by conditions of overcrowding and ill-ventilation." Dr. B. W. Richardson's suggestion as to the cause of the disease should lead to the careful microscopic examination of all breadstuffs and farinaceous preparations in use among families and communities where the disease breaks out, and the disuse of such as maybe of doubtful character.

Curative.—The treatment of epidemic cerebro-spinal meningitis is as unsatisfactory as that of cholera. The evidence of the course of the disease having been beneficially affected in any outbreak by the administration of medicine is very doubtful. The too common rapid progress of the malady to death, as in cholera, and the nature of the lesions determining death, necessarily set at naught efforts to check it; medicine not being guilty either of inaptitude or inactivity. The control of this disease, as of cholera or trichiniasis, is a question of preventive rather than curative treatment, and must depend upon the discovery and limitation of its cause. In the earlier outbreaks, epidemic cerebro-spinal meningitis was treated, as an acute inflammatory affection, by bleeding and purgatives, with the general result that the fatality of the malady was probably almost invariably augmented. During the outbreak of 1866 in Philadelphia, it was found that, in the more sthenic cases, cupping the nape of the

[1] Dr. H. Day (Lancet, vol. i. 1867, p. 731) expresses the opinion that an eruption would probably be more commonly found in cases of sporadic cerebro-spinal meningitis if it were more carefully looked for; and he remarks that the petechial spots which he has observed in cases coming under his own notice, sometimes do not appear until after death. My own observations coincide with those of Dr. Day. In connection with this subject it is well to bear in mind Trousseau's so-called *cerebral* or *meningeal macula* (Lectures on Clinical Medicine, Bazire's Trans., vol. i. p. 459), a phenomenon of wider occurrence in diseases of the central nervous system than even the distinguished professor suspected. I may add, that a short time ago I was present at the examination of the body of a patient who had died from a syphilitic disease of the right hemisphere of the brain. I was particularly struck with a peculiarity in the after-death lividity which seemed to me not uninstructive with regard to the purpuric forms of cerebro-spinal fever. Life had been extinct eighteen hours. The greater part of the depending portions of the body was ecchymotic; but, in addition, many livid, circular spots, of about the size of a split pea, a few of larger size, were scattered over the upper aspect (dorsal) of the feet, and of the legs beneath the knees; also, over the upper aspect (dorsal) of the hands, and of the arms beneath the elbow. These spots, had they been observed in a case of cerebro-spinal meningitis, would have been designated "purpuric."

neck was "of essential service in mitigating, and generally, indeed, in wholly removing the neuralgic pains which form so prominent and so severe a symptom in many cases of the disease" (Stillé). When the state of the patient forbade the abstraction of blood, dry cupping used in the same locality afforded signal relief, and rendered the effects of vesication more prompt and complete. This was the experience in one of the least fatal outbreaks recorded. The experience of the majority of epidemics has been against any blood-letting, local or general. The deduction to be derived as to depletion from the general state of the circulation, entirely coincides with the results of practice. For, as a rule, the pulse from the very outset contraindicates the withdrawal of blood; and, if in any case it should seem from the general symptoms that depletion might exercise some control over the central mischief, a thoughtful regard should be given to the future. The application of *cold* to the head and spine, either by means of ice or a freezing mixture, in Esmarch's india-rubber bags, is not open to the same objection as blood-letting, and has furnished by far the most satisfactory results of all direct treatment of the acute cerebro-spinal symptoms. In its use care should be taken not to prolong the application so as to depress, or increase the depression already existing of, the whole system. When the acute nervous symptoms are accompanied by marked prostration, it is advisable during the application of the ice to swathe the limbs in hot flannels, to pack the legs and thighs with hot-water bottles, or bags filled with hot sand or salt, and to cover the abdomen with thick layers of flannel or cotton-wool. From the very outset of the disease, care should be taken to economize the temperature of the body, and anticipate its fall; and in cases characterized by collapse, or much vital depression, the application of external heat in the manner just suggested is a cardinal point of treatment. Of medicaments directly addressed to the nervous symptoms, *opium* is the most valuable. It is especially indicated when there is much restlessness, acute delirium, sleeplessness, hyperæsthesia, or painful spasm. *Morphia* is the best form of administration, and subcutaneous injection perhaps the best mode. The drug should be given in decided and frequently-repeated doses, and carefully watched. Stillé says of its use during the late outbreak in Philadelphia: "We were in the habit of giving one grain of opium every hour, in very severe, and every two hours in moderately severe cases, and in no instance was produced either narcotism or even an approach to that condition. Under the influence of the medicine the pain and spasm subsided, the skin grew warmer, and the pulse fuller, and the entire condition of the patient more hopeful. It seemed probable, however, that the full benefit of the opium treatment could be received by those only who were subjected to it in the early stages of the attack. Direct experience is here in perfect accord with the expectation which a knowledge of the pathological processes involved in the disease would naturally suggest."

A Committee of the American Medical Association has reported favorably of the *sulphate of quinia* in large doses, given at the very beginning of the disease. In some instances the drug seemed to abort the attack. The committee speaks also of favorable results reported from the combined use of *ergot* and *chloride of iron*. [Dr. Joseph Klapp, of Philadelphia, asserts the recovery of a number of cases, some of them of a very threatening character, under the early and free use of tincture of chloride of iron; 20 to 25 drops every two or three hours.—H.] Some American physicians have given *ergot* in combination with *belladonna* and *belladonna* in combination with *quinine*, but with equivocal benefit. *Mercurials* have been freely used, particularly in the form of *calomel*, but their effect has been most questionable, except as purgatives. Their indiscriminate use is to be utterly condemned, and their use at all to be discountenanced. A host of other medicaments have been made use of, of which it is requisite to note only *iodide of potassium*, *bromide of potassium*, and *arsenite of potash*. The circumstances under which the two former drugs have been used, and are most likely to prove beneficial, will suggest themselves to the practitioner. It does not appear that any decided good has arisen from their administration. In protracted cases of convalescence the arsenite of potash may prove a valuable remedy.

Of the general treatment of the patient the hot bath (102°-106°) is, when practicable, the most important feature. The Committee of the American Medical Association recommend, when the surface is cold, friction with hot, coarse towels, or even with warm oil of turpentine, after the bath. The *regimen* should be generous and nutritious from the beginning of the disease. In the acute stages soup of some kind or other, or milk, is needed; and as soon as appetite returns, *solid* viands of any digestible character must be given. In the graver cases, when there is much restlessness and spasm or stupor, and food cannot be given by the mouth from the patient's refusal or inability to swallow, an attempt should be made to administer it by the rectum: when there is much thirst, the patient's fierce desire for drinks may be freely indulged. The state of the pulse is the principal guide to the use of *stimulants*.

Their administration as a special remedy independently of the indications which generally govern their use has not been followed by good results; but they are called for when the condition of the pulse and the aspect of the patient show manifest flagging of vital power. The *sequelæ* of the disease must be traced on ordinary principles.

Too frequently the state of the patient as to delirium, spasm, and irritability of the stomach limits the use of medicine to subcutaneous injections, prevents the proper administration of food, and restricts even the application of external measures. To this unhappy combination of unfortunate and uncontrollable conditions the inefficiency of treatment may partly be attributed.

Bibliography.—In addition to the references in the text may be noted: The Eighth Report of the Medical Officer of the Privy Council, containing Mr. J. Simon's Memorandum on the Disease, and Dr. J. Burdon Sanderson's Report on the Epidemics prevailing about the Lower Vistula in the beginning of 1865.—Discussion in the Medical Society of the College of Physicians of Ireland; The Medical Press and Circular for May 29th, June 5th, 12th, and 19th, 1867.—Transactions of the American Medical Association, vol. xiii. 1866, containing a Report of a Committee on the Disease.—Dr. W. H. H. Githen's Notes of 98 cases; The American Journal of the Medical Sciences, July, 1867.—Dr. S. Gordon; Dublin Quarterly Journal of Medical Science, May, 1867.—Dr. C. Murchison; The Lancet, 1865, vol. i. p. 41.—Prof. A. Hirsch, Handbuch der historisch-geographischen Pathologie, 1866, vol. i. p. 163: Die Meningitis Cerebro-spinalis Epidemica vom historisch-geographischen und pathologisch-therapeutischen Standpunkte, 1866.—Dr. Stillé, Epidemic Meningitis or Cerebro-spinal Meningitis, 8vo., 1867, Philadelphia. This work contains a very copious bibliography, particularly valuable for its references to American monographs.—G. Tourdes, Histoire de l'Epidémie de Méningite Cérébro-Spinale observée à Strasbourg en 1840 et 1841. Paris, 1842.—J. Ch. M. Boudin, Traité de Géographie et Statistique Médicales et des Maladies de Endémiques, vol. ii. p. 564; Paris, 1857.—Dr. Sandford B. Hunt, on Cerebro-spinal Meningitis (Contributions relating to the Causation and Prevention of Diseases, and to Camp Diseases. Edited by Austin Flint, M.D., for the United States Sanitary Commission. Ch. xi.).—Dr. Ed. W. Collins, Report upon Epidemic Cerebro-spinal Fever (Dublin Quarterly Journal of Medical Science, August, 1868).—Consult Hirsch's monograph and great work, to which reference has been made already.

THE PLAGUE.

By Gavin Milroy, M.D.

Definition.—The Plague may be briefly defined to be a fever, usually of an adynamic type, accompanied with bubos, carbuncles, and petechiæ.

Synonyms.—This is the λοιμός of Hippocrates and Galen; the Pestilentia of Celsus and other Roman writers; the Pestis; Typhus pestilentialis; Typhus gravissimus; Typhus bubonicus, &c., of many nosologists. It is the Febris Adeno-nervosa of Pinel, and the Peste Orientale, Typhus d'Orient, of other French authors; the Black Death, Levant Plague, Pestilential Fever, of English writers. Dr. Copland terms it the Septic or Glandular Pestilence.

Symptoms.—The bubos may be in the groins, axillæ, or the neck; occasionally, but very rarely, the popliteal glands have been affected. The carbuncles are generally on the upper or lower extremities—most frequently on the legs, but sometimes on the chest, back, or cheek. Their number may be from one or two to a dozen or more, and they vary much in size and in the tendency to become gangrenous. The petechiæ and vibices may be scattered over every part of the body. The pyrexial symptoms of the Plague differ in no respect from those in other forms of pernicious or malignant fever. There are the usual prodromal phenomena of lassitude, rigors, nausea, headache, and vertigo; oppression about the præcordia, anxiety and restlessness, with a heavy stupid expression of countenance, and a muddy or suffused state of the eyes. Then follow heat of the skin and great

thirst, frequent vomiting, a coated tongue and fetid breath, a rapid, weak, or irregular pulse, prostration with, perhaps, tendency to syncope, in some cases high excitement and delirium, and in other cases heaviness and stupor. The bowels are more frequently relaxed than constipated, and the stools are generally dark and very offensive. The matters vomited are sometimes nearly black; and the urine, which is often very scanty, and in bad cases almost suppressed, is occasionally sanguinolent. Hemorrhage from the mouth, stomach, and bowels, or from the respiratory passages, is not an unfrequent accompaniment. In some cases the intellect remains unclouded to the last, while in others the patient dies convulsed or comatose.

To describe at length the different varieties of the Plague, which have been enumerated by authors, would be very unprofitable, and only serve to obscure a subject which has often been made unnecessarily intricate by extreme verbiage in the attempt at over-subtle distinctions. The fever may vary from a simple synochus, or even an urgent synocha with violent delirium, &c., to typhus of a putrid type, with rapid sinking and speedy death. The three forms or varieties of some recent writers appear to be merely three degrees of malignancy, according to the intensity or virulence of the febrific poison, the constitution and condition of the patient, the sanitary state of the locality attacked, and the general sickliness of the season. In an epidemic outbreak the fever is usually much more malignant and deadly at first than at a later period of the invasion, its intractability and fatality very sensibly abating after a period. Sydenham tells us that, "in the infancy of the Plague (in 1665), scarce a day passed but some of those who were attacked died suddenly in the streets, without having had any previous sickness; the purple spots, which denoted immediate death, coming out all over the body; whereas after it had continued for some time, it destroyed none unless a fever and other symptoms had preceded." It seems not improbable that panic has often had a good deal to do with the very rapidly-fatal cases to which the name of 'Peste foudroyante' has been given by French writers. Clot-Bey, in his account of the Plague in Egypt, says that the worst cases usually proved fatal on the second or third day, the cases next in point of severity on the fifth or sixth day, and that in the milder cases death did not generally occur till the second or third week after the first setting in of the symptoms.[1]

[1] De la Peste observée en Egypte. Paris, 1840.

During an epidemic, many persons have often been affected with glandular pains and swellings, and occasionally also with carbuncles, but with so little febrile disturbance that they have been able to follow their occupations, and have speedily got quite well under very simple treatment. Such cases have often been the occasion of no little controversy as to whether the persons should be considered as infected with the Plague, and therefore liable to an enforced segregation under the old system of quarantine police. In connection with this point, it may be noticed that in Egypt and some other countries, where the Plague used to be a frequent visitant, glandular swellings and carbuncular disease are extremely common affections in most seasons.

Diagnosis.—If the presence of bubos, carbuncles, and petechiæ were an invariable and necessary feature of the Plague, and of no other febrile disease, there would, of course, be no greater difficulty in discriminating it than there is in discriminating smallpox or measles from other pyrexiæ. But such is far from being the case. Fevers have repeatedly been alleged in certain countries, and at certain epochs, to be cases of the Plague, although they were at the time unattended with these external phenomena; and, on the other hand, fevers accompanied with these symptoms, occurring in other countries and at other epochs, have as frequently not been designated or considered cases of the true pest. Diemerbroeck, who saw much of the Plague in Holland, during the early part of the seventeenth century, distinctly states that there is no one characteristic or pathognomonic symptom of the Plague; and the remark of Heberden, that, "on first breaking out, the disease has never been known to be the Plague," strictly accords with the observation of all the most experienced writers of the present century, as well as of former times. It is well known that in Constantinople, or in Cairo, no physician ever ventured to say what was the true nature of a prevailing fever, however fatal it might be, or would give it the name of the Plague, until a case occurred in which a distinct bubo or carbuncle was seen. This hesitation was mainly due to the universal unwillingness to admit the presence of a disease, the bare mention of whose name carried with it such dire consequences to the freedom of personal and commercial intercourse; and the result was, that the pestilence had generally existed among a community for a considerable time before any prophylactic or precautionary measures were adopted.

That glandular swellings, and occasionally also carbuncles, may be present in other forms of pernicious fever, malarial

or not, besides the Plague, has been frequently noticed by writers of different countries. For example, the endemic fevers of the Danubian Principalities, which were so terribly destructive to the Russian armies in the campaign against the Turks in 1828–29, as on all former occasions, and which were called sometimes putrid typhus, and at other times pernicious intermittent, are described as being often accompanied with bubos, carbuncles, and purple blotches on the skin. Their greatest malignancy was in the months of August and September. In the earlier part of the year dysentery, with ordinary intermittents and remittents, were very common and fatal; the latter insensibly lapsed into the pestoid fever. The worst cases were evidently undistinguishable from the Plague; but the authorities studiously avoided all mention of the word, from dread of the panic among the troops that would have inevitably ensued.

In the fever known as the "Pali Plague" in India, to which reference will be subsequently made, the symptoms were often closely akin to, if not identical with, the pestilence of the Levant, &c.; and the same has been the case in some other pernicious fevers, both in India and in other tropical countries. In the endemic typhoid fevers of Syria petechiæ and enlargement of the parotid glands have been noticed as being frequently present, so that it has been difficult at times to distinguish them from the true pestilence; and it is well known that glandular swellings, and even carbuncles, not unfrequently occur in the typhus of our own country. Some striking instances are on record of a fever directly produced by the inhalation of putrescent animal effluvia, exhibiting all the characteristic phenomena of the Plague. In the Medico-Chirurgical Review for January, 1825, is related a case of this sort, which occurred to four sailors at Whampoa, who had gone on shore to bury the body of a comrade, who had died of dysentery. On digging the grave, they accidently opened a coffin which contained a putrid corpse. Two of the men were immediately struck down with the horrible stench, and soon afterwards were attacked with fever, accompanied with petechiæ over the breast and arms; in one of the patients a bubo formed in the right groin and axilla. Both men died—one on the fourth, the other on the fifth day. On dissection, most of the inguinal and axillary glands were found enlarged and hardened; several of them, when cut into, contained matter. Another man of the party did not sicken with fever until the eighth day after exposure; but for two or three days previously one of the inguinal glands had been swollen and painful. The symptoms were serious for a few days, but eventually the patient recovered. The fourth man was but slightly indisposed.

To make use of so uncertain and variable an attribute as the contagiousness, or the degree of contagiousness, of an existing fever, as a diagnostic mark of the Plague—as some nosologists and other medical writers have done—is obviously illogical, and must inevitably serve to mislead. In the case of the malignant Danubian fevers, several of the Russian medical officers denied their pestilential character, on the sole ground that no distinct proofs of "*contagion par attouchement*" had been observed; while they admitted that all the symptomatic characters of the true Plague were present. Many similar instances might be cited where this fallacious test has been trusted to. Indeed, most of the absurd errors in the history of the disease during the present and last century may be traced to this very source.

Morbid Anatomy.—The necroscopic appearances observed by Bulard,[1] Clot-Bey, and other French and Italian physicians in Egypt, were in most respects the same as have been noticed in the bodies of patients who have died from malignant congestive fevers, continued or remittent, in other countries, tropical and temperate. The viscera and their investing membranes, whether of the head, chest, or abdomen, exhibited marks of great venous injection, and there was usually more or less serous effusion into the cerebral ventricles, and the cavities of the pleura and peritoneum. All the parenchymatous viscera were loaded with fluid dark blood, and were generally much more lax and softened in texture than in health; the spleen in an especial degree. On the peritoneal covering of these organs, and also of the stomach and intestines, patches of ecchymosis and petechial spots were frequently met with. The mucous surface of the gastro-intestinal canal frequently exhibited the same appearance; and the stomach often contained a quantity of dirty viscid fluid, like a mixture of bile and semi-putrid blood. Some writers have asserted that the mesenteric glands, and indeed the whole lymphatic glandular system, internal as well as external, are always more or less diseased, swollen, discolored, and often softened, or otherwise altered in structure. Bulard has found the entire chain of glands from the groin to the solar plexus enormously developed, forming a compact mass, to which the veins, arteries, and nerves closely adhered, and imbedded in blood effused into the

[1] De la Peste orientale d'après les Matériaux recueillés à Alexandrie, à Smyrne, &c., pendant les Années 1833 à 1838. Paris, 1839.

surrounding cellular texture. Similar appearances have been observed along the course of the auxiliary glands, when they were chiefly affected. Clot-Bey remarks, that the bubos in the Plague are always formed by swollen lymphatic glands; those in the neck and about the angles of the jaws being independent of the salivary glands, which usually remain unaffected. Even in cases which had proved fatal before the outward appearance of any bubos, some of the lymphatic glands were, he states, almost always found on dissection to be affected; the morbid change varying according to the stage of the disease, from simple enlargement and increased hardness to dark-colored softening and putrescent degeneration.

The blood, whether drawn during life or observed only after death, has very generally been found to be darker and more fluid than in health, and only imperfectly coagulating; the clot being loose and pliable, and never exhibiting a true fibrinous or buffy coat, while the serum is often excessive in quantity, and occasionally more or less deeply sanguinolent. After resting for some time, oily globules have been sometimes noticed on the surface, and the whole mass has been observed to pass rapidly into putrefaction.

Curative Treatment.—There is little on this head in medical writings at all satisfactory or encouraging in respect of the recovery of the sick, but much that is admonitory as to the baneful effects of an over-active and meddlesome medication, and of neglecting the prime essential in the treatment of all fevers, viz., the inhalation of a pure atmosphere, of equable temperature, at all times, both night and day. Without this indispensable condition, other remedies will be of comparatively little avail.[1] The perusal of the recorded histories of cases of Plague, as observed at Malta in 1813, and in Egypt in 1835, leaves the impression on the mind that the patients would have fared better had they been treated with light nourishing food and cordials frequently administered, together with simple saline or acid medicines, and without active purgation, blood-letting, and such energetic measures. The treatment which is most suitable for ordinary typhus is doubtless that which is applicable to the Plague. In anticipating or in estimating results, it is always most needful to have regard to the period of an epidemic, when the remedies have been employed; otherwise, the most misleading mistakes may be fallen into respecting the value of remedies or modes of treatment. I cannot better close these few remarks on this head than by quoting the words of a recent experienced writer,[1] in regard of the treatment of other bad forms of fever, viz., the pernicious remittent and yellow fevers of tropical climates.

"In considering this subject, it should ever be kept in mind that not only in different situations and countries, but also in different years, these diseases, whatever the form of fever, may vary more or less, and if not in type and character, at least in intensity and complications; so that the remedial means which may have been found useful in one epidemic may fail in another, each, it may be, having a constitution of its own. We are told by Sydenham how difficult he found it, on the breaking out of an epidemic, to determine on the best mode of practice to be pursued, and how he came to a decision only after *ingenti adhibita cautela, intestique animi nervis*—an example, this, well deserving to be followed."

Natural History, Causation, Prophylaxis, &c.—Prior to the end of the seventeenth century, the Plague seems to have been as truly endemic—with occasional outbursts of epidemic violence—in most of the countries of Europe, including our own, as it was in the Levant and in Egypt from remote times, and continued to be during the first forty years of the nineteenth century. In London, for example, during the first seventy years of the century, not a year passed without some deaths from the Plague being registered; and epidemics occurred in 1603, 1625, 1636, and 1665. The last, known as the Great Plague, was followed by a marked decline in the prevalence of the fever; the number of deaths from it became fewer and fewer, and after 1679 none have been recorded in the bills of mortality of the metropolis. Whether it continued to linger in other parts of England after this date, I am unable to say; for it is to be remembered that the disease had not been limited to London, but was widely spread over different parts of the kingdom, just as typhus is at the present

[1] Nowhere have the effects of crowding the sick, and the neglect of hygienic measures in fever, been so dreadful as in the pest hospitals or lazarettos, even within the last thirty years. Dr. Bulard said of the hospital at Smyrna, "Il n'est que le vestibule du sépulcre:" and it was doubly, literally true, for the cemetery was within the walls of the establishment. In the British colony of Malta matters were no better in 1813; few of the patients sent to the lazaret left it alive. Of twenty-eight inmates seen by Sir B. Faulkner on one occasion, and some of whom then seemed to have not been seriously ill, all perished within forty-eight hours; the only attendants were convicts! See his Treatise on the Plague, from facts collected during the author's residence in Malta. London, 1820.

[1] On Diseases of the Army, by Dr. J. Davy, F.R.S. 1863.

day. In Holland, too, the decline and disappearance of the pestilence seem to have taken place about the same time as in England, or somewhat later.

During the eighteenth century, although there was a marked diminution in the persistency of the disease in a sporadic form, and in the frequency of occasional wide-spread outbursts throughout Europe generally, many severe and very fatal epidemics occurred in different countries, as in Poland, including Dantzic, and other ports in the Baltic, in 1710; in Provence and other parts of Southern France, and especially Marseilles, in 1720–21; at Rochefort in 1741; at Messina and other towns in Sicily in 1743; in several districts of Portugal in 1757; in Wallachia, Podolio, &c., in 1770; and at Moscow in 1771.

In the present century the chief seats of the pestilence have been in Egypt, Syria, Asia Minor, and the coast of Barbary. For details on this point I would refer to a "Sketch of the Geography, &c., of the Plague," in the Brit. and For. Med.-Chirurg. Rev. for April, 1864.

The most recent recognized appearance of the pestilence occurred in the neighborhood of Benghazi, between Alexandria and Tripoli, on the African coast, in 1858. It has been described by Dr. Bartoletti, who was sent by the Turkish Government to investigate its history, in a memoir addressed to the Imperial Society of Medicine of Constantinople, in August of that year, and of which an abstract is given in the Quarantine Parliamentary papers afterwards referred to. A brief notice of the "Pali Plague" of India may be here introduced.

This pestoid fever was first recognized in Cutch, in the summer of 1815, after a season of great scarcity and distress. From that year to 1820 it prevailed in different places in Guzarat, spreading to Scinde in a N. W. direction, and also towards Ahmedabad and other places in the British possessions eastward. The fever was remittent in character, with a great tendency to become continued, of a very adynamic type, and extremely fatal. In most cases there were glandular swellings in the groins, axillæ, and neck. Carbuncles or petechiæ are not mentioned as being present. There was often dyspnœa with cough and bloody expectoration. Vomiting, of at first bilious matter, and subsequently of a dark coffee-colored fluid, was likewise a not unfrequent symptom. In some cases the urine was sanguinolent, and blood oozed from the gums. After the beginning of 1821, there was no recurrence of the fever known until 1836, when it was observed in the town of Pali (lat. 26° N. and long. 74° E.), then the principal depôt of traffic between the coast and the N. W. Provinces of India. It spread to numerous places in Marwar in that year, and in 1837 on to the middle of 1838. In 1849 there was a similar fever in Gurwah and Kumaon, on the southern slopes of the Himalayas, and in 1853 in Rohilcund. As to the nature and affinities of the fever, Dr. Morehead, in his valuable Clinical Researches on Disease in India, remarks:—"The description of jail or hospital fever by Pringle, in his work on the Diseases of the Army, has considerable resemblance to that of the fever at Pali. . . . The causes were supposed to be crowding, filth, and effluvia from decomposing animal and vegetable matters."—*Second Edition*, p. 158.

In every country where the Plague (and pestoid fevers in general) has prevailed, certain local conditions have been found to favor its *development and spread*. "The principal of these are, residence upon marshy alluvial soils along the Mediterranean, or near certain rivers, as the Nile, Euphrates, and Danube; the dwellings of the people being low, crowded, and badly ventilated; a warm, moist atmosphere; the action of putrescent animal and vegetable matters; insufficient and unwholesome food; and physical and moral wretchedness."[1] An elevated site, even in the immediate neighborhood of an infected city, has often remained quite exempt, although intercommunication was not interrupted—*e. g.*, the citadel of Cairo, and the village of Alem-Daghe, near to Constantinople. The higher parts of Valetta suffered very little in the Malta epidemic of 1813; the ratio of the attacked became greater and greater in descending from the higher to the lower levels of the city, towards the foul shores of the harbor; and so much less frequently were the occupants of the upper and more airy stories of the lofty houses attacked than those of the basement floors, that it was a common remark, Dr. Hennen (in his Medical Topography of the Mediterranean) says, that "the Plague was a disease which seldom went up stairs." It has been very generally in the crowded and filthy parts of a town that the earliest cases of an epidemic have occurred, and the chief sufferers have been invariably the poor and neglected. The state of most Turkish or Egyptian towns in the present age represents very nearly what London and many other European cities were in the seventeenth century.

Outbreaks of the Plague, as of typhus, have often followed in the wake of famines and other desolating calamities. This was strikingly the case with the visitation at Benghazi. For two or three years previously there had been an unusual drought,

[1] Rapport à l'Académie Royale de Médecine, sur la Peste et les Quarantaines, fait, au nom d'une Commission, par Dr. Prus. Paris, 1846.

and the cattle had perished in great numbers from an epidemic disease. In 1857 the destitution of the Bedouin tribes became extreme, and it was then that the pestilence commenced. "Why it was the Plague rather than typhus, I know not," says Dr. Bartoletti; "but the fact was so; and I may add that the great epidemic of Plague at Erzeroum, in 1841, was also preceded by a terrible famine. One of the essential conditions for the production of typhus was wanting, viz. the agglomeration of human beings in a confined space."

Epidemic Plague has generally been preceded by a sickly season,—the sickliness consisting in the great prevalence and severity of the ordinary endemic fevers, of fluxes and other forms of bowel complaints, and not unfrequently also of catarrh. Sydenham's account of the epidemic constitution, previous to the great Plague of London, may be taken as typical of what usually occurred in respect of the disease during the sixteenth and seventeenth centuries in this and in other countries of Europe. In the Spring of 1665 catarrhs and pulmonic disorders were very prevalent and fatal. About the same time a bad form of fever, attended with vomiting, diarrhœa, &c., began to be very common, and this was increased in severity as the season advanced. Towards Midsummer, cases of this fever (which Sydenham calls "pestilential") were accompanied with bubos and carbuncles on the surface; then only was it recognized and designated as the Plague. The pestilence went on increasing in deadliness until the third week in September, when nearly 8000 died in the course of the week, although two-thirds of the inhabitants had by this time fled from the city. It had then reached its acme, and forthwith began to abate. It very nearly ceased on the approach of cold weather; a few sporadic cases only occurred during the winter and following spring. The same form of fever, however, as had preceded the first recognized cases of the Plague, was again observed throughout 1666; but it was not so general as in the previous year. Sydenham expressly says that it was of the same species as the Plague, only not so violent—*revera enim cum ipsissima Peste specie convenit, nec ab eâ nisi ob gradum remissiorem discriminatur.*

Morton, a contemporary of Sydenham, has remarked of the Plague, as he saw it in London, that "it often appeared under the form of a continued or remittent fever; but this changed into the other, and *vice versâ;* and that each in its turn became epidemic, the one yielding to the other." A similar remark has been made by many other writers in former, as well as in more recent, times. The epidemics in Egypt in 1835 and 1841 were ushered in by the unusual prevalence of the ordinary endemic fevers, in some places of a continued, and in other places of a periodic, type.

The influence of *season* on the prevalence of epidemic Plague will be seen from the following facts. In England the pestilence was most severe in the epidemic years 1603, 1625, 1636, and 1665, from about the middle of July to the first or second week in October. The Plague at Marseilles raged most fatally in the autumn months; and the same was the case at Moscow in the epidemic of 1771, when upwards of 1200 deaths took place for several days out of a population of 150,000. In Constantinople it has generally reached its acme in September; in Smyrna about a month sooner; and the same may be said of Tunis, Algiers, and other places on the North African coast, where it has usually manifested itself in spring, and committed its greatest ravages in July and August. Malta suffered most in July. In Syria the summer months have ordinarily been the most fatal. In Egypt most epidemics have commenced toward the end of the year, and gradually advanced, reaching their acme in March or April, when the southerly winds are most prevalent. The disease generally ceased in the second or third week of June. At Cairo it never continued, it has been said, beyond St. John's Day, 24th June. It thus seems that in Egypt the chief prevalence has generally been a good deal earlier than in other countries. Volney remarked on this subject:—"The winter stops the Plague at Constantinople, because the cold is great; and the summer lights it up, because the heat is then humid: while in Egypt the winter favors it, because the climate is then warm and moist; and the summer stops it, because it is hot and dry. The heat is only injurious when associated with humidity." That the peculiar meteorology of the climate of Lower Egypt may have something to do with the point in question seems very probable.

Mention is often made, in the history of Plague epidemics, of the weather having been before, and during, their prevalence remarkably oppressive and distempered. This was notably the case, according to Maitland in his History of London, in the Great Plague, when, for months, scarcely a breath of air was to be felt, and the little that there was came from the south; it was generally hot and stagnant, and mildews were abundant. A like state of atmosphere has often been noticed in other countries. Dr. Brayer specially alludes to the circumstance in his account of the Plague in Constantinople. Dr. Hennen, in his narrative of the visitation at Malta, relates the fact, as indicative

of a distempered atmosphere, that, during the prevalence of the fever, "every whitlow festered, and every scratch became an ugly sore; a tight shoe was sufficient to produce a livid boil. The military hospitals were crowded with such cases."

The mode in which the pestilence has been wont to manifest itself in a place, and to spread among a community, may be gathered from what has been said above respecting the usual antecedent state of the public health, and the physical conditions, &c. of the localities chiefly attacked. It has been often alleged that outbreaks of the Plague have been known to occur among a population previously quite healthy, immediately or very soon after the arrival or introduction *ab extra* of a person or object already affected with the disease, or believed to be impregnated with its infective poison; and that from the first case or cases, as from a focus or centre, the subsequent extension could be distinctly traced. But whenever there has been an opportunity of ascertaining the real and complete history of the facts, the course of events has been found to be much less simple and consecutive. Unfortunately, most of the past history of Plague epidemics rests on mere rumor, or on most imperfect information. It has only been within the last thirty years that thoroughly trustworthy records on the subject have been published. The admirable Report of the French Academy has shown that the pestilence has generally appeared in single cases or small groups of cases, at or about the same time, in different spots or localities of a town, or perhaps in different districts of a region, often considerably distant from each other, and without any traceable direct communication between the persons attacked. Sometimes several towns have become the seats of the fever nearly simultaneously, the intermediate villages remaining exempt, while at other times it has advanced in a more regularly progressive manner, invading a number of places "*de proche en proche*," and in succession, over an extensive tract of country. That the Plague may be diffused by transmission from the sick to the healthy near to them—in other words, by personal *contagion*—cannot be reasonably doubted; and it is equally certain that both its development and spread have often occurred independently of such agency. Extravagant opinions on this subject were in vogue amongst most physicians until of recent years, when the accurate observation of facts and a rigorous examination of previously recorded testimony sufficed to show that the influence of contagion in the dissemination of the Plague is very much the same as in the dissemination of the bad forms of typhus, and that the laws which regulate its activity and power in the one disease are equally true in respect of the other. The limits of the present paper preclude any discussion of the various important questions of State Medicine connected with this subject, and which relate to the public measures to be adopted for the prevention or arrest of the Plague, and for the protection of countries from its introduction by shipping and other channels of intercourse. To such measures the term of *Quarantine* has been usually applied. For full information on the subject I would refer to the Report of the French Academy,[1] and to the elaborate documentary evidence, and report thereon, prepared by a committee of the National Association for the Promotion of Social Science, and printed, by order of the House of Commons, in May and August, 1860, and August, 1861.

[Early in 1879, alarm was produced in Europe by an outbreak of Plague in Astrakhan, which proved very destructive in some villages, and threatened to extend into different parts of Russia.

There is evidence that its existence in the same and neighboring regions really dated back as far as May, 1878.[2] Even in 1877, it appears from Russian official information, that Plague had been widely scattered in Russia; especially near the river Volga. At the beginning of the year 1879, its devastations became known, as they occurred in Vetlianka and a number of other fishing villages, extending on both sides of the Volga for a hundred miles. In a few places, the mortality was as great as 95 per cent. of those attacked. These villages are described as being in an extreme state of filth. Nothing but fire could disinfect such localities. By order of the Imperial government, a number of villages were destroyed; their inhabitants being elsewhere cared for at the public expense. The immediate danger of the spread of the disease appeared then to be averted.

From reports and papers by Dr. G. Milroy, J. Netten Radcliffe, and others, it appears that this outbreak in Astrakhan was only one of several occurring in different places, especially in Mesopotamia and Persia, since 1873. In 1875 it was fatal to about 4000 people; and a yet larger number perished from it in 1876; although it ceased in the hottest weather of that year, Bagdad lost about 1700 lives by it in 1877. At Resht, in the province of Ghitan, near the Caspian Sea, it destroyed about 4000 lives.

---

[1] A summary of this Report, with Introductory Observations, Extracts from Parliamentary Correspondence, and Notes, was published by the writer of this article in 1846.

[2] The Plague as it Concerns England; From Official and other Sources. London, Hardwicke & Bogue, 1879.]

It does not seem irrelevant to the practical purposes of this work, to cite here the conclusions of two eminent medical and sanitary officers, in regard to the prevention of danger from Plague, as well as from other diseases kindred to it. Dr. E. C. Seaton,[1] in a memorandum "On the Systematic Action in Use in England to Prevent the Importation of Infectious Diseases," uses the following language:

"Of systematic action adopted in England for the prevention of the importation of infectious diseases, the system of quarantine (in the commonly received sense of that term) forms an extremely small part, if, indeed, it may not be said to be abandoned; an altogether different system, called the system of medical inspection, having for some time past been employed."

Dr. Seaton quotes, in the same memorandum, with approval, this expression of his predecessor, Mr. Simon:

"A quarantine which is ineffective is a mere irrational derangement of commerce; and a quarantine of the kind which insures success is more easily imagined than realized. Only in proportion as a community lives apart from the great highways and emporia of commerce, or is ready and able to treat its commerce as a subordinate political interest, only in such proportion can quarantine be made effectual for protecting it. In proportion as these circumstances are reversed, it becomes impossible to reduce to practice the paper plausibilities of quarantine. The conditions which have to be fulfilled are conditions of national seclusion."

Accepting these views as correct, it needs only to be added, that "what is true of typhus," in regard to prevention by measures of local sanitation, "ought to be true of Plague." In any but tropical or subtropical countries, it must be much easier to exclude Plague than typhus. There is abundant reason to conclude that the non-appearance of Plague in England since the visitation of 1665 has been due, not in any sense to measures of quarantine, but altogether to improvements in local and general sanitation.—H.]

ERYSIPELAS.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—An acute specific disease characterized by fever of a low type and a peculiar inflammation of the skin. This inflammation exhibits a marked tendency to spread over the surface, to induce serous infiltration and suppuration of the areolar tissue in its neighborhood, to affect the lymphatic vessels and lymphatic glands, and to cause serous exudation between the cutis and the cuticle.

Synonyms.—*Scientific Names.*—ἐρυσίπελας (Greek); Febris erysipelatosa (Sydenham); F. erysipelacea (Hoffmann); Rosa (Sennert); die erysipelatöse Dermatitis (German); Erysipèle (France); Risipola (Italian).

Popular Names.—Ignis sacer (Latin); die Rose, der Rothlauf (German); the Rose (Scotland); Saint Anthony's fire (England).

Natural History.—Causes.—Local irritations of the skin, such as the application of blistering fluids, or of boiling water, produce dermatitis, but they do not, of necessity, cause Erysipelas. The inflammation they produce may be very severe, and may resemble that disease; but the differences between such common inflammation and the special malady we are now describing are greater in number and more important in kind than are their points of resemblance. There is, then, some "cause" of Erysipelas over and above that, whatever it may be, which produces simple inflammation of the skin. There are some facts which go far to show that this cause, or that some one or more of a number of concurrent causes, may exist in the "individual;" for it is well known that some persons are liable to suffer from repeated attacks of Erysipelas, and these either with or without the slightest provocation; whereas others may be blistered, burned, cut, torn, or otherwise injured, without exhibiting any indication of the existence of such tendency. On the other hand, it is equally clear that Erysipelas sometimes has an "epidemic" character; and it is still more common to find it haunting certain

[1 Seventh Annual Report of the Local Government Board; Supplement: Report of the Medical Officer for 1877.]

localities, and thus exhibiting the features that we term "endemic;" so that in these cases its most effective cause would seem to be outside the individual, viz., in some external circumstances. If we admit, as indeed we must do, the special liability of some individuals or families to the occurrence of this disease, then the conditions underlying such liability must be regarded as "predisposing causes" of Erysipelas, and we must seek still further for the so-called "exciting causes" of the affection; and these may commonly, but not invariably, be found in accidental or other injuries to the skin, such as exposure to cold or heat, to moisture, or physical abrasion. If we accept, on the other hand, the presence of a distinct morbific agent—either epidemic or endemic—as the efficient cause of Erysipelas, then the constitutional state of the sufferer sinks into comparative unimportance, although we may still retain some belief in its action as a predisponent.

The truth as to causation lies, most probably, not between these two ideas, but in their combination; and such conclusion is by no means at variance with the belief that sometimes the one and sometimes the other factor is the more influential. It may be that either one may sometimes be so potent as, *per se*, to produce the disease; but it is more probable that, in all cases, there is some constitutional predisposition, and also some agent operating from without.

Among constitutional predisponents the most important is that of which we know nothing more than the fact of its existence, viz., an individual or family proclivity to the disease. The next in value is age. Erysipelas is common in newly-born infants, but rarely occurs between the first year of life and the twentieth; after this period it is frequent, as an acute affection, till the fortieth year; whereas in more advanced age it is seldom seen except as a chronic, or subacute, and less important malady. It has been stated that women are much more liable than men, and especially so during menstruation, and at the climacteric period; but such statement is not supported by reliable facts, although a woman, subject to Erysipelas, may exhibit the disease with especial frequency during the catamenial flow. Little that is of any value can be said of "temperament" as a predisponent; but it appears probable that the "gouty diathesis" increases the frequency of its occurrence.

In addition to the "exciting" causes already mentioned—viz., all undue impressions upon the skin—we must enumerate errors in diet, and especially the taking of certain things, such as shell-fish, or improperly smoked, dried, salted, or otherwise "half-preserved" meats. But by far the most important cause, acting from without, is the "poison," whatever may be its nature, which exists in one case and can be communicated to another, either by inoculation, simple contact, transmission through the air, or by fomites.

It is not intended in this work to deal with Erysipelas as it is commonly seen in the surgical wards of hospitals, but with that form of it which, arising often without any distinctly defined external cause, and certainly in the absence of any proved contagion, has a history differing widely from that of the disease as it is seen to follow surgical operations, under circumstances of apparently endemic aggravation. It is not asserted that the disease is essentially different in the two classes of cases; but it is held that their clinical history is so different that it is desirable to treat them separately, and to confine the description given in this "System of Medicine" to the latter, which usually falls under the care of the physician.

Symptoms.—These are both general and local: on the one hand there is fever, on the other definite structural change in the skin, mucous membrane, and, it may be, in the subcutaneous and submucous tissues. When fever precedes the appearance of redness on the skin, the disease resembles one of the exanthemata: when local inflammation exists before marked pyrexial change, the case, in its general career, is more like that of pneumonia, pleuritis, or inflammation of any other organ. The former course is the more common of the two.

The *commencement* of Erysipelas is usually marked by uneasiness of not very definite character; rigors, slight shivering, or only a feeling of chilliness may mark the onset of the malady; but more commonly rigors do not exist at the beginning, they occur after several hours of discomfort, and either immediately before or simultaneously with the appearance of local inflammation. Usually, then, prior to the occurrence of rigors, there is malaise, aching of the limbs, loss of appetite, thirst, nausea, or vomiting, diarrhœa, soreness of the throat, increased heat of skin, and frequency of pulse, headache, giddiness, confusion of thought, feeling of depression in spirits, epistaxis—as an occasional event—and, in fact, all the signs of pyrexial disturbance, without any such special predominance of any one of them as should enable the physician to forecast the nature of the impending evil.

It is not possible to affix a definite duration to these symptoms: sometimes they commence after, sometimes simultaneously with, the local changes; but more commonly they precede the latter by a few hours, *i. e.*, from eight to ten, or by

two or even three days. It often happens that a man feels well in the morning and at mid-day, but towards evening is uneasy; passes a restless night, growing worse from hour to hour; and on the morning of the next day observes some redness of his nose or ear. Or feeling better, but not well, on the second morning, he goes through a day of increasing discomfort, which becomes very considerable towards evening; passes a second night worse than the first, sometimes accompanied by delirium; and the special phenomena of Erysipelas appear on the third day. But their appearance may be delayed until the fourth or even fifth day from the onset of symptoms.

On the appearance of the cutaneous inflammation there is no remission of the pyrexial symptoms; on the contrary, they are sometimes aggravated in intensity. The usual site for their development is some part of the head; but they may appear in any other portion of the body. Local disease or injury of the skin, or even of the subcutaneous tissues, may determine the place of commencement. Abrasions, scratches, or wounds, wherever they are situated, may be the starting places of specific inflammation of the skin; whether this be of the kind we call Erysipelas, measles, or scarlet fever. But this determining power is not limited to such injuries or affections as "break the skin;" for the locality of appearance, and even recurrence, of Erysipelas may be determined by the presence of gout in a particular joint of either the upper or lower extremity, and also by the irritation of diseased teeth in either the upper or lower jaw.

Usually, however, the nose or the ear is the point at which the inflammation may first be seen. The change is, so far as my own observations extend, commonly seen to commence in close proximity to one of the passages through the skin, *i. e.*, where the skin undergoes that transition which consists in its becoming what we term mucous membrane. Thus, not only at the nose and at the ear does Erysipelas begin, but it commences just where the skin of the nose turns upwards into the nostril, or just at the point where the skin of the ear loses the dryness and other characteristics of ordinary skin. Again, Erysipelas often is noticed first at the angles of the mouth, or at the edges of the eyelids; it is met with at the anus, about the genital organs, and in the neighborhood of the recently-divided umbilical cord.

To the patient the part affected feels hot and irritable; and, upon touching it, sore, stinging, and smarting. It is of red color and shining aspect; it is warmer and harder than the surrounding tissues, swollen, and, as the disease advances, very tender to the touch. The inflammation extends from the spot first affected, sometimes in all directions, but more commonly in one much more rapidly and more widely than in another. For instance, Erysipelas starting from the ear will sometimes extend downwards and not upwards, backwards and not forwards, and so on; whereas in other but, I believe, rarer cases, the progression appears to radiate about equally in all directions. At the advancing edge of Erysipelas the elevation of skin (swelling) may often be not only felt but seen, and that most distinctly; whereas at the receding margin there is so gradual a decline of swelling that it would be difficult to say with certainty that it existed. Where the inflammation is advancing the line is marked not only by elevation of the surface, but by sharply-defined difference of color; the white or pale healthy skin is invaded by a distinct line of red, with an occasional streak, branched or not, in advance of the general boundary. Where the skin-affection is receding there is no such abrupt transition, but the heightened tint of the most active inflammation is gradually shaded down through medium and mixed tints until, without any clear line of demarcation, the skin is found in its ordinary healthy state.

Sometimes the amount of swelling is not considerable; at other times it is enormous; and the disfigurement is such that none would recognize the features of the sufferer, nor for a moment think that they were features at all, or even parts of any human being. The amount of swelling is greatest where the skin is the most loosely attached to the subjacent structures, and where there is much areolar tissue which can be distended with fluid. Thus we find the eyelids and the neighborhood of the mouth the most disfigured in appearance, and all trace of the former may be completely obliterated by the effusion. The swelling, when confined to the skin, is moderate in amount, uniform in elevation, hard to the touch, pitting only slightly on pressure, and shading off on the side of recession, but terminating more or less abruptly on that of advance. When the areolar tissue is much infiltrated, the swelling is carried to a higher degree, its surface is irregular in elevation and consistence, and there is often deep pitting upon pressure. Under the latter circumstances there is generally suppuration, probably determined by the sloughing of small or larger portions of the subcutaneous areolar tissue.

The surface of the inflamed skin remains, in mild cases, intact throughout, exhibiting, besides redness, only slight increase of desquamation as the malady dies away. In more severe and more common cases there is some vesication of

the surface; little bladders are seen like those produced by a blistering fluid, or a scald. Whereas, in very severe cases, large bullæ, of irregular shape, make their appearance; they soon burst, and leave dry and thick crusts, which render still more hideous the face that they have covered. It often happens that the inflammation is extending in one direction and receding in another, so that Erysipelas in all its stages may be witnessed at the same time in the same individual; but the maximum of redness and of swelling is usually reached on the second or third day.

In almost all cases there is distinct inflammation of the neighboring lymphatic vessels, with pain, swelling, and tenderness of the lymphatic glands: but suppuration of the latter is not met with in ordinary cases, although severe. Inflammation of the lymphatics is most commonly observed when Erysipelas has been set up by a poisoned wound.[1]

The pulse is generally full and with a frequency varying from 100 to 120 in the minute; the heat of skin is well marked, perhaps over the inflamed surface unduly marked in comparison with the elevation of the temperature generally. This may be owing to the local arrest or diminution of transudation and evaporation.

In a severe case, recently under my care, the temperature in the morning of the eighth day of illness, but fifth day of eruption, was 104° Fahr.; on the next day 102⅗°; on the following 103½°; then came twenty-four hours in which it ranged between 99⅗° and 100⅕°; to be followed by another rise,—coincident with some extension of the inflammation,—to 102⅗°; after which it became normal. The evening temperature in this case was daily lower, and sometimes considerably lower (2° to 4° and even 5°) than that of the morning. A similar relation between the morning and evening temperature was observed in another almost equally severe case occurring at the same time. The prevailing relation between morning and evening temperatures is, however, similar to that observed in other acute specific diseases, viz., an excess of elevation in the evening; but the variation from this general type is, I believe, more common than is supposed not only in Erysipelas but in other allied maladies. It is quite clear that relapses are attended by renewed rise of the thermometer; and such relapses may occur in the morning as well as in the evening, and so pervert the characteristic febrile course. Such relapses may possibly be overlooked, as in some cases the symptoms are almost imperceptible to the patient, and may occur in some locality which is not necessarily exposed to the eye of the physician; and yet, although so slight as not to attract attention in any other way, they may do so by their effect on the thermometer. Lately, for example, in a patient apparently convalescent,—feeling tolerably well, and taking food with relish, the temperature having been normal for two days,—I have seen a sudden rise in the thermometer, unattended by any return of pain or malaise, but, upon examination, shown to be coincident with renewal of the inflammation below the shoulder blades; it having commenced at the ears, extended to the back of the neck, and for some days stopped at the level of the scapular spines.

The fever, as measured by the thermometer, is very variable in *duration;* and the temperature, after having returned to the normal amount, may exhibit several re-elevations coincident with extensions of the inflammation. Usually the maximum is reached on the third day of eruption, and the decline commences on the fifth or sixth.

The pulse,—usually exhibiting an increased frequency bearing direct relation to the abnormal elevation of temperature,—may revert to the habitual standard at the end of the third or fourth day, and not again rise far above this, although one or more relapses may occur, each of which is marked by a rise in the thermometer.

Albumen appears in the urine in many cases. It may make its appearance from the fourth to the eighth day, or even later, in relapse. Unless there be pre-existent disease of the kidneys, it is small in amount and of short duration; it has been noticed to be absent on the fifth day, present on the sixth, and again absent on the seventh. The quantity of urea is increased, while that of the chlorides is diminished.

The *course* of symptoms varies widely. In one class of cases there is a speedy diminution of their severity both locally and generally; whereas in others the reverse is observed. Sundry local changes, proportionate to the amount of swelling, may occur, and become excessively annoying. Such, for example, are blindness, deafness, and impossibility of breathing through the nose. But, beyond these and the relapses which have been already mentioned, there are others depending upon the sloughing and suppuration of areolar tissue. When such changes take place, the symptoms become much more distinctly adynamic; and in bad cases the tongue is brown, the lips and gums are covered with sordes, the pulse rises in frequency and loses in force so that it is often quite uncountable; there is low muttering delirium, with jerking contractions of the limbs, and, indeed, all the other signs of impending dissolution.

[1] Niemeyer, Lehrbuch der speciellen Pathologie und Therapie. Bd. ii. p. 396.

DIAGNOSIS.—It is not very easy to confound Erysipelas with any other malady when once its ordinary symptoms are developed; but it may sometimes be inferred to be present before the skin has shown signs of inflammation. Where there is marked pyrexia, with vomiting at its commencement, without notable pain in the back, or obvious change in the mucous membrane of the throat or nose, but with enlargement, pain, and tenderness of the lymphatic glands in the neck, Erysipelas may be, and ought to be, suspected. Attention to the description of symptoms already given will be sufficient to prevent any errors of diagnosis. The spreading character of the inflammation, as this is seen in the skin and also in the subcutaneous cellular tissue, distinguishes Erysipelas from true phlegmon, where lymph speedily circumscribes the swelling. Although the symptoms of Erysipelas occasionally exhibit intermissions, and by no means rarely some remissions, it would be difficult now for the mistake between it and intermittent fever to recur.

From what has been termed "diffuse cellular inflammation," Erysipelas may be distinguished by a constant presence of inflammation of the skin, which latter bears a direct ratio to the affection of the areolar tissue, and precedes it, as a rule, in regard to time.

Erythema differs from Erysipelas in the comparative mildness of its general symptoms, in the absence of swelling of the skin, and in the tendency which it exhibits to form patches, of various sizes and shapes, which show no marked tendency to spread.

PATHOLOGY.—There can be no doubt that some cases of Erysipelas resemble those of simple erythema, or simple inflammatory redness of the skin, such as may be produced by a mustard poultice. Nor can there be any doubt that, on the other hand, there are cases of Erysipelas which resemble more closely diffuse cellulitis and pyæmia. Between these extremes there are patients whose symptoms are those of phlebitis or of inflamed absorbents; and others whose cases can hardly be distinguished from genuine phlegmon. In all these we may have no doubt of the existence of Erysipelas; but the idea we entertain of the "pathology" of this disease will be determined by the frequency with which we find it assuming this or that typical form, the amount of importance we attach to these several associated conditions, and the clearness of our knowledge about them. Thus, Erysipelas may be regarded as a disease having its first local manifestation in the absorbent system, or it may be held to be essentially a blood disease, always called into play by some external injury, however slight that injury may be; it may be thought to be so distinctly hæmic, that it should find its place among the symptoms of pyæmia; while, on the other hand, the view may be entertained that a true inflammation of the cutis is its one essential condition—that Erysipelas is a *morbus per se*, having powers of easy association with each of those maladies alluded to, but being, at the same time, essentially distinct from all of them. Those who entertain the last opinion, again differ among themselves, some regarding the inflammation as identical in its essence with that which might be produced by an irritant; others, as being of special type, the result of one specific poison, the presence of which in the system is the *sine quâ non* of the existence of that form of inflammation which we term Erysipelas. And here, in this last resort, diversity of opinion may still be found; for some hold that the poison is developed from within, that it arises from "crudities," from "digestive derangements," and the like; while others believe that it is always imported into the body from without; and it would seem that yet a third view might be taken, viz., that it is by the conjunction of these two elements that the disease is established; that is to say, neither internal conditions (predisponents), existing alone, nor external poisons (septics, or endemic influences), acting by themselves, can produce Erysipelas; but that the outside poison, however active, or however greedily swallowed by the organism it may be, is inoperative unless it finds in that organism the proper, *i. e.*, "special," nidus, conditions, or material for its development.

It would be possible so to select and arrange the facts about Erysipelas as to make them support any one of these theories or pathological positions; but, taking them without selection, and only arranging them so far as to render the teaching intelligible, the conclusion most consistent with them appears to me to be that which is expressed the last in the foregoing paragraphs, and in the definition of the disease: viz., that Erysipelas is an inflammation of the skin, that it may involve the absorbent or the venous systems, that it may change the character of the blood; but that it may act independently of any such complication; further, that it is an inflammation of "special" character; and, lastly, that it depends upon the action of a particular poison upon a peculiarly predisposed constitution.

MORBID ANATOMY.—The slighter cases of this disease rarely furnish opportunities for the study of their anatomical conditions, but analogous states of the skin and neighboring tissues may be observed in the outskirts of the severest inflamma-

tion in cases of fatal Erysipelas. In them, after death, as well as during life, the disease may sometimes, but of course at different points, be observed in all its stages, and in all its degrees of intensity.

The skin is thickened by increase of vascularity and of serous infiltration, while there is a marked absence of lymph so thrown out as to circumscribe the swelling. In mild cases, no suppuration is observed, but, in the less mild, there are detached patches of suppuration, sometimes affecting the cutis only, but more commonly extending into the areolar tissue; whereas, in severer cases the skin may be completely separated from the subjacent tissues, and this for a very considerable extent, by large quantities of pus, in which shreds of sloughing or dead areolar tissue may be found, semi-detached or floating. The disease, although commonly limited to the skin and its immediately connected tissues, sometimes extends to the deeper and more important structures, and then muscles and ligaments and bones become involved in the general mischief.

Mr. C. de Morgan[1] states that a very important fact had been "mentioned" to him by Busk, viz., "that in all the fatal cases which he examined, the lungs were highly congested, and that, on close inspection, the smaller pulmonary vessels were always found to contain pus; that, in fact, a minor degree of pyæmia was always present. He (Mr. Busk) has observed the same thing in the small vessels of the head, when that part has been the seat of Erysipelas." Dr. Bastian has found minute embolic masses in the small arteries and capillaries of the gray matter of the cerebral convolutions. Some of these masses, which are small, appear to be made up of white blood-corpuscles; but the larger, irregular, and rounded bodies are, in Dr. Bastian's opinion, amorphous masses of albuminoid material, separated from the blood-plasma.[2] Dr. Copland[3] says, "The veins proceeding from the part chiefly affected are often inflamed, or contain pus, as first observed by M. Ribes and confirmed by Messrs. Dance, Arnott, and by my own observations." It is well known that in many cases of fatal Erysipelas evidences of disease may be found in the spleen, liver, kidneys, lungs, bronchi, larynx, trachea, and fauces; but there is nothing specific in the character of the changes discovered in these organs—nothing, that is, which is peculiar to the disease called Erysipelas—nothing, indeed, which depends upon the Erysipelas *per se;* but all that may be found is only the sign of such general blood-change as may be associated not only with the disease now under consideration, but also with that large group of maladies which stand in close relation with pyæmia. (See p. 330.)

Occasionally, gangrene is found in some portions of the inflamed skin or cellular tissues; and this particular termination of Erysipelas has appeared to me the most common in those cases which are associated with dropsical effusions. Sometimes the Erysipelas has appeared around natural or artificial openings made for the escape of serum, and then either portions of the integuments of the lower extremities, or, more commonly, of the genital organs, become greatly inflamed and gangrenous. The association of gangrene with dropsy may be due, in some measure, to the altered blood-condition—which almost always exists in extensive anasarca, whatever may be the seat of that mechanical obstruction to which it is referred, and which determines the locality of its appearance,—an altered blood-state, moreover, which in some dropsies appears to constitute the whole of the essential condition for their development. But, in addition to the blood-change, there is, in the fact of the existence of dropsical effusion in the cellular tissue, a change in the nutrition of the vessels, and an altered relation of the fluids and solids of the parts involved: and there is, further, a simple mechanical interference with the circulation in the skin, exerted by the pressure of the effused fluid not only on the capillaries, but on the vessels which lead to and from them. Thus, dropsical limbs are, unless reddened by Erysipelas or other conditions not essential to the dropsy, ill-nourished, pale, and cold; and a slight addition of difficulty to the already embarrassed circulation of the part, often ends in gangrene.

Prognosis.—A case of Erysipelas is bad in proportion to the predominance of the symptoms of blood-poisoning over those of simple inflammation. It is bad just in the degree to which it resembles typhoid fever or pyæmia, rather than simple dermatitis. Mere extent of inflammation is not of itself of evil augury; a high degree of inflammation is of no greater value; but a very rapid and weak pulse, with a dry brown tongue, and low muttering delirium, with marked prostration of the strength, is of almost fatal omen, although the local changes may be closely limited in both distribution and severity.

Cases which arise from the introduction of poisonous matter are worse than those in which the malady appears spontaneously; and this, whether the poison has been introduced by a wound made with an infected implement, such as a dissection wound, the prick by a bone from dis-

[1] Holmes's Surgery, vol. i. p. 237.

[2] British Medical Journal, January 23, 1869.

[3] Dictionary, art. "Erysipelas."

eased meat, &c.; or whether it has been conveyed through the air, or by other means, to a wounded surface, placed where Erysipelas is endemic.

The extremes of age, the presence of disease in either the kidneys or the liver, the dropsical constitution, a state of chronic alcoholism, or of any morbid blood-condition, are prognostic of an unfavorable termination.

The extension of Erysipelas to the throat may introduce a source of danger altogether different from that which belongs to the disease itself. Life may be threatened and indeed terminated, and that too very suddenly, by apnœa. The inflammation of the fauces may bring about serous or other infiltration of the neighboring submucous tissues, and the opening into the windpipe may be closed. In Erysipelas of the head or neck it will always, therefore be necessary to examine the throat most carefully, and to observe the manner and number of respirations, as well as the tint of the skin, in order to guard the prognosis against a false security. It is the more necessary to do this, because in some cases the patient—owing probably to the dulled state of his sensations and perceptions, brought about by the poisoned condition of his blood—may make no complaint of dyspnœa, or of discomfort in the throat; whereas an altered tone of voice or cough, an occasionally hurried respiration, a slight lividity of the lips or finger nails, an undue movement of the alæ nasi, or any other signs of impaired respiration, may lead to the discovery of danger the most grave and imminent.

Again, the appearance of symptoms of disturbance in the nervous centres, over and above, or out of all proportion to that which might be accounted for by the general febrile condition, is of very serious omen. The occurrence of delirium, and especially of nocturnal delirium, is of comparatively little importance; but a marked drowsiness—sometimes alternating with delirium, sometimes persisting and increasing in intensity—is a very serious symptom, forecasting that mode of termination which is by no means rare in Erysipelas, viz., "coma" from either effusion within the cranium or impairment of the brain-nutrition by embolism of the small vessels. It is, however, by no means probable that in all cases terminating thus, there is or has been an extension or metastasis of inflammation to the membranes of the brain. In some instances there are symptoms of "meningitis" observed during life, and evidences of its presence and its results may be discovered after death; but in others the cerebral symptoms are those of oppression rather than excitement, and the post-mortem appearances are those of effusion only, or of that embolic occlusion of vessels described by Dr. Bastian. In the latter class of cases the symptoms are probably due partly to the direct effect of altered blood upon the nervous centres, and also to the indirect effects of that alteration in leading to passive effusion, or unobstructed circulation. The relation between dropsy and Erysipelas has already elicited remark.

Erysipelas is a much more serious disease when epidemic or endemic than when it occurs sporadically; and the "type" of the epidemic—as observed in other instances which have been watched to their termination—will be the basis for an opinion as to the probable issue of a particular case.

It is said that the wandering or "erratic" form of the disease is attended with considerable danger, but in my own experience cases have exhibited this character to a high degree and yet have not only terminated happily, but have never exhibited the slightest disposition to do otherwise.

The cases of Erysipelas which are the most dangerous are those which, commonly occurring after wounds of considerable extent—either accidentally or scientifically produced—are attended with much diffuse cellular inflammation; and which, finding their way, if not from their commencement, at least very early in their history, into the hands of the surgeon, will not be described in this place.

TREATMENT.—As I believe that the class of cases which have been described in such manner as to justify the use of antiphlogistic treatment, do not exist except in the histories of the past and the imaginations of the present, it appears to me unnecessary to say how much blood should be taken from the arm of a man, provided that he is found in a condition that we may never meet with.

The *general* medical treatment of Erysipelas resembles rather that of the adynamic fevers than of inflammations, even supposing that the latter should present occasionally what is called a "sthenic" form. Almost all the cases—so far as my own experience reaches, all—that come under the care of the physician from the first, not only bear well, but are positively benefited by, supporting and tonic treatment. The kind and degree of such treatment must be determined by, and proportionate to, the severity of the symptoms which have been already described. In some cases, stimulants are required from the first, the conditions which necessitate the employment being identical with those which are common to that large group of diseases in which Erysipelas finds its place. When stimulants are not required at the onset, little or nothing

is gained by such use of salines, or any other general treatment, as shall do more than maintain a normal amount of the secretions. Thus, in very mild cases, in persons of average health, one or two doses of the simplest saline aperient may be all that is requisite. When the disease is more severe, and exhibits a tendency to spread after the balance of secretion has been restored, the patient at the same time becoming restless and exhausted, the most efficacious general treatment consists in the administration of bark with ammonia, during the day, and an efficient but not heroic opiate at night. Should the adynamic symptoms increase, large quantities of alcoholic stimulant are required at short intervals; and the amount that may be taken with advantage is as large as that which has been found useful in any of the specific fevers.

[In the period preceding the recent prevalence of alcoholic stimulation in practice, ample opportunity occurred for the observation of cases of hospital Erysipelas, in the treatment of which the use of alcohol was the exception rather than the rule; and which often did very well without any stimulation at all.[1] The same experience has been prolonged much later with cases occurring in private medical practice. The conviction has hence resulted, that neither alcohol nor the tincture of iron is a necessity in *all* cases of Erysipelas. Like other inflammatory affections (see the last sentence under PATHOLOGY, *supra*), it may occur with either a *sthenic* or an *asthenic* type.—H.]

The tincture of the sesquichloride of iron of the London Pharmacopœia is by far the most useful medicine that I know of in the treatment of these cases. So marked is its action that it has been thought by some to exert a "specific" influence in Erysipelas; but without asserting that it possesses such power in the strict sense of the word "specific," it may be well to mention that its utility appears equally great in diphtheria, and perhaps still greater in cases of diphtheroid sore throat. The essential condition of its success is its administration in large and quickly repeated doses; it has often happened that disappointment has arisen in the use of this tincture of iron, but in most of these instances the tincture has been given in doses of ten or fifteen minims three times daily, and such doses are certainly useless. But when the tincture is given in doses of forty minims, or even more, every four hours, the results have usually been most favorable. The most convenient form for its administration is a mixture containing in each dose forty minims of the tincture with an equal quantity of spirit of chloroform and glycerine, with an ounce and a half of water. The effects of this medicine may be seen sometimes after the first, often after the second, dose: the local inflammation ceases to extend; the inflamed part becomes paler, less tender, less swollen; the feeling of exhaustion is diminished, and with it such symptoms of exhaustion as exaggerated frequency of pulse, and dry brown tongue; the temperature falls; and sleep frequently ensues. As soon as such changes take place the quantity of the tincture may be reduced. It is not, however, safe to trust to this medicine alone; alcoholic stimulant is often required at the same time, but the action of the former has been too obvious in numerous cases for it to be confounded with that of the latter, or to be mistaken for those curative processes which occur in the natural history of the disease.

In the *local* treatment of Erysipelas two things are to be strenuously avoided; the one anything which shall expose the skin to variations of temperature, and the other anything which shall interrupt its natural function. Among the former are included exposure to draughts, and to the chilling effect of wet applications; among the latter the covering of the skin with any oily matters, ointments, &c. It has occurred to me frequently to see Erysipelas spreading rapidly under the use of "cooling lotions," and to see it arrested by their discontinuance, and the application of simple dry flour, violet powder, or oxide of zinc—the inflamed part being sometimes covered lightly with dry cotton wool; the latter, however, being really necessary only when wishing to protect the patient from such draughts of air as are almost unavoidable in any large rooms, and which are sometimes quite unavoidable, and absolutely pernicious to certain classes of cases which find their way into the very well-ventilated wards of hospitals.

The application of collodion has appeared to me of use only when the Erysipelas has been closely limited in extent. Its application over a large surface has not only failed to do good, but, in consequence of its cracking and leaving rough edges, has done positive harm.

[Not only by this application, but by nitrate of silver, acetate of lead, ice water, &c., the direct suppression of the cutaneous affection over an extended surface appears to be unsafe; being sometimes followed (as in the analogous instances of scarlet fever, measles, &c.), by cerebral or pulmonary congestion.—H.]

Various attempts have been made to arrest the spread of the inflammation, by some applications to the sound skin in the direction of, but beyond, its extending margin. The most approved of these has

[1] *E. g.*, in the Pennsylvania Hospital, 1845–50, *et seq.*—H.]

been, and is, the application of nitrate of silver, and there are facts to warrant this approval. It is desirable, however, not to attach too much importance to this measure. When nitrate of silver is applied in such manner as to affect the integrity of the true skin, I have seen Erysipelas start from the line of its application as from a new focus; and when, on the other hand, this evil has been carefully avoided, I have seen the Erysipelas extend through the line upon which it had been used.

[At the very *beginning* of an attack of Erysipelas, a facility of arrest by the mildest emollient applications, especially fresh lard or cold cream (unguentum aquæ rosæ), reminds one of the prevention of a conflagration by the extinction of a fire at its commencement. Probably no local application will do more good at any stage, than such unguents, unless when the subcutaneous areolar tissue, lymphatic vessels or veins are so deeply involved as to make appropriate the use of poultices of bread, slippery-elm bark, or flaxseed meal.—H.]

As already stated, the tendency to death from Erysipelas exists in three principal directions. To that by asthenia I have already referred; the other two which are most important, are either by implication of the brain, or by obstruction to the respiration. In the former the symptoms may be due to the blood-poisoning, or to extension of the inflammation to the meninges. When the cerebral symptoms are referable to the condition of the blood, no change in the treatment is required; but when to meningitis or meningeal congestion—supposing that this diagnosis can be accurately made—the application of ice to the head, and warmth to the extremities, together with free purgation, will be found useful. It is certain that such measures will relieve and lead to the cure of many cases of a most unpromising appearance.

When the danger to life depends upon interference with the respiration, either the fauces or the glottis is the most usual seat of mischief. It is then necessary to relieve, if possible, the swollen mucous membrane, and so allow of the passage of air through the larynx; but when this cannot be accomplished, the surgeon should be ready to perform laryngotomy or tracheotomy.

When pus is known or is supposed to exist, even in small quantity, in the neighborhood of important organs, such as the eye or the glottis, it is necessary that incisions should be made for its evacuation. When it exists in larger quantity under the skin of limbs, in the neighborhood of joints, or glands, the same plan should be adopted; and *à fortiori*, when large tracts of cellular tissue are so affected, and the case resembles that of "diffuse cellular inflammation," free incisions are necessary. For all the details of the treatment of Erysipelas, when thus requiring surgical interference, the reader is referred to systematic and other treatises on surgery.

In conclusion, it need only be stated that all those hygienic measures, such as good feeding, fresh air, and quiet, which are essential in the treatment of all acute specific diseases, are required and under similar direction in the treatment of Erysipelas.

VARIETIES.—The following terms have found their way into ancient and modern books, and have been used to denote the several so-called varieties of Erysipelas:—

E. neonatorum.
E. complicatum, et simplex.
E. idiopathicum, et symptomaticum.
E. verum, et spurium.
E. phlegmonosum, vesiculare, bullosum, &c.
E. acutum.
E. erraticum.
E. nervosum, œdematodes, &c.

The above list shows that the principles of nomenclature have varied widely; names having been constructed on the fact of their representing such conditions as—the age of the individual attacked; the presence or absence of complications; the supposed mode of origin; the fact of the disease being Erysipelas or something else; the degree of development of one or more anatomical elements of the disease; the time of its duration; the mode of its distribution; and the nature of its complications.

It does not appear to me to be necessary to explain these words; their enumeration is sufficient to show their unscientific character and practical inutility, and to lead towards the hope that they may not be perpetuated.

PYÆMIA.

By John Syer Bristowe, M.D.

The present article will be limited to the consideration of that morbid state of the system to which the term "Pyæmia" has of late years been generally applied. This morbid state is closely related to phlebitis; with which disease it was until recently confounded, and on which it undoubtedly often supervenes. It is related to those diseased processes which Virchow has named "thrombosis" and "embolia;" for the secondary deposits which characterize Pyæmia immediately depend, in great measure if not solely, on the obstruction by solid material of the vessels leading to the spots in which these deposits occur. It is related to erysipelas, and such like "unhealthy" inflammations; for not only does it occur as a sequela of these affections, but their neighborhood serves often to induce Pyæmia in patients who but for this neighborhood would have escaped. Again, it is related to puerperal fever; or, to speak more precisely, the loosely applied term "puerperal fever" includes, with many other diseases, a large number of cases of puerperal Pyæmia. And lastly it is related to several more or less well-defined morbid conditions of the system (septicæmia) brought on by the entrance into the blood, through the veins or lymphatics, of various non-specific animal poisons. These related affections will all be fully discussed in their proper places; and will be noticed here so far only as the due elucidation of the subject before us renders necessary.

I. Etymology.

The word "Pyæmia," derived from the two Greek words πύον and αἷμα, signifies literally pus in the blood. Its English synonyms are "purulent absorption" and "purulent infection." Every one of these terms implies a theory, viz. — that an essential feature in the disease to which it relates is the presence in the circulating fluids of the elements of pus. In this respect they are all objectionable; for while some authors accept this theory of the disease, others (whose opinions are equally well entitled to respect) reject it, and to them the name is necessarily the embodiment of error. The term "Pyæmia" is nevertheless a convenient one; it has come to signify to the practising medical man, quite apart from all theoretical considerations, a form of disease attended with certain definite symptoms and certain definite anatomical lesions; and even if some equally euphonious but less objectionable term were now to be proposed, it is more than doubtful whether the inconvenience of its substitution would be attended with any adequate compensatory advantage. In this qualified sense the term "Pyæmia" will be used in the following pages.

II. Definition of Pyæmia.

Pyæmia is a disease originating often in contagion, and attacking for the most part those who are suffering from the results of serious injuries attended with wounds, or who have undergone grave surgical operations, or who are laboring under acute suppurative inflammation involving bones, or in whom parturition has recently occurred. Its onset is usually sudden, and marked by the occurrence of a severe rigor followed by profuse perspiration. Rigors succeeded by perspirations for the most part recur; the pulse becomes feeble, rapid, variable, often intermittent, the respirations shallow and frequent, and cough (attended or not with expectoration) commonly shows itself; the tongue generally becomes dry and furred; the appetite fails; and nausea, vomiting, and diarrhœa not unfrequently supervene; the surface generally soon gets sallow or even distinctly jaundiced; the patient acquires very much the aspect of a person suffering from enteric fever—delirium at night often comes on; but he remains for the most part conscious, at least when roused; and soon becomes excessively feeble and prostrate. It happens often that, in the progress of the malady, inflammation, or even suppuration, occurs in some accessible part or parts, especially in or about the joints and in the muscular and cellular tissues. The duration of Pyæmia is generally from about four to ten days; but it now and then becomes chronic, and may then last for several weeks or even longer. Its result is almost invariably fatal. The chief lesions discoverable after death are patches of hemorrhage, or of inflammatory consolidation, or abscesses, scattered in various proportions among the different

organs and tissues of the body, but occurring far more often and far more numerously in the lungs than elsewhere. The part antecedently affected is generally found in a state of suppuration, and unhealthily inflamed or sloughy.

The most characteristic features of Pyæmia seem to be:—*first*, its supervention on certain special conditions of the system; *second*, the occurrence of rigors with perspirations; *third*, the presence of jaundice; *fourth*, the formation of external abscesses; *fifth*, the great prostration and early death; and *sixth*, the occurrence of certain characteristic lesions, easily to be recognized after death.

III. Pathology of Pyæmia.

1. *Morbid Anatomy.*

Discarding, in our detailed account of the morbid anatomy of Pyæmia, those abnormal conditions of organs and tissues which may be now and then discovered after death from this disease, but have only a fortuitous connection with it, we will limit ourselves to the consideration of those morbid changes only which form, so to speak, an integral part of Pyæmia.

(*a*) *Morbid Anatomy considered generally.*—The lesions which characterize Pyæmia consist in local congestions, extravasations of blood, inflammatory deposits, abscesses, and necroses. Simple congestion is a phenomenon which is apt to disappear after death, or to be modified and masked by mere post-mortem changes; it is frequently observed during life to accompany superficial pyæmic inflammations, it is frequently recognized after death in the vicinity of so-called "secondary deposits," and doubtless as a rule it precedes all the more important changes which attend this disease. Congestion is therefore a real and important link in the chain of pyæmic events, but its presence or absence *post mortem* cannot in all cases be taken as trustworthy evidence of its presence or absence during life. Extravasations of blood are of almost constant occurrence; sometimes they appear as petechial spots or vibices; sometimes as clots infiltrating the tissues of organs—abruptly marginated and resembling patches of pulmonary apoplexy; sometimes they form decolorized fibrinous masses, much like the fibrinous "blocks" observed under other conditions in the spleen, in the liver, and in the kidney. Inflammatory deposits are rarely if ever absent; and may be associated or mixed up with other morbid conditions, such as extravasations of blood, or may occur independently of them. In the former case they often surround the extravasations, and in connection with serous surfaces form distinct false membranes. In the latter case they constitute, in the lung, patches of lobular hepatization; and produce in other organs nearly equally well-marked changes. The term "secondary abscess" has been largely employed to designate the localized morbid processes taking place in the course of Pyæmia. And in most cases of Pyæmia abscesses doubtless exist. Sometimes, especially in joints and certain other places, suppuration takes place so instantaneously that the formation of pus would almost seem to be the first evidence in them of a departure from the condition of health. But more commonly the formation of an abscess is distinctly a later process, supervening on the extravasation of blood or on the effusion of lymph, and commencing either at the margins of the diseased patch, or at some central point in it, or involving the patch simultaneously in its whole extent. In some cases the cavities which pass for abscesses would seem rather to be the results of circumscribed gangrene than of true suppuration; for they yield a gangrenous odor, and contain a soft shreddy material, more or less adherent to the parietes, and infiltrated with a dirty-looking fetid puriform fluid. A few words will comprise all that need be here said generally in regard to the microscopic appearances observed in the several morbid conditions above described. The elements of blood, more or less modified, may of course be discovered in abundance in the hemorrhagic patches; fibrillated lymph, of the usual character, may be recognized in the inflammatory deposits, especially in those occurring on serous surfaces; and in those deposits occupying the parenchyma of organs granular matter, exudation corpuscles, and compound granule-cells are generally abundant; true pus cells are by no means infrequent in pyæmic abscesses, but the puriform fluid is sometimes found to consist of the debris of tissue, oily particles, and disintegrating cell-forms only.

Although the description which has just been given is generally applicable to pyæmic lesions, in whatever part of the body they may occur; there are yet so many differences between different organs in regard to the relative frequency with which they become involved, in regard to the relative frequency with which the various forms of lesions above enumerated affect them, so many minor differences dependent probably on structural and other peculiarities in the organs themselves, that a special description of these lesions, as they occur in the more important organs at least, can scarcely be avoided.

(*b*) *Morbid Anatomy of Lungs.*—In the lungs the diseased patches are scattered irregularly, but are generally most abun-

dant in the lower part. Their numbers vary considerably; sometimes they are exceedingly numerous, while one lung is affected, the other lung is quite healthy. Most of them abut more or less extensively on the surface of the lung. The individual patches range generally in size between that of a filbert and that of a pea. Sometimes they are smaller, and not infrequently larger; but in the latter case the increase of size is generally produced by the coalescence of contiguous patches. The characters of these patches vary very considerably. Sometimes they are distinctly apoplectic; that is to say, they are reddish-black, void of air, firm, abruptly marginated, yield blood-stained serum on pressure, and differ in no respect, but that of size, from the pulmonary extravasations due to mitral-valve disease. More frequently, although still distinctly apoplectic, they have undergone changes; they have become more or less decolorized, the margin has assumed a pale buff color, and the more central portions a rusty or brownish hue; at the same time some degree of softening has generally taken place. This softening often begins in, and may be limited to, the outer buff-colored layer, which then forms an interrupted puriform interval between the bulk of the diseased patch and the surrounding healthy tissues; at other times this process commences internally, probably in connection with the bronchial passages leading into the affected portion of lung; at yet other times a more general softening takes place, and the whole patch comes to form an abscess-like cavity. Sometimes, though much more rarely, the patches of lung disease are rather pneumonic than apoplectic, and then strictly resemble the patches of lobular pneumonia which supervene on laryngitis, diphtheria, and other diseases obstructing the larger air-passages. Under these circumstances the patches vary considerably in color, according to the relative degrees in which congestion and inflammatory deposits may be present in them, but are granular and have the ordinary aspect of inflamed lung tissue. Pneumonia of this kind, however, is more often combined with other pyæmic changes in the lung, than it is an uncomplicated phenomenon; sometimes surrounding apoplectic and other patches, and it may be combining several of these into a common mass of consolidation; sometimes forming independent patches scattered indiscriminately among them. Abscesses or collections of puriform matter are common, but very far from universal; often they are formed, as has been described, by the breaking down of clots, still more often they result from the purulent infiltration of pneumonic patches, and in both such cases are found in combination with patches of hemorrhage, or of inflammation, or of both. But there are some cases of Pyæmia where abscesses alone, sometimes surrounded by solid infiltrated parietes, sometimes by breaking-down tissue, are discovered. In these cases it would almost seem that the process is different from the beginning; that in some of them the formation of pus takes place coincidently with, if not prior to, whatever other inflammatory changes may be found associated with it; that in others the diseased and puriform patches are the simple result of necrosial disintegration or sloughing.

The lung tissue in which the diseased patches are imbedded may be, and often is, healthy; but very frequently more or less of it is congested or œdematous or even carnified. The bronchial tubes mostly present an excess of secretion, and those which are directly connected with the diseased tracts often contain pus, and sometimes the rusty tenacious fluid which characterizes pneumonia.

The subpleural tissue, especially that investing the lung, is generally the seat of extravasations of blood; and appears therefore studded more or less thickly and more or less irregularly with petechial spots and vibices. The surface of the pleura may be smooth and healthy-looking; or it may be invested in its whole extent by a layer of recently effused lymph; and the cavity may be occupied in a greater or less degree by transparent, opaline, or even distinctly purulent fluid. But, perhaps more commonly, the lung is only partially covered with lymph—each lump of pulmonary disease, which abuts on the surface, forming a centre of inflammation and of a disk of inflammatory exudation which is thickest at the centre and becomes thinner and thinner as it recedes from that point, until at length it ceases. This lymph presents a reticulated surface, and differs in no degree, microscopically or otherwise, from that of ordinary pleuritis.

(*c*) *Morbid Anatomy of Heart.*—The surface of the heart, like that of the lung, is often studded with extravasations of blood; and these are generally most abundant about its basal portion. Similar extravasations may also be seen in the substance of the muscular parietes, and beneath the endocardium. In the walls of the heart, too, may not infrequently be discovered (generally in the midst of extravasated blood, or at all events within a zone of congestion) yellowish spots, from the size perhaps of a horse-bean downwards, which consist either of muscular tissue infiltrated with some inflammatory exudation or of a cavity full of pus or puriform fluid, or of broken-down and disintegrated tissue. The muscular fibres in and around these spots will be found under the microscope to be more or less

broken into fragments, devoid of transverse markings, and studded thickly with minute oily molecules. The diseased patches in the heart vary much in number; sometimes not more than one is present, sometimes they are almost innumerable. They vary too as regards their position; perhaps they are most common about the base of the ventricles, but no part is free from liability to them, and they are sometimes found in the musculi papillares. As in the case of the lungs, so here, the patches of disease act as centres of inflammation. When they reach the pericardial surface, they induce inflammation in that membrane, with exudation of lymph; and when they reach the endocardial surface, they may lead to important changes in the endocardium itself. In the former case the exudation is of the same nature as that which occurs in connection with the pleura; in the latter case the endocardium itself is apt to become thick and granular, from interstitial inflammatory deposit, and its free surface to be studded with so-called "vegetations." Occasionally vegetations become deposited upon the valves. Excepting the various morbid conditions just described, the heart is generally found quite healthy.

(*d*) *Morbid Anatomy of Liver.*—Jaundice is a marked, though not an invariable symptom of Pyæmia; yet nothing has been detected *post mortem*, in the condition of the liver to explain its occurrence. Frerichs asserts[1] that "the bile ducts are open, and usually pour out a little thin secretion," and that "the organ itself is in most cases anæmic and dry." The liver in these cases has, in fact, a healthy appearance, except in so far as it happens to be the seat of special pyæmic changes. These changes consist in congestions, inflammatory exudations, and localized disintegrations or suppurations. They have no special seat. The earliest condition of disease, and one that is often alone seen, is the presence of congested patches of a port-wine hue. These vary in size and shape, have often a superficial area of two or three square inches, dip to a greater or less extent into the substance of the organ, and for the most part include irregular patches of unnatural pallor. Such patches often differ, so far as can be ascertained, in color only from the surrounding healthy tissues. Sometimes studding these patches, sometimes occurring independently of them, spots may be seen of an opaque buff color, in which the liver tissue is evidently infiltrated and softened, and it may be broken down into a puriform pulp. These spots have usually around them a halo of congestion, and their contents consist sometimes chiefly of pus-corpuscles, sometimes of disintegrated liver substance only. But besides these, larger abscesses are not infrequently met with—abscesses the size of a filbert, a chestnut, a hen's egg, or of still larger dimensions. These generally contain a greenish-colored purulent fluid; and are sometimes, judging both from their odor and from their appearance, distinctly gangrenous.

(*e*) *Morbid Anatomy of Spleen.*—When the spleen is secondarily affected in Pyæmia, the morbid appearances which it presents are very much like those observed in cases of heart disease. They consist generally either of circumscribed extravasations of blood, or of fibrinous "blocks," which are both often of considerable size. The "apoplectic" clots tend to become decolorized at the surface and to break down variously into a puriform pulp; the fibrinous blocks are usually softer and more juicy than those of heart disease, and tend, like the clots, to liquefy. Distinct abscesses, too, of various sizes are often scattered throughout the organ.

(*f*) *Morbid Anatomy of Kidneys.*—The kidneys are frequently involved; and the morbid changes which occur in them are observed both more frequently and to a greater extent in the cortex than in the medulla. The medulla, however, by no means escapes. Sometimes these changes are limited to a single spot in one kidney, sometimes they affect both organs almost universally. But more commonly they are present in both and in some intermediate degree of severity. Occasionally no abscesses have formed, but almost the whole tissue of the organ is mapped out by tracts and bands of deep congestion, which alternate with and surround patches, of which the color is unnaturally pale. More frequently distinct abscesses are present; these are generally small and tend to become clustered; and both the individual abscesses and the groups of abscesses assume a linear arrangement, perpendicular to the surface of the kidney. The abscesses contain a distinctly purulent fluid, have invariably a margin of intense congestion, and vary generally in size from that of a pea or horse-bean to an extreme degree of minuteness. Sometimes, however, they are so large as to contain an ounce or two of pus. They can almost always be seen on removing the capsule of the organ, and very often the removal of the capsule allows the contents of the more superficial ones to escape. The formation of pus in these abscesses seems to take place, originally at least, in the intertubular tissue; and very often in the early stages the Malpighian bodies and tubules in the affected spot are quite healthy.

(*g*) *Morbid Anatomy of other Abdominal*

[1] Klinik der Leber-Krankheiten: Sydenham Soc. Transl., vol. i. p. 162.

Organs and of Peritoneum.—Congestions and petechial extravasations are apt to occur, both in the gastro-intestinal mucous membrane, and in that of the genito-urinary apparatus. As regards the first-named mucous tract, it is an interesting fact that there are occasionally observed upon it, and more especially on that part of it which belongs to the cæcum and colon, patches of granular exudation. Occasionally, too, the intestinal submucous tissue becomes the seat of well-marked pyæmic deposits, which may lead to the destruction of the mucous surface over them, and the production of a sloughy ulcer, not unlike the ulcer of enteric fever or that which follows the opening of a boil.

As regards other organs connected with the abdomen little need be said. They are rarely affected secondarily, and even when they are thus affected they present few points of importance or interest either to the pathologist or practitioner. Of them all, the prostate and the testicle probably most often undergo suppuration.

The pyæmic affections of the peritoneum resemble those of the pleura and pericardium. Sub-serous extravasations of blood are common; and inflammatory changes occurring within viscera (especially the liver and the spleen) lead to inflammation in the serous surface external to them, which may remain limited in extent, or become general peritonitis. Very often an abscess forms between the surface of the diseased lump and whatever organ or part is in contact with it—the abscess being limited laterally by adhesions, which correspond accurately to the margin of the lump. The same thing, though on a much more minute scale, is of general occurrence in the case of the lungs and pleura.

(*h*) *Morbid Anatomy of Brain.*—The brain does not appear to be a very frequent seat of pyæmic changes. When present, however, they consist of congestions with extravasations of circumscribed softenings, and of abscesses. The extravasations affect chiefly the surface of the organ, and though perhaps generally petechial, sometimes become sufficiently abundant to occupy an extensive tract of the subarachnoid tissue. The circumscribed softenings and abscesses occupy indifferently any part of the brain—the gray matter, the white matter, the cerebrum, the cerebellum, the corpus striata, the optic thalami, the pons Varolii—no part necessarily escapes. The former are yellowish, more or less congested, more or less softened, patches, such as are met with in cases of so-called "embolism" of the brain, but of smaller size, varying mostly from that of a horse-bean downwards; the latter are distinct abscesses containing glairy greenish-yellow pus, and sometimes attain considerable dimensions. The number of foci of disease present at one time varies very considerably; sometimes not more than one or two are discovered, sometimes they are so numerous that scarcely any part of the brain, so large even as a chestnut, is found free from them. The softened patches contain, in addition to disintegrated nervous tissue, vast numbers of compound granule cells.

(*i*) *Morbid Anatomy of Organs of Sense.*—Of the organs of sense the eye only calls for special remark. This organ occasionally becomes the seat of suppurative inflammation, especially in cases of puerperal Pyæmia. In these cases the affection of the eye is characterized[1] "by redness of the conjunctiva, intolerance of light, and contracted pupil; rapidly followed by opacity of the cornea, and excessive chemosis." The eye ultimately sloughs, and its contents escape.

(*k*) *Morbid Anatomy of Bones and Joints.*—Suppuration sometimes takes place in connection with bones. The affected bone or portion of bone then becomes rapidly denuded of periosteum; fetid pus accumulates upon its surface, while at the same time probably pus infiltrates its cancellous texture, and rapid necrosis ensues. The joints are much more frequently affected than the bones. The synovial fringes become intensely congested, and the synovia increased in quantity or replaced by pus or puriform fluid. The capsule of the affected joint becomes distended, and the parts external to it become more or less inflamed. When the fluid within the joint assumes a purulent character, which is by no means always the case, it often happens that the parts of the lining membrane which had been congested become pale, that destruction of cartilage takes place, that the joint, in fact, becomes disorganized. All joints, small as well as large, are liable to be affected.

(*l*) *Morbid Anatomy of Cellular Tissue and Muscles.*—Again, the secondary effects of Pyæmia show themselves constantly among the muscles and in the cellular tissue of the body generally. Extravasations of blood here are exceedingly common, inflammatory congestions and exudations frequently occur, and abscesses (often of large size) form rapidly, and almost without warning. These morbid changes are often observed in the walls of the chest and belly, and in the neighborhood especially of joints, for inflammation of which latter parts they are then very liable to be mistaken. Pyæmic abscesses have been met with in the tongue.

[1] Arnott, Medico-Chirurgical Transactions, vol. xv.

(*m*) *Morbid Anatomy of Skin.*—The skin necessarily partakes sooner or later in any morbid process which is going on immediately below it; and hence discoloration of skin is frequently observed over superficial pyæmic infiltrations and abscesses. When jaundice is present, the skin necessarily partakes in the general icteroid tinge. Apart from the above, the morbid conditions of the skin in pyæmia are not very important. Petechiæ are not very common; sudamina are frequently present; and occasionally vesicular and pustular eruptions have been observed.

(*n*) *Relative Frequency with which Organs are affected.*—There is considerable difference in the relative frequency with which the various organs and tissues of the body become secondarily affected in Pyæmia. The lungs rarely escape, and not infrequently are the only parts in which morbid changes are observed. The viscera affected next in frequency to the lungs would seem to be the kidneys. After these the liver, spleen, and heart. Then perhaps the brain. Among organs less often affected may be enumerated the intestinal canal, the testis, the prostate, and the eye. The joints and the general cellular tissue of the body become of course very frequently the seat of secondary affections. And indeed, from the great extent of the one and the great number of the other, disease in these parts is without doubt constantly overlooked. The serous membranes, at least one or two of them, are rarely found uninflamed: this condition may occasionally depend on morbid processes originating in themselves, but in the great majority of cases, as has been already explained, is due to the extension of inflammation from some subjacent organ.

(*o*) *Morbid Condition of Blood.*—The chemistry of the blood in Pyæmia has not, so far as we know, been investigated. We must content ourselves therefore with the discussion of its physical properties. With certain important exceptions, which will be presently fully considered, the blood in Pyæmia presents no important differences from the blood of health; it retains its natural color, the blood disks and the white corpuscles preserve their due numerical relation to each other and to the mass of the blood, and so far as can be recognized their normal characters. Sometimes the blood appears to be unusually fluid, to present in the cavities of the heart and in the larger vessels only traces of coagulum. More commonly it coagulates in the usual way; and we find distinct clots in one or more or all of the cavities of the heart. Sometimes these are ordinary-colored post-mortem clots, sometimes they consist wholly or almost wholly of pure fibrine; and in either case they may be prolonged in a cylindrical form into the large vessels. There is no special tendency for the right side of the heart to be occupied, far less to be occupied exclusively, by these clots. They may be found there, it is true, and found there while the left cavities are contracted and empty. But in many cases, while the right side is empty, the left is distended with them. In fact there is nothing in the situation or character of these clots to distinguish them from those which are found in many other forms of disease. The adherent rounded clots, which soften in their interior into a puriform pulp, have sometimes been supposed to characterize Pyæmia. But this is clearly a mistake.[1] Such clots are far more commonly observed in other cases; indeed are altogether exceptional in Pyæmia; and when present are evidently accidental and probably trivial complications.

The most important and characteristic changes of the blood in pyæmic cases are manifested more particularly in the veins of the part at which infection is supposed to have occurred, and in the small arterial twigs leading to the spots in which secondary lesions have become developed. The veins leading from the seat of supposed infection have been examined over and over again with extreme care; and the general results of these examinations may be shortly summarized. In some cases the veins, though traced into suppurating and even sloughing regions, are found, both as regards their walls and their contents, apparently entirely healthy; in other and more numerous cases they are seen to be in various ways and degrees diseased. Their parietes are thickened and indurated; they may be seen to communicate by orifices, resulting from ulceration or some other cause, with the morbific elements in which they are lying imbedded; and their interior is occupied by coagula. These coagula are mostly adherent, and more or less decolorized; they may be solid throughout, but more commonly are reduced in their interior into a reddish or yellowish puriform pulp or fluid. This fluid appears generally to consist merely of disintegrated fibrine, but in some cases is undoubtedly true pus.[2] It is mostly, but not always, sepa-

[1] See Papers on Softening Clots in the Heart, in the Transactions of the Pathological Society of London. Vol. ii. p. 134, and vol. xiv. p. 71.

[2] The following case bears out the statement in the text. A man died of erysipelas of the face and Pyæmia. "The brawny tissue of the face was infiltrated with pus, and pus oozed from numerous divided vessels. The facial vein of the right side was thickened and surrounded by indurated adherent tissue, and its canal was dilated. The first inch of its course was occupied by thick purulent fluid; to this succeeded a cylindrical adherent

rated from the venous walls by a layer of still consistent fibrine; and is generally shut out from the proximal portion of the venous channel in which it lies by a continuation of this layer of fibrine, which forms a kind of septum or diaphragm between them. Generally the rounded extremity of the hollow fibrinous cylinder thus formed has adherent to it and prolonged from it a process of ordinary colored clot. Sometimes one, sometimes several veins are found thus affected, and sometimes a considerable length of one is converted into an elongated abscess.

The ultimate arterial twigs, distributed in the lungs to the masses of diseased lung structure, seem to be invariably occupied, indeed distended, by a soft pulpy yellowish material, or by something more nearly approaching to ordinary coagulum, This material is found to consist mostly of mere disintegrated fibrine presenting the debris only of cells. But sometimes it contains distinct pus[1]—that is to say cells resembling in all their visible characters pus-corpuscles or the white corpuscles of the blood, but so abundant and so closely aggregated as wholly to negative the notion of their being normal blood-elements. Similar coagula have been detected in the small vessels leading to the diseased patches occurring in other organs besides the lungs—in the vessels for example of the heart, the spleen, and the kidneys.

(*p*) *Connection between blocked-up Condition of Vessels and Secondary Deposits.*—So constantly are these coagula found in the small arteries, if looked for carefully, that the conclusion is forced upon pathologists that there is between them and the patches of diseased tissue a relationship of cause and effect. It might be surmised that the coagula in the bloodvessels are secondary to the local pyæmic formations with which they are connected, due to obstruction in the capillaries of the affected part and consequent stagnation of blood in the vessels leading to them. The characters of the coagula show, however, that this cannot be the true explanation of their mode of formation. They are not mere coagulated blood, nor even mere coagulated fibrine; but, if fibrine at all, are fibrine which has undergone changes, requiring time for their production, and often in point of time clearly in advance of the changes which have taken place in the patches of diseased tissue. There can be no doubt indeed that the sequence of events occurs in the reverse order; that the small afferent vessels become blocked up, and that on this blocking up supervene those changes which, according to circumstances, end in extravasation of blood, inflammation, purulent infiltration, or gangrene. This view is partly based on direct observation in Pyæmia itself, partly on corroborative evidence derived from other sources.

Thus, it has been clearly established by experiments that if a small artery leading to any spot be obstructed, that spot becomes the seat of congestion and inflammation. The careful experiments of Mr. Wharton Jones[1] show that if in the frog's foot such an artery be divided, and the capillary area to which it leads be thus cut off from all direct supply of blood, these capillaries nevertheless become filled with blood by regurgitation from neighboring anastomosing vessels; and further, that since by the same operation they have been cut off from the direct influence of the heart's systole, the blood which is poured into them becomes stagnant there, and intense congestion results. Again, the experiments of Cruveilhier, Sédillot,[2] Henry Lee,[3] and others show that, if mercury, oil, pus, fibrine, be injected into the veins, they become impacted in the small arteries connected with the network of capillaries next beyond the seat of operation, occlude them, and induce congestion, if not hemorrhage, and inflammation in the respective areas to which they lead. Each of these experiments has no doubt some point of special interest, but all concur in establishing one common fact of fundamental importance, viz., that the sudden stoppage of the direct supply of blood to a limited area tends to the production in that area of congestion and inflammation—of the very processes in fact which mark the secondary effects of Pyæmia.

The analogies afforded by other forms

---

fibrinous coagulum, the distal extremity of which formed a hollow cone. The lower or proximal extremity gradually dwindled away, and was succeeded by purulent fluid: this latter continued throughout the rest of the facial vein as far as its junction with the jugular. No coagulum or adhesion separated this fluid from the general circulation. The purulent fluid found in the veins presented under the microscope large numbers of corpuscles, but they were mostly smaller and more irregular in shape and size than normal pus corpuscles; and few, if any, presented division of their contents under the influence of acetic acid. There were numerous secondary abscesses in the lungs."—*Manuscript Notes of Post-mortem Examinations, St. Thomas's Hospital.* Nov. 2, 1857.

[1] See Transactions of Path. Soc. of London, vol. xiii. pp. 203, 204. See also Dr. Wilks's Report on Pyæmia, in the Guy's Hospital Reports, vol. vii. 1861.

[1] Astley Cooper Prize Essay, "On the State of the Blood and Bloodvessels in Inflammation." Guy's Hospital Reports, Second Series, vol. vii. p. 23 et seq.

[2] Sédillot, De l'Infection purulente.

[3] Lee on Phlebitis.

of disease are still more to the point. When arteries become obstructed either from morbid changes in their walls, or from plugs in their interior, the parts which they supply fall into an unhealthy condition. It is needless to dwell upon the changes which take place in the lower extremities, when in old age the arteries become closed by accumulated atheromatous and earthy deposits, or even upon the circumscribed softenings in the substance of the heart (leading to rupture), which attend similar changes in branches of the coronary artery. The effects, however, of thrombi and emboli must be considered a little more in detail. It has been clearly ascertained by the researches of Virchow, Kirkes, and succeeding observers, that, in cases of heart disease with vegetations on the valves, these vegetations are apt to be detached, carried with the onward current of the blood, and impacted in the first artery they reach which is too small to permit of their transit. It has been clearly ascertained that such detached fragments, or "emboli" as they are termed, become fixed in the arteries of the brain (more especially in the middle cerebral artery), and lead, in the brain structure beyond, to circumscribed congestion, inflammatory softening, and disintegration of tissue; that they become fixed in the small arteries of the spleen, and lead to extravasations of blood and so-called "fibrinous blocks;" that they become fixed in the renal arteries, and lead at one time to exudation of blood and lymph, at another time to minute abscesses; that they become fixed in the arteries of the retina, and lead to similar results there; that in fact they may occlude any artery of any organ, and thus lead to specific changes in the bit of tissue which that artery supplies. It has been clearly ascertained also that clots or "thrombi" formed in the interior of veins break down and crumble; and that their fragments, swept away by the stream of the blood, pass onwards with it from the smaller to the larger veins, through the cardiac cavities and orifices, and thence still onwards along the arteries, until, like the broken off cardiac vegetations, they become impacted, and by their impaction produce identical results. Further, it has been ascertained that clots form spontaneously, so to speak, not in the veins alone, but in the heart's cavities, and in the arterial system; and that the clots thus formed in the latter situation occlude the arteries in which they arise, and lead in the parts beyond to the same changes as have been described in connection with emboli. Here again, throughout the whole series of allied but not identical processes, we find that obstruction of the supplying artery causes in the part supplied precisely those lesions which occur as the specific local manifestations of Pyæmia.

It has been already stated that the secondary effects of thrombosis and embolia are identical with those of Pyæmia; and essentially no doubt they are so. Yet there are between them certain minor differences—differences chiefly of degree—which it may be desirable to consider. This will be most conveniently done by taking three or four important organs and comparing the effects of these diseases upon them. In the brain the influence of embolia or thrombosis is almost invariably limited to a single spot; Pyæmia produces many spots of disease. The region affected in embolia is generally larger, at least in the beginning, than the individual regions affected in Pyæmia. Moreover, in the former case breaking down of tissue is far less rapid than in the latter case, and actual suppuration rarely if ever occurs. The pulmonary apoplexy attendant on heart disease is we believe generally, if not always, due immediately to thrombosis of branches of the pulmonary artery belonging to the apoplectic region, followed by congestion and rupture of the capillary network of the part. Now the clots of ordinary pulmonary apoplexy are almost identical with the pulmonary clots of certain cases of Pyæmia. Like them they become decolorized upon the surface, like them they may become more or less perfectly surrounded by a rim of softening or suppuration, or may present similar changes in their interior, and like them, when they abut on the surface of the lung, they lead to the deposition of a layer of fibrine on the overlying pleural lamina. But as a rule they are less numerous, and individually much larger, than pyæmic clots; and their tendency to soften, to suppurate, to slough, is far less. In the spleen, the wedges or blocks of effused blood, or fibrinous exudation, which so commonly result from cardiac emboli, are in their general aspect almost exactly like those connected with Pyæmia; but here again the tendency to rapid suppuration or decomposition distinguishes for the most part the one form of deposit from the other. In the case of the kidneys the differences are less pronounced: in both affections minute abscesses are of common and early occurrence; in both, hemorrhages and exudations of fibrine alone are occasionally met with. It would seem then that the chief distinction between pyæmic deposits and those resulting from simple embolia resides in the fact of the greater tendency of the former to undergo changes of degeneration and destruction. But this after all is chiefly a difference of degree; and the difference in this respect between them is no greater than the difference which may often be observed between actual cases of Pyæmia. Thus in one case

of Pyæmia hemorrhagic effusions only will be discovered in the internal organs, in another case patches of inflammation only, in a third abscesses, in a fourth gangrenous excavations; though more commonly doubtless these various conditions are to a greater or less extent commingled.

2. *General Pathology of Pyæmia.*

(*a*) *Conditions of System essential for the Development of Pyæmia.*—Exclusive of a few cases in which, from want of a trustworthy history or from some other cause, it has been impossible to determine what has been the original seat of disease; and of a few other cases which may be found recorded, wherein after very minute and careful investigation nothing that could be regarded as a starting-point for Pyæmia has been discovered, and which may possibly, therefore, have been idiopathic—exclusive of these, all cases of Pyæmia appear to take their origin in some one or more well-marked local conditions of disease; some coming naturally under the care of the physician, others under the care of the accoucheur, others under the care of the surgeon. We will enumerate them without particular reference to the department of practice to which they respectively belong. *First.*—Pyæmia frequently follows on accidental injuries, such as extensive burns or scalds, bruising and lacerating of tissues, and compound fractures, especially on fractures of the long bones, and of the bones of the head and pelvis. Such accidents often, of course, become repaired without any untoward complication; often they are followed by serious results, and even death, quite independently of anything approaching to Pyæmia; but often, and even at a time when they appear to be progressing favorably, the symptoms of Pyæmia come on and the patient dies rapidly of this disease. The occurrence of Pyæmia in these cases is generally distinctly preceded by sloughing, unhealthy suppuration, by erysipelatous inflammation, or some allied process. *Second.*—Pyæmia is the bane of certain operations. No operation possibly can be regarded as absolutely free from liability to the supervention of Pyæmia; but large operations, operations that is to say which leave extensive raw surfaces, especially therefore amputations of the larger limbs, operations too in which bones are involved, and operations in which certain parts (the bladder, prostate, and urethra, to wit) are implicated, are especially liable to the supervention of Pyæmia. But here again the pyæmic symptoms are mostly preceded by the occurrence of unhealthy processes at the seat of operation. *Third.*—Pyæmia is peculiarly apt to follow on acute suppuration taking place in connection with bones. Sometimes from an accident (unattended by breach of surface), sometimes, so far as can be made out, spontaneously, acute inflammation is suddenly lit up in connection with one of the bones—probably one of the long bones—suppuration rapidly takes place on the surface of the bone, between it and the periosteum, and in its interior; the bone dies; and in the course of these processes the symptoms of Pyæmia suddenly declare themselves. *Fourth.*—Phlebitis, as the disease is called, whether it be idiopathic or whether it be induced by injury or by operation,[1] is a pregnant cause of Pyæmia. This complication has been especially observed in connection with the operation of phlebotomy, in operations on varicose veins and hemorrhoids, and in connection with the wounding or tying of large veins in the course of certain other operations. *Fifth.*—Pyæmia is by no means an uncommon sequela of suppuration involving certain of the organs of sense, such as the eye and the internal ear. *Sixth.*—So-called "low inflammations" attended with suppuration—in the male in connection with the bladder, prostate, and urethra, and in the female in connection with the ovaries and other genito-urinary organs—are not infrequently succeeded by Pyæmia. *Seventh.*—The period immediately after parturition is peculiarly obnoxious to the occurrence of Pyæmia. "Puerperal fever" is the generic term which is used for a variety of diseases occurring shortly after child-birth. What these diseases are it is not our province now to discuss; but certainly one form of so-called "puerperal fever" and one which causes no inconsiderable proportion of the deaths ascribed to puerperal fever, is Pyæmia. *Eighth.*—Certain forms of so-called "unhealthy" inflammation are not infrequently followed by Pyæmia. Such are phlegmonous erysipelas, diffuse cellular inflammation, carbuncle, dissecting wounds, malignant pustule. *Ninth.*—Pyæmia is described as taking place occasionally in the course of certain febrile affections, such as typhus, enteric fever, and variola.

Now, in reviewing the above series of cases in which specially Pyæmia is apt to occur, several facts come into prominent relief. It would seem in the first place that, in such cases, Pyæmia is almost invariably, if not always, preceded by some local suppuration, and that this suppuration is erysipelatous, gangrenous, or otherwise unhealthy. Such is the case after injuries, after operations, after affections of the bones, of the organs of sense, of the genito-urinary organs, of the veins; such too is certainly often the case in puerperal

[1] Pyæmia is described as following on the operation of tying the funis in the new-born child.

women. It would seem in the second place that Pyæmia is peculiarly apt to supervene in cases in which bones are involved in these morbid processes. It would seem further that in the great majority of cases, if not in all, there is reason to believe that veins are in some way or other specially implicated. Thus it has been conclusively determined that wounds and injuries of veins, and suppuration taking place in connection with them, not infrequently lead to Pyæmia; it has been pointed out that the veins in the interior of bones are peculiarly thin-walled, and at the same time from their connections prevented from readily contracting, and that when inflammatory processes are going on in the interior of bones these vessels are necessarily peculiarly implicated; as regards the skull, again, it is clear that the bones which form it, besides having in their interior the veins of the diploe, are related by their inner surface to the venous sinuses—channels which if not thin-walled like the veins of bones, are like them permanently patent; the eye and the ear stand in much the same position as the skull itself, they are bounded in fact by osseous tissue which almost necessarily becomes involved when serious inflammation occurs in the adjacent structures, and they communicate almost directly by special veins with the sinuses, the peculiarities of which have been pointed out; again the prostate and neighboring parts are supplied with an almost superabundant net-work of veins; and lastly in parturient women the uterine portion of the placenta is provided with huge thin-walled venous sinuses which receive blood from the curling arteries and pour it into large uterine veins: at the time of parturition these are necessarily ruptured, and although by contraction of the uterus their orifices become in great measure closed, it is obvious that they are so circumstanced as to be peculiarly exposed to the influence of poisonous and other injurious processes going on in the interior of the uterus, or in connection with its lining membrane.

(*b*) *Essential Cause of Pyæmia.*—We are now in a position to consider what is the essential cause of Pyæmia. The sudden onset of the disease, the markedly febrile and characteristic symptoms which it exhibits, the limited term of its duration, and its terrible mortality, together with the occurrence of specific lesions, all suggest a close analogy on the one hand between it and certain contagious fevers—typhus fever, enteric fever, smallpox, diphtheria, and the like; on the other hand, between it and certain diseases, such as glanders and hydrophobia, arising from the inoculation of animal poisons. In each of these analogous cases the disease is due to the entrance into the system of some morbid poison, to the circulation of this poison through the vessels with the blood, the chemical changes thereby induced in the blood, and through the agency of the blood in the system generally. In Pyæmia, too, it is manifest that the symptoms are due to the entrance into the blood of some *materies morbi*, and to changes thereby induced in that fluid and in the tissues through which it circulates. There are, however, marked points of difference as regards the mode in which the several classes of poisons above referred to enter the system. In contagious fevers it is mostly by the breath that the contagium takes effect; in glanders and in hydrophobia it is by inoculation; but Pyæmia, though in a certain sense contagious, is never imparted through the instrumentality of gaseous exhalations alone, and never even by inoculation, except the appropriate condition be present of a raw, suppurating, or sloughing surface. Further, though it may be imparted by some contagious influence, it may, equally originate *de novo*, but never probably becomes developed even in the latter case except in connection with some area of suppuration, and through the direct agency of that area. The poison which produces Pyæmia is evidently something more gross, something less subtle, than the poisons of those diseases with which we have compared it, and is capable only of acting on parts especially prepared as it were to receive and to develop it.

It is important to determine what this poison is, and how it gains an entrance into the system. It was at one time believed that pus, as such, is absorbed by the veins from the region of primary suppuration, and carried bodily to the various localities in which secondary accumulations of pus are discovered—that a true process of metastasis takes place. Again, it was imagined that this disease is simply phlebitis, in which the inflammation of the veins has extended to the vena cava and the heart. Arnott[1] maintained that the cause of what is now termed "Pyæmia" is "inflammation of the veins, the consequent production of pus in their cavities, and the entrance of this into the circulation." And since the period at which Mr. Arnott wrote, his views, with various more or less important modifications, have generally found acceptance. Thus, some have believed with Mr. Arnott that pus finds its way into the blood in consequence of the secretion of pus by the inner surface of some vein or veins inflamed by the extension of inflammation from surrounding parts; others have considered that pus is absorbed from some suppurating region by the open orifices of veins—orifices existing naturally (as in the

[1] Med. Chir. Trans. vol. xv.

uterus), or made by operation or disease; others again, have supposed that the pus-corpuscles carried with the blood become arrested in the capillary vessels of the lung, and there produce the characteristic lesions of Pyæmia, either by multiplying by means of cell-growth and thus forming an abscess, or by acting as foreign bodies and thus inducing congestion and inflammation in the surrounding parts. In support of some at least of these views, it has been pointed out that the veins connected with the seat of primary disease are often thickened, and occupied by adherent coagula containing within them a puriform fluid; it has been maintained that pus-corpuscles may be recognized in the circulating fluid; and it has been found experimentally that the introduction of pus into the veins leads to changes in remote organs like those of Pyæmia. But Virchow[1] (whose researches in connection with Pyæmia are most important) maintains that the puriform fluid in the affected veins is not pus, but simply disintegrated clot, that the clot is formed in the veins wholly independent of phlebitis, and that the diseased condition of the venous walls is not the cause, but (if related to it at all) the consequence of the clot within it. Again, though Sedillot[2] has taken pains to show that pus-corpuscles circulate in the blood in cases of Pyæmia, and may by their microscopic characters be recognized there, it is now generally allowed that pus-corpuscles do not mingle with the blood in the manner supposed, and that even if they did it would be impossible to distinguish them from the white corpuscles of the blood itself. And lastly, although it has been shown that pus introduced artificially into the systemic veins may produce lobular inflammation of the lungs, it has been shown that the pus acts in such cases as an embolus, and much in the same way as other substances which lead to mechanical obstruction of the small pulmonary vessels. The theory which at present finds perhaps most general acceptance is that of which Virchow[3] is the chief exponent. He denies that in Pyæmia pus (meaning, by pus, pus-corpuscles) enters the blood; he denies that pus is ever found either in the thrombi occupying the veins of the region primarily diseased, or in the small vessels leading to the patches of secondary disease; he asserts that what had been regarded as pus is merely disintegrated fibrine, and that the material choking up the small afferent vessels of a secondarily-diseased tract is simply an embolus resulting from the crumbling away of the fibrinous material occupying the veins at the seat of primary disease; he maintains that all secondary pyæmic formations and changes are thus the result of embolia, but that the differences which these formations exhibit in different cases are due to the difference of process which has led to the disintegration of the original thrombus. So much with regard to his explanation of the mode of production of secondary pyæmic lesions: to explain, however, the general symptoms of Pyæmia, to explain certain diffused inflammatory processes (as inflammation of joints and of serous surfaces), which do not seem to be easily explicable on the embolic theory, he assumes that in many cases of Pyæmia, at least, certain ichorous juices are also absorbed into and act upon the system. Thus, according to Virchow, it would appear that Pyæmia is a complex condition; that from the veins at the seat of a primary disease solid matters and poisonous fluids are circulated throughout the system; that the solid matters lead to the more material secondary lesions, the fluid matters to the more subtle changes, which combine to constitute the disease under consideration.

These views are intelligible, and give a plausible explanation of most of the phenomena of Pyæmia; but they do not, we conceive, explain all the phenomena of the disease, neither, as it seems to us, are they based on an impartial appreciation of all the facts. We admit that in the majority of cases the puriform fluid in clots is simply disintegrated clot, but we maintain that the true pus is occasionally met with in venous clots and in the clots of arteries, and that pus is occasionally discovered *in transitu* in the blood—not, we allow, in the form of scattered pus-cells, but in that of soft pellets.[1] We see

[1] Virchow's Cellular Pathology, translated by Dr. Chance.

[2] De l'Infection Purulente.

[3] Cellular Pathology, Lectures IX. and X.

[1] The following case of malignant and rapidly fatal scarlet fever may be quoted in confirmation of this statement:—"There were no pyæmic deposits; but the following was the condition of the blood in the heart's cavities: The left ventricle was empty, but all the other cavities were filled with largish fibrinous coagula. The greater part of the clot in the right ventricle consisted of perfectly decolorized, recently deposited fibrine, straw-colored, elastic, and semi-transparent. Embedded in its substance were a few small opaque whitish masses, which looked like clots of older formation entangled in the substance of the more recent one. These increased in number towards the pulmonic valves, and were very numerous in the cylinder of clot occupying the trunk of the pulmonary artery and its left branch, rendering it in fact somewhat nodulated. All these masses had an opaque, buff-colored, creamy aspect, were irregular in shape, and appeared in the majority of instances to consist of convoluted, folded, wrinkled, or twisted fragments as

no sufficient reason to believe that veins do not share in the morbid changes which are going on around them, or that phlebitis may not exist at a time when no appreciable thickening of their walls has taken place;[1] indeed, we have reason to believe that pus may be formed not only on the lining surface of these tubes, but even in the interior of clots adherent to them, by the communication to them of those tendencies to cell-production which are a part of the inflammatory process. We believe that in the above views undue importance has been attached to the embolic theory, too little to the independent formation of thrombi within the arterial system. We do not see how the embolic theory explains satisfactorily those cases of Pyæmia, starting from some portion of the systemic venous system, in which the lungs escape in great measure, or entirely, while secondary deposits are found, it may be, abundantly in other organs. Lastly, it seems to us as erroneous to regard the corpuscular element of pus only as pus, as it would be to attach that name to the liquor puris exclusively. Pus consists of both a solid and a fluid portion. On the whole, we are disposed to believe that, owing to some form of unhealthy process supervening in the region of primary disease, unhealthy pus or the element of unhealthy pus (call it ichor if you will) finds its way into the circulating fluid, and poisons it; that this poisoning partly shows itself in producing in the blood a tendency to coagulate in the smaller vessels, partly shows itself by inducing more subtle but even more serious effects upon the system at large. We are not disposed to deny that some of the local effects may really be due to embolism, some even to the impaction of coagulated masses of pus-cells; but we believe that thrombosis alone is the more general explanation of that obstruction of the minute vessels which leads to the secondary deposits.

(*c*) *Cause of relative Frequency of Pyæmic Deposits in different Organs.*—On either view of the question, it is easy to understand why the lungs should be, as they generally are, first and most seriously affected. For since the majority of cases of Pyæmia originate in connection with the systemic venous system, the poisonous matters which induce the disease must reach first the pulmonary capillaries; and in connection with these, which act as a kind of filter and purifier, their effects are naturally earliest manifested. Further, since, as regards the circulation, the lungs may be regarded as the equivalent of the whole of the body besides, it is obvious that even if all the morbid effects of Pyæmia throughout the system were produced simultaneously, the lungs would be still (if the morbid processes in them held any relation to the amount of blood passing through them) the equivalent in quantity of pyæmic disease of all the rest of the body, and would therefore far surpass in their liability to secondary deposits any other one viscus. It is not so easy to understand the differences presented by other organs as regards their relative liability to disease; why, for example, the brain should so often escape, why the spleen, the liver, and the kidneys should so often suffer: all are equally exposed to the effect of emboli originating in the lung; all are equally liable, it might be supposed, to the formation of thrombi in their smaller vessels. To explain these differences there must, we imagine, be something in the character of the circulation, something in the formation of the various organs, which modify both the tendencies to morbid changes in the blood circulating in them, and the mode in which these morbid changes affect their tissues.

(*d*) *Cause of different Character of Pyæmic Deposits in different Cases.*—What, we may now ask, is the explanation of those differences as regards the character of the local deposits (described on a former page)

---

though they had been formed and moulded in other parts, and had become entangled and compressed by the surrounding clot. The masses were somewhat soft, and could be separated readily from the fibrine investing them; and when separated some of them could be unfolded, but at the same time gave no clear indication as to what their original shape had been; some looked as though they might be collapsed bags, others were possibly cylindrical; none contained fluid. The same appearances were found in the right auricle. The left auricle contained, besides an ordinary clot, a single soft mass of the same kind as those that were found on the right side. Under the microscope the ordinary fibrinous coagula presented the usual characters of such formations; but the soft masses consisted entirely of corpuscles, which had the size and general characters of pus, and of which (under the action of acetic acid) the nuclei were divided into two or three spherules."—*Manuscript Notes of Post-Mortem Examinations, St. Thomas's Hospital*, September 15, 1858.

The soft opaque masses above described were very likely emboli carried from the seat of suppuration in the tonsil; but they were also, so at least it seems to the author, undoubtedly pus.

See also Transactions of the Path. Soc. of London, vol. ix. p. 279.

[1] If the presence of phlebitis is to be denied in all cases where there is an absence of thickening and congestion of the venous walls, it may with equal justice be denied that bronchitis has been present when the bronchial mucous membrane is found after death neither congested nor thickened. But in many cases of fatal bronchitis the mucous membrane itself looks quite or nearly healthy.

which distinguish one case of Pyæmia from another, and cases of Pyæmia from cases of ordinary embolia and thrombosis? Something is doubtless due to the different dates at which patients die. In those persons who succumb early, local processes of disease have had but little opportunity to develop themselves; in those who die late the later stages of suppuration and sloughing have had ample time to become established. But this explanation does not apply to all cases; neither does it apply to the differences observed between cases of embolia and cases of Pyæmia. Here, as elsewhere, those subtle chemical changes (termed vital) so deeply interesting, so little understood, come into play. Why is it that when a cancerous growth has appeared in one part of the body, the whole system speedily becomes influenced, and diseased processes occurring elsewhere assume also the cancerous character? Why is it that when tubercular disease has manifested itself in one organ, the same form of disease ere long becomes developed in other organs? Why is it, again, that amongst all the varieties—shades—of cancer and of tubercle, that one variety which has first shown itself in any case is the pattern upon which the subsequent deposits of the same disease are formed? It would seem that morbid processes, limited in the beginning to one spot, influence the chemistry of the blood, and that of the system generally, and thus produce in the tissues a tendency to repeat, under the influence of exciting causes, those very morbid processes out of which the tendency arose. Again, certain conditions of unhealthiness, dependent on a variety of causes, give a type to the morbid changes accidentally occurring in different parts of the body. Thus, according to the former rule, the occurrence of gangrene at one part tends to the production of gangrene in other parts; the existence of suppuration in one corner of the system tends to render inflammation suppurative elsewhere: thus, according to the latter rule, a certain condition of system (as that accompanying typhus) is apt to favor the occurrence of gangrene, another condition of the system (as that accompanying convalescence from various febrile affections) to favor the formation of local collections of pus; and thus on one or other or both of these principles it doubtless depends that Pyæmia, which is mostly sequential on some localized mortification or suppuration of tissue, presents in the character of its secondary processes not only those differences which distinguish it from embolia and thrombosis, but those differences (not due to relative duration of disease) which distinguish cases of Pyæmia from each other.

(*e*) *Certain Varieties of Pyœmia Considered.*—We may here add a few words in regard to certain, real, or supposed varieties of Pyæmia. Pyæmia is generally an acute and quickly fatal disease: and when this is its character, pyæmic deposits may almost invariably be found. The deposits however are not generally related numerically or otherwise to the severity of the case; and cases are sometimes observed which, judging from the symptoms during life and from other evidences, are truly cases of Pyæmia, yet in which no pyæmic deposits are discovered. It would seem that Pyæmia resembles the exanthematous fevers in this respect, viz., that the blood-poison occasionally produces death ere local lesions have had time to manifest themselves. Sometimes cases of Pyæmia become chronic; the evidence that such cases are pyæmic being furnished chiefly by the occurrence of successive suppurations, in joints,[1] in the cellular tissue, in the eye (it may be), and in other parts. Cases of this kind are sometimes observed after parturition, and such are some of the cases following on enteric and other fevers. The frequent occurrence of pyæmic deposits in the lungs only has suggested the possibility of a local Pyæmia—a Pyæmia in which the poisoned condition of the blood is confined within certain limits, and effects its secondary changes within those limits only. By such a local condition of Pyæmia, confined within the ramifications of the portal system, Dr. George Budd[2] has endeavored to explain the occurrence of hepatic abscesses in cases of dysentery. This explanation of the frequent occurrence of dysentery and abscess of the liver is probably erroneous;[3] and, indeed, our knowledge of the progress of true Pyæmia does not justify us in admitting that the poison of Pyæmia can be limited in the manner suggested.

(*f*) *Origin of Pyœmia in Contagion.*—Pyæmia, which probably only occurs in the classes of cases which have been enumerated—cases presenting the common features of some unhealthy suppurating surface—may arise in them either spontaneously or as the result of some con-

[1] Dr. Wilks, in the Guy's Hospital Reports, remarks that there is a special tendency in Pyæmia to produce inflammation of the joints; that in cases rapidly fatal this tendency has scarcely time to manifest itself; but that in chronic cases (cases, that is to say, in which the blood-poisoning has not been excessive and in which visceral inflammations have been but little pronounced) this special feature of Pyæmia has full time for its development.

[2] Budd, On Diseases of the Liver.

[3] See Frerichs, Clinical Treatise on Diseases of the Liver; Syd. Soc. Translation, vol. ii. p. 113 et seq.: also Trans. of Path. Soc. of London, vol. ix. p. 241.

tagious influence. Cases of spontaneous origin are not infrequent. Many of the cases in which (whether as the result of injury or not) acute suppuration rapidly involves some large portion of bone are cases in which there has been no previous ill-health, no exposure to morbic influences. Many of the cases occurring after compound fractures or after operations arise under personal and surrounding conditions of good health. Other cases are induced by modes of dressing wounds which prevent union by first intention, and promote suppuration and unhealthy discharges. Many of the deaths ascribed to puerperal fever, and occurring sporadically, arise under similar conditions, and, so far as we can see, wholly independently of contagion. There are many cases however, and these are in all respects the most important, where the occurrence of Pyæmia is distinctly due to the agency of some contagium. These cases are particularly met with in surgical practice and in obstetrical practice. As regards surgical practice, it is well known that Pyæmia may be often absent from a hospital ward in which cases of serious accident and cases of operation are in course of treatment, and may continue absent for a considerable period; that after a while a case of Pyæmia, or a case of erysipelas, may be introduced into the ward or may originate within it, and that from that time operation case after operation case, accident case after accident case, may be attacked with pyæmic symptoms. There can be no doubt here that the spread of the disease is due to the presence in the ward of some contagious influence—not of a pyæmic contagium, for the disease cannot be excited in any patient who is not suffering from a wound, nor in any wound probably unless it have become first unhealthy; but of a contagium which excites first unhealthy processes in the wound, and, by means of these unhealthy processes, Pyæmia. This contagium originates not only in cases of Pyæmia, but also in cases of erysipelas, diffuse cellular inflammation, phagedænic processes, and the like, and in cadaveric poisons. There is no doubt that the accumulation of many wounds in a limited space not merely promotes the diffusion of such a poison, but serves even to engender it.[1] It is not improbable that the poison exerts an influence, to some extent at least, through atmospheric diffusion, and that this mode of spread is largely aided by overcrowding and bad ventilation. But Pyæmia arises even more frequently from actual inoculation of a healthy wound, either by the fingers of those who are engaged in attending on the sick, or by the dressings and appliances which are employed upon them. The same remarks, with scarcely any modification, apply to puerperal Pyæmia. Puerperal fever has been known over and over again to be conveyed by the clothes, and more particularly by the hands, of nurses and practitioners; and has been thus carried, not merely from other cases of puerperal fever, but from the poison of erysipelas and other unhealthy inflammations, and from the dead-house. Further, the fact of the heavy mortality from this disease in lying-in hospitals,[1] compared with the mortality from the same disease in patients treated at home, is well established; and the occasional terrible outbreaks of puerperal fever, which blacken the annals of all these institutions bear witness, if not to its spontaneous origin therein, at all events to its virulence of contagion under circumstances favoring its spread.

(*g*) *Conditions modifying the Tendency to Pyæmia.*—It has been asserted that the presence of organic visceral diseases; the debility attending convalescence from various acute maladies; the cachexiæ which result from intemperate habits, from insufficient quantity and quality of food; untoward circumstances attendant occasionally on serious accidents and grave operations and parturition—shock, hemorrhage, nervous depression, and the like; that these, and many other circumstances tending to impair the general health, predispose in various ways to the occurrence of Pyæmia. It is difficult either to prove or refute such assertions; it is difficult to believe that unhealthy conditions of system, however produced, should be without influence in favoring the attacks of Pyæmia and diseases related to it; but, on the other hand, it may be confidently asserted that the vast majority of pyæmic patients have not been suffering from chronic visceral diseases; that but few of those attacked are recovering from acute diseases; and that many, very many, victims of Pyæmia have enjoyed the best of health up to the moment of the accident or the operation of the disease which has exposed them to the danger of Pyæmia; and that even in many cases the wound (if wound there be) has been progressing favorably up to within a few days of the sudden onset of pyæmic symptoms. It may be added as regards the subjects of amputations, that many more in proportion die of Pyæmia of those whose limbs have been amputated for injuries than of those whose limbs have been amputated

[1] See on this subject Report on the Hospitals of the United Kingdom, by Dr. Bristowe and Mr. Holmes, in the 6th Report of the Med. Officer of the Privy Council.

[1] Consult again Dr. Bristowe's and Mr. Holmes's Report, and also Dr. Barnes's Lectures in the Lancet of February, 1865.

for disease—many more, therefore, in this particular case of those whose bodily health has been good until within a short time of the occurrence of Pyæmia, than of those who have been reduced by previously existing disease. Time of year, age, sex, have also been considered among predisposing causes. Season has probably no very important influence. No doubt adults more frequently suffer than children, and men than women; but whatever differences in these respects may be observed are certainly due in an overwhelming degree to the relative frequency of grave accidents in the respective sexes, and at the respective ages. Neither infants[1] nor the aged are exempt. Want of ventilation, and filth, are important predisposing causes; but in order to predispose it is necessary that the want of ventilation should co-exist with undue accumulation of traumatic or puerperal cases, or with the presence of unhealthy inflammatory processes; that the filth should comprise offensive and other animal discharges. Mere dirt, mere deficiency of ventilation, have not, so far as we know, a very obvious relationship with Pyæmia; at least this may be said with certainty in reference to the Pyæmia of puerperal women.

IV. Symptoms of Pyæmia.

1. *Symptoms considered collectively.*

The symptoms which usher in an attack of Pyæmia are generally well marked, unless the condition of the patient, or the nature of the disease under which he is laboring at the time of its supervention, mask the pyæmic symptoms. In some cases the accidental injury, or the operation, or the puerperal process, seem to be going on quite satisfactorily, up to the very moment when Pyæmia manifests itself. In other cases the wound made by accident or operation has taken on for a shorter or longer period some unhealthy action—the discharge from it has become ichorous and offensive, the process of union has become arrested or has retrograded, or sloughing has attacked the part, and constitutional symptoms in sympathy with these local conditions have appeared; or, in the case of the puerperal female, the lochia have become scanty and offensive, or have ceased, the abdomen has perhaps become tender, and high febrile symptoms have shown themselves. But, whatever the previous condition of the patient may have been, whether it have been one of perfect health or not, the first symptom to attract attention is almost without exception a sudden, severe, and prolonged rigor, followed by profuse perspiration. The patient soon recovers from this, and may for a time appear so well that the fear inspired by the first rigor gives way to the hope that it has been a mere accidental phenomenon, of no serious import. But before long, it may be the next day or at some earlier period, the rigor returns with its after sweating stage; and again and again, at varying intervals, the rigors and sweats recur. In the course of a day or two the conjunctivæ and the skin assume a sallow tinge; the patient becomes dull and heavy, or it may be restless, and acquires very much the aspect and manner of a patient suffering from some form of continued fever. In company with the symptoms above described, or in succession to them, others of more or less importance show themselves. The pulse, which at the beginning may have been unchanged, becomes rapid, even exceedingly rapid, weak, and perhaps intermittent; and these evidences of feebleness of the pulse increase as the disease advances. The tongue is often clean at the outset, but soon becomes glazed and fissured or furred, and after a time dry and brown; the lips also become parched, and sordes accumulate probably about the teeth. The appetite disappears; the patient becomes thirsty; and often there is nausea or vomiting. Diarrhœa, attended with offensive stools, occurs very commonly. The respirations become shallow and frequent; cough often supervenes, attended it may be with pains in the chest, with evidences of consolidation or of excess of secretion into the bronchial tubes, and with expectoration. The skin, in the intervals between the rigors and perspirations, and after they have disappeared, is generally hot and dry, and may present sudamina, and even it is said a pustular eruption. The sallowness generally increases, and often before death amounts to well-marked jaundice. Pain and swellings in or around the joints, or in other parts of the cellular tissue, often present themselves, and pus may form in these situations rapidly. As the above symptoms develop themselves and the disease advances, the patient becomes excessively prostrate, his face becomes shrunk and generally pale, his mental functions become more and more disturbed and impaired, slight delirium comes on, and possibly coma, or, but very rarely, convulsions; and at the end of a short period, generally between four and ten days, he dies. During the progress of the pyæmic symptoms the primary seat of the disease (even if it were apparently healthy up to the moment when Pyæmia supervened) assumes an unhealthy character. Sometimes Pyæmia takes a more chronic

[1] A child nine months old was recently admitted into St. Thomas's Hospital with Pyæmia following on acute necrosis.

course ; the symptoms are altogether less pronounced ; the fever attending them resembles hectic fever, and abscesses form in the external parts, as the joints and the cellular tissue ; and the patient sinks, perhaps after a few weeks, of exhaustion; or after a protracted convalescence, during which abscesses cease to form, is restored to health.

2. *Symptoms considered in relation to the various Organs, etc.*

We will consider separately the symptoms referrible to different parts of the system.

(*a*) *Aspect, Skin, &c.*—The aspect of the Pyæmic patient may vary a good deal ; but for the most part it resembles that of one suffering from enteric fever, or typhus. At first it may be healthy-looking or nearly so, but soon it becomes heavy and oppressed. The face is sometimes highly flushed, sometimes extremely pallid, and these conditions often alternate. Towards the close of the disease, pallor generally becomes established ; and the countenance, unless modified in its expression by delirium or other conditions, becomes shrunken and anxious, or settles down into the expressionless dull aspect of the last stages of febrile diseases. The rigors are some of the most marked and prominent symptoms of Pyæmia. Cases are sometimes observed in which they have either been slight and so have escaped notice, or in which they have been wholly wanting. But in the great majority of cases they cannot possibly be overlooked. They vary much in number and frequency. Sometimes they recur at short and irregular intervals ; sometimes they are quotidian, and resemble, and have been mistaken for, attacks of ague ; generally they cease after two or three days, and the subsequent progress of the case is free from them. Their duration varies; sometimes each shivering fit lasts for half-an-hour or so, sometimes for only a few minutes. They are always followed by profuse perspirations. The temperature of the body rises considerably during the rigors.[1] In the intervals, the skin is generally harsh and dry. Sudamina, as might be supposed, not infrequently appear and are sometimes surrounded by a zone of congestion. They may then by a careless observer be mistaken for the spots of typhus or of enteric fever. A pustular eruption has been described as of occasional occurrence, by both Mr. Henry Lee and Dr. Wilks.[2] Sometimes livid discolorations appear; but these correspond for the most part to subcutaneous abscesses, or to tracts of diffuse cellular inflammation ; ecchymoses are rare on the surface of the body. In a large proportion of cases the skin and conjunctivæ become distinctly jaundiced. This is a very important and characteristic symptom. It generally comes on shortly after the first symptoms of Pyæmia have shown themselves, and continues to increase up to the fatal issue. The jaundice, however, rarely if ever becomes intense, and is often so slight that in a bad light or from hastiness of observation it may pass unnoticed.

(*b*) *Organs of Respiration.*—The respiratory movements early become, as in other febrile affections, hurried and shallow. And this condition generally becomes more pronounced as the disease advances. Then the respirations not infrequently amount to forty or fifty in the minute, and are sometimes more numerous than this. After a while they are apt to become moaning or groaning in character. It has been asserted that the odor of the breath is in these cases peculiar and characteristic. The respiratory acts assume the characters just described, independently of all pulmonary disease, and in cases where the lungs are not at all affected, or where the affection is so slight as not to have caused special symptoms. But in the greater number of pyæmic cases the lungs and pleuræ become secondarily affected ; cough comes on, which may or may not be violent ; secretion takes place from the bronchial mucous membrane, or fluids get poured out from the air-cells into the bronchial tubes ; and the cough consequently becomes loose, and attended with expectoration, which may according to circumstances be simply mucous, or purulent, or even distinctly pneumonic. The local phenomena correspond more or less to the morbid processes taking place in the chest. Tracts of dulness may sometimes be recognized on percussion, and sometimes uniform dulness at the base ; but partly from the scattered arrangement of the patches of pulmonary disease, and partly from the absence ordinarily of any large amount of effusion of fluid into the pleura, dulness is often scarcely or not at all recognizable. Pleural friction-sounds again may occasionally be detected ; but owing probably to the limited extent of the false membranes, and to the shallowness of the breath movements, they are not heard so often as might be supposed. The local signs most commonly present are such sounds as are heard in bronchitis, viz., crepitation, often amounting to gurgling and rhonchus. Pleuritic stitches may be complained of.

(*c*) *Organs of Circulation.*—The pulse in Pyæmia is specially remarkable for its feebleness. At the onset of symptoms it

[1] John Simon, Holmes's System of Surgery, vol. i. p. 94.

[2] Op. cit.

may differ little in frequency or in any other respect from its previous healthy condition. But generally it becomes from the very beginning rapid, or if not rapid at least variable, so that the slightest exertion of mind or body raises it twenty or thirty or even forty beats in the minute. The rapidity of the pulse however is generally considerable, and its rapidity tends to increase as the disease advances; so that not infrequently the beats of the pulse amount to 140 or 160, and may even rise to upwards of 200, in the minute. With this increase of rapidity, and with this variableness, the pulse also becomes very small and very compressible; and very often as the patient's general debility increases the pulse becomes irregular and intermittent. There is nothing generally very characteristic in the cardiac phenomena. The action of the organ corresponds with that of the pulse; and the sounds, unless they become masked by other sounds, are healthy though feeble. Pericardial friction may be looked for, but will not always be heard even when pericarditis is present.

(*d*) *Organs of Digestion.*—The organs concerned in digestion always sympathize more or less with the general condition of the system. The tongue in the beginning may be clean; but it soon assumes an unhealthy character. There is nothing uniform however as regards its condition. Sometimes it becomes morbidly red and glazed, and may be fissured; sometimes it becomes thickly furred; but generally its final condition is one of dryness and brownness. Nausea and vomiting are frequent but not invariable symptoms. They are often amongst the first to appear; but they may arise at any time in the progress of the case, and may persist throughout its whole duration. The appetite mostly fails early, and thirst is generally present. Sometimes however the patient retains his appetite for a day or two, and may be persuaded to take even a good deal of stimulus and nourishment throughout the whole course of his illness; and thirst is by no means necessarily excessive, nor is it always complained of. Diarrhœa often shows itself, and the stools are then described as being highly offensive. This complication belongs to no particular period of the disease, and may either be persistent or temporary. It is interesting to bear in mind, in connection with this symptom, the tendency to slight inflammatory changes, and even to pyæmic deposits, manifested in cases of Pyæmia, by the intestinal mucous membrane. The frequent occurrence of jaundice has been already spoken of. That the sallow discoloration in these cases is jaundice is proved by its presence in the conjunctiva as well as the skin; and by the fact that bile-pigment has been recognized in the urine, in the serum of the blood, and in the effusions into serous cavities. The jaundice has no dependence on the formation of pyæmic deposits or abscesses in the liver, and is frequently present indeed when the liver seems to be altogether healthy. Frerichs[1] remarks that "to all appearances the jaundice is here the result of an impaired consumption of bile in the blood, arising from an abnormal condition of the metamorphic processes which go on in that fluid."

Abdominal pain is sometimes complained of; but generally it is local, and the result of inflammatory processes going on in the internal organs (as the liver and spleen) and of circumscribed peritonitis in connection therewith. In puerperal Pyæmia, general peritonitis and tympanites are more apt to occur than in other cases.

(*e*) *Genito-urinary Organs.*—There is little to say in regard to the genito-urinary organs. From the inflammatory processes which so often go on in the kidneys, it is not surprising that the urine is occasionally found albuminous. We might naturally expect to find occasionally in it blood or pus. Unless the uterus be the primary seat of disease, there are rarely, if ever, any important symptoms referrible to that organ.

(*f*) *Organs of Locomotion.*—It is a common thing in Pyæmia, especially in the more chronic cases, to have inflammation and suppuration occurring in joints, and in the cellular tissue, and in connection with bones and other organs. The morbid anatomy of these processes has already been considered. It remains therefore only to add that these superficial abscesses are more common in the chronic than in the acute forms of the disease, that they often attain considerable dimensions, that their origin and progress are often attended with excruciating pain, and that it frequently happens that pain and swelling attack joints and other superficial parts, and subside without leading to any further mischief. Muscular debility is marked from the beginning, but generally soon becomes excessive.

(*g*) *Nervous System.*—The nervous symptoms which appear in connection with Pyæmia are almost identical with those which accompany enteric fever or typhus. The patient is at first perhaps a little heavy and dull and drowsy; but generally he becomes ere long (more especially at night-time) restless and somewhat delirious; yet usually he can easily be recalled to his senses, and to this extent remains conscious up to the time of death. The cerebral symptoms vary, however, in different cases. Sometimes the patient

[1] Frerichs, op. cit. vol. i. 162.

has little or no delirium, and is perfectly rational throughout his illness. Sometimes the delirium becomes violent, and he may become partially or even wholly comatose before death. But generally when coma, and especially when convulsions or paralysis appear, there is some actual disease going on in the brain to account for these symptoms.

The evacuations may or may not be passed unconsciously.

3. *Further Considerations in regard to Pyœmia.*

(*a*) *Time at which Pyœmia arises, and Duration of Disease.*—As regards the time at which Pyæmia appears in relation to the state of system on which it supervenes, nothing very definite can be said. In cases of accident and operation Pyæmia may come on at any moment, from the time when a suppurating surface is first established until the wound is perfectly healed. In cases of carbuncle and of erysipelas Pyæmia probably does not supervene until suppuration has taken place. In cases of acute suppuration connected with bones and acute necrosis, pyæmic symptoms are present sometimes almost from the first. In puerperal cases Pyæmia usually comes on between about the third or fourth and tenth or twelfth day after labor. The disease is generally very rapid in its course; occasionally its duration is limited to three or four days, more commonly it lasts from six to eight days, and it may be for a fortnight. In chronic cases, especially such as recover, the duration of the disease may be much protracted.

(*b*) *Prognosis and Mortality.*—The prognosis of Pyæmia is exceedingly unfavorable. In surgical practice nearly all pyæmic cases die; in midwifery practice a larger proportion probably recover. There is much difficulty, however, in arriving at the exact truth in reference to this point; for, although the symptoms of a typical case of Pyæmia are collectively ample proof of the existence of this disease, there is no one symptom, like the rash of typhus or the exudation of diphtheria, absolutely distinctive, and no one symptom which is invariably present. Hence the diagnosis of the least well-marked cases of Pyæmia is not always to be relied upon; and as cases which are said to have recovered mostly belong to this class, there is generally some, more or less justifiable, room for doubt in regard to the true nature of cases which are recorded to have got well. Still, there can be no reasonable doubt that of cases of Pyæmia coming under the care of the surgeon a certain proportion recover. Now and then cases are met with, having most of the usual symptoms of Pyæmia, and in which it is at least reasonable to suppose that Pyæmia, in a mild form perhaps, exists, which yet escape from the toils in which they seem to be involved. The probability that such cases are truly pyæmic is enhanced by the fact that they are apt to occur in a ward in which Pyæmia is prevalent, and that in some of them abscesses in external parts appear from time to time during their progress, and stamp their real character. But cases of recovery, with or without the formation of external abscesses, are far more common in obstetrical cases; at least it is in lying-in hospitals chiefly that, during the epidemic prevalence of Pyæmia, recoveries not infrequently take place after the supervention of symptoms, which in other cases usher in a rapidly fatal illness. Nevertheless, the disease is one of the most fatal with which practitioners have to deal; its premonitory symptoms are ground for the gravest alarm, and from a fully developed and unmistakable attack recovery is almost quite hopeless.

(*c*) *Diagnosis.*—The diagnosis of Pyæmia is not generally difficult, if the circumstances of the case and the symptoms be all considered. Still, its own symptoms are often so mixed up with those of the disease out of which it arises, or with those of the complications which become developed during its progress, and are often so modified by them, that the Pyæmia may be recognized with difficulty or even wholly overlooked; and further, there are several diseases with the symptoms of which its own have a decided, and even close, affinity, and with which therefore it is apt to be confounded.

It would be impossible to enumerate, still more to discuss, the various conditions which mask the onset, and it may be the progress, of the disease. An example or two must suffice. A patient has had an injury to the skull; after a while rigors come on and perspirations; and, with these, cerebral symptoms. An abscess has probably formed beneath the skull. Now the symptoms here are almost, if not quite, identical with those of commencing Pyæmia. But whether Pyæmia has come on as well is a point that probably cannot be then determined. The further progress of the case may clear up the doubt, but not always. Again, a patient, suffering from a large carbuncle, or from extensive diffuse cellular inflammation, becomes pyæmic; but it is more than probable, if the patient be suffering largely at the time from "constitutional irritation," that the additional "constitutional irritation" due to Pyæmia will be inappreciable. So again in a case of acute deep-seated suppuration connected with some bone (say the femur), and so again in puerperal peritonitis, the

symptoms of the primary disease may be so sudden and so severe, and at the same time in many respects so like those of Pyæmia, that the supervention of the latter disease is very apt to pass unobserved.

The diseases, which above all others Pyæmia resembles, are typhus and enteric fevers, internal acute inflammations (especially of the lungs), urethral and bladder affections in which the kidneys have become involved, and acute rheumatism. The resemblance to fever is proved by the fact that even surgeons of experience occasionally mistake Pyæmia for typhus or enteric fever. The general symptoms and the aspect of the pyæmic patient are indeed almost identical with the general symptoms and aspect belonging to the fevers just named, and the frequent presence of diarrhœa approximates Pyæmia particularly to enteric fever. The liability to error is necessarily much increased when the Pyæmia depends on some deep-seated suppuration, which possibly escapes detection. The differences however are generally well pronounced; the severe rigors and perspirations of Pyæmia have scarcely any counterpart in either form of fever, in which for the most part rigors are scarcely marked, and the skin is dry. Moreover, the eruptions characteristic of typhus and of enteric fever are absent in Pyæmia, and the jaundice which generally attends the latter disease is rarely present in either of the former. The morbid anatomy and the progress of the several diseases will suggest other marks of distinction. In pneumonia not only is the general aspect of the patient like that of a patient suffering from Pyæmia, but the rigors, the profuse perspirations, the jaundice, and even the diarrhœa, may all be present; while in Pyæmia more or less of the lung is mostly involved, and there may even be pneumonic expectoration. It is obvious, therefore, that there might be great difficulty, even impossibility, in distinguishing a case of pneumonia, secondary (say) to a compound fracture, from a case of Pyæmia, supervening on a similar injury. On the other hand a case of Pyæmia, in which the source of pyæmic affection is not obvious, might without much carelessness be taken for a case of pneumonia. Again, when inflammation and suppuration of the kidney-structures come on as a result of vesical inflammation, or of any other disease obstructing the passage of urine, febrile disturbance with delirium follows, and the combined symptoms differ often but little from those of Pyæmia supervening on the same local diseases; and here the difficulty of distinguishing between them is often greatly enhanced by the fact that both forms of disease are not uncommon sequelæ of suppuration occurring about the neck and base of the bladder. Further, acute rheumatism has many features in common with Pyæmia: in both there are profuse perspirations; in both inflammation in connection with bones and joints is common (and it must be recollected that joints often inflame in Pyæmia without suppurating, and that in some cases of Pyæmia pain in the course of a bone, with subperiosteal suppuration, is the first evidence of disease); in both, again, pericardial complication is not infrequent. We have considered somewhat in detail the resemblances between the several diseases above enumerated and Pyæmia, partly because they are really striking, partly because we have known them lead to errors of diagnosis. We have not however dwelt generally on the points which serve to distinguish them, for to discuss these completely or even usefully here would be to forestall needlessly descriptions of diseases which will be fully given elsewhere. There are yet other affections which, under certain circumstances, Pyæmia may simulate: such are delirium tremens, tubercular meningitis, and other inflammatory conditions of the brain or its membranes, ague, &c. It is needless, however, to do more than mention them.

V. Treatment of Pyæmia.

The treatment of Pyæmia is exceedingly unsatisfactory. But, as in so many other instances, although the medical treatment of a case of the disease may be of little avail either to arrest or modify its course, preventive measures are often in the highest degree useful both against its origin and its spread.

1. *Prophylactic Treatment.*[1]

In considering the subject of preventive measures, the simplest plan will probably be to take the case of a private patient, on whom some grave operation—amputation, for example—has been performed. To take precautions against Pyæmia is to take precautions also against those other unhealthy conditions out of which Pyæmia mostly arises. To prevent, so far as may be, their occurrence it is important to maintain both the general health of the patient and the healthy progress of his wound. To this end the patient's strength should be supported by appropriate and adequate nourishment; pain, sleeplessness, and irritability should be treated with opiates and seclusion from needless visitors and intruders; ample ventilation should be secured, and in aid of this curtains and all unnecessary hangings should

[1] See Bristowe and Holmes, loc. cit.

be removed; further, perfect cleanliness should be maintained, and especially all evacuations, all offensive discharges, all dressings, should be removed at the earliest opportunity, and never allowed to accumulate in the patient's room. As regards his wound, that should be lightly dressed and kept cool, and never treated with the abundant dressings which are employed in some foreign hospitals, and which there promote suppuration and, in our belief, erysipelas and Pyæmia. The dressings should be of the simplest kind, and neither these nor sponges and such like things which have been used for the purpose of cleansing the wound should be used a second time. If erysipelas or sloughing or suppuration ensues, the treatment appropriate to these conditions must be employed; in the case of sloughing, charcoal, carbolic acid, and other antiseptics are valuable applications; but above all things it is essential to allow early and very free escape of pus and ichorous fluids. The same remarks apply to the treatment which should be adopted in the case of compound fractures and other injuries.[1] And in regard to cases of deep-seated suppurations, acute necrosis, carbuncle, and diffuse cellular inflammation, there is no doubt that free and early incisions are especially important in preventing the supervention of Pyæmia. In hospitals, or other places where many sick are accumulated, the precautionary measures above insisted on become doubly important, especially those of ventilation and cleanliness. In discussing these two measures we open up the old subject of hospital construction and hygiene—a subject which, even if in many respects appropriate to the present article, is far too extensive to be considered even briefly here. It may however be stated generally, that there should be abundant cubical space to each bed, abundant space between the beds, plenty of ventilation by means of open windows, aided by open fireplaces; that the walls, the floors, the ceilings, should be kept scrupulously clean; that the wards should be periodically emptied; that water-closets should be so arranged as in no degree to infect the ward; that sponges should never be used in the cleansing of wounds—nor anything absorbent in fact which might be used on a second occasion or in the treatment of another case. But the danger of Pyæmia, incidental to wards or places in which many sick are accumulated, is less due to mere accumulation of sick than to undue accumulation of such as are suffering from certain forms of sickness, such namely as are suffering from open wounds, whether arising from accident or disease or design. The presence of what has been termed a "traumatic atmosphere" would seem to have quite a special influence over the development of Pyæmia, and for that reason the creation of a traumatic atmosphere should be as much as possible avoided. This may be effected partly by the means just adverted to—viz., free ventilation, and avoidance of overcrowding—but especially by so distributing traumatic cases as to allow of no undue accumulation of them in any one ward of a hospital, or generally in any one spot.

The presence of erysipelas, diffuse cellular inflammation, phagedæna, or any other unhealthy inflammation, and especially the presence or recent occurrence of Pyæmia in a ward, should be the signal for redoubled vigilance in securing that all sanitary regulations are as far as possible systematically carried out. Then, especially, all danger of inoculating the healthy with the unhealthy secretions of the diseased should be most watchfully guarded against; all dressings, &c., should be destroyed the moment they are done with; and neither nurses, nor dressers, nor surgeons, should pass from attendance on those whose wounds are unhealthy, above all from handling their wounds, to attendance on those whose wounds are healthy, until by proper precautions their persons, and especially their hands, are thoroughly disinfected. It may be added further that no one should at any time come direct from the dissecting room or post-mortem theatre (especially if he have taken part in the dissections going on there) to the treatment of surgical cases, without thorough purification and disinfection. Again, when diseases of the kind above specified have been received into a ward, and especially if they have shown any disposition to spread, it may become necessary either to remove them thence, or, still better, to avoid receiving into that ward for a time all accident cases attended with wounds, and to avoid operations on patients who happen to be therein. But notwithstanding all such precautions, these diseases will sometimes be found to cling as it were to a ward. Under such circumstances it becomes absolutely necessary to empty and dismantle the ward, to purify it thoroughly, and to keep it unoccupied for a shorter or longer period. Sometimes those forms of disease out of which Pyæmia is apt to arise, sometimes Pyæmia itself, may (dependent apparently on atmospheric conditions, or at least on local conditions of insalubrity) prevail over a district. If such be the case, the importance of temporarily suspending the performance of operations in that district becomes obvious.

The various precautions, of which the importance in relation to surgical practice has just been discussed, are if possible of

[1] See Professor Lister's papers on the treatment of compound fractures, &c.

still greater importance in relation to the practice of midwifery. In sporadic midwifery (if the term may be thus used), as in sporadic surgery, precautions are less absolutely needed than where midwifery is concentrated, as it is in a lying-in hospital. But in the former case puerperal Pyæmia has been known over and over again to be conveyed by the nurse or the medical attendant, who have brought infection in their person or in their clothes either from other puerperal cases, or from cases of unhealthy inflammation, or from the dead-house. In the latter case (the case of lying-in hospitals) the presence, so to speak, of a "parturient atmosphere" intensifies the liability to Pyæmia as it does to other infectious puerperal diseases, and leads often to terrible mortality. There is no doubt that no attendant is justified in running the risk of conveying such infection from one patient to another, and that if he have from circumstances become a possible source of danger, he is bound for a while to abstain from midwifery practice. As regards lying-in hospitals, every possible precaution ought to be systematically taken; and on the very first appearance of infection they ought at once to be emptied and purified. But we are very strongly of opinion that such institutions are dangerous institutions, and ought not, unless under exceptional circumstances, to exist; certainly they are not required as schools of midwifery, certainly every poor woman, who has a home however mean in which to be delivered, will be far safer in that home than in a hospital.

It must not be forgotten, however, that Pyæmia is not limited to hospital practice; that even in hospitals it may arise quite independently of hospital influences; and that many cases originate in private quite independently, so far as we know, of external deleterious agencies. Against such cases prophylactic measures are of course out of the question.

2. *Medical Treatment.*

The treatment of a case of Pyæmia resolves itself into the treatment of the original lesion, the treatment of the disease, the treatment of the complications of the disease, the general hygienic treatment of the patient.

(*a*) *Treatment of Primary Lesion.*—As regards the original lesion, it has been shown that in cases where Pyæmia supervenes on wounds (whether from accident or from operation) the wounds have generally already assumed an unhealthy aspect. According to the nature of this unhealthy process must be the local treatment; but especially it would seem important that the parts should be kept clean and cool, that disinfectant applications should be employed, that free incisions to admit of the escape of pent-up pus should be made. In cases where the wound appears to be healthy, there is probably some deep-seated suppuration in progress; and here, though the exact seat of suppuration may be difficult to detect, it is most important that it should be detected, and the pus therein thoroughly evacuated. The same rule applies with equal force to those cases where the primary disease is an abscess involving some deep-seated bone, or tract of cellular tissue. The reasons, on which the adoption of the above plan of treatment is based, are *first*, that by this plan the further entrance of poisonous matters into the blood may possibly be obviated; *second* (and most important), that by its early adoption the entrance of poisonous matters may be wholly averted and a threatened attack of Pyæmia warded off. With the same view it was proposed to cauterize the superficial veins on the proximal side of the diseased part. As regards obstetrical cases, it has been asserted by some that puerperal fever is mostly preceded by an imperfectly contracted condition of the uterus; but both by those who hold this view, and by those who do not, the importance of securing complete contraction of the uterine fibres and consequent closure of the ruptured uterine veins, by the use of ergot, and other means, has been strongly urged. It has been recommended also to cleanse the cavity of the uterus by the injection of disinfectant and astringent fluids.

(*b*) *Curative Treatment.*—Of the curative treatment of Pyæmia we fear little is known. Various plans of treatment have been from time to time adopted, and all probably have by some been supposed to be beneficial, all have by the majority of practitioners been found useless. Some of these plans have been based on the notion of the elimination of the disease; others on the notion of introducing into the system substances capable of battling with the pyæmic poison in the blood itself, and overcoming it there; others have been based on analogy; and others have been wholly empirical. Thus, acting on the assumption that the perspirations and the diarrhœa of Pyæmia are efforts of nature to eliminate some morbid poison from the system, warm baths and diaphoretics have been employed by some; by others the diarrhœa has not only not been restrained, but has been encouraged by laxative and purgative medicines. Thus, too, from the resemblance which the remittent rigors and perspiration of Pyæmia sometimes bear to the more regular attacks of the same kind which characterize ague, it has been imagined that quinine and arsenic, which are certainly remedial

in the case of ague, might be remedial in the case of Pyæmia. Thus, again, guided we presume by the acknowledged fact that ample ventilation is one of the most important preventives against Pyæmia, some have looked on fresh air as absolutely curative, and have exposed their pyæmic patients to all the winds of heaven. And thus, others regarding the disease as one of putridity, have treated it with various forms of antiseptic agents. It would be useless to argue seriatim against the above and other modes of treatment; it would be unwise to oppose *à priori* the trial of any as yet untried modes of treatment; suffice it to say that, so far as we know, the mortality of Pyæmia is just as high now as it ever has been, and the antidote to it remains to be discovered.

There is, however, one mode of treatment, suggested within the last few years by Professor Polli,[1] of Milan, which, from the scientific character of the investigations which led to its proposal, and the manner in which the proposal has been brought under the notice of the medical profession, deserves a respectful mention. It will be necessary to go a little into the history of Professor Polli's investigations, and to trace shortly the steps which led him to his final conclusions. He assumed with most other physicians that septic poisons introduced into the blood produce their injurious effects through acting on the blood as a kind of ferment; and he assumed, as again others have assumed, that if any substance could be introduced into the blood, which, while not acting injuriously either on that fluid or on the system generally, would arrest this process of fermentation, the exhibition of such substance in cases of septic poisoning would not improbably be curative.

Having long studied the effects of sulphurous acid, he had ascertained that it is not merely a powerful antiseptic, but that it equally prevents the vinous fermentation, and those other fermentations by which starch is converted into glucose, by which the pancreatic juice acts on fatty substances, and emulsine on amygdaline; and he came to the conclusion that in sulphurous acid we possess a substance capable of arresting every form of catalytic action.

But sulphurous acid cannot with impunity be introduced into the animal economy. Hence Professor Polli sought for other agents which, while having the virtues of sulphurous acid, should be free from its disadvantages; and he found such agents in the compounds of sulphurous acid with soda, potash, magnesia, and lime. He found that these sulphites, equally with sulphurous acid, prevent all forms of fermentation; he found that they may be given safely in large doses over a considerable period of time; and he further found that when taken into the stomach they become absorbed, diffused throughout the system and eliminated without undergoing any chemical change, or at most only a very partial chemical change.

He assumed therefore that that power of preventing putrefaction and fermentation which they exercise outside the body they would exercise probably equally well within the body. Any further experiments made by him upon the lower animals, by the introduction of putrid matters into their blood, and by putting them at the same time under the influence of the sulphites, led him to believe that he had in these agents discovered valuable remedies for the various forms of septicæmia. It would seem that large doses of these sulphites (from thirty to sixty grains three or four times daily) may be given with impunity. We are not aware whether this plan of treatment has been so largely tested as the promises it seems to hold out might justify. We know of one case of supposed Pyæmia in which it was believed to have wrought a cure, but we know that in the practice of Mr. Simon, at St. Thomas's Hospital, it has entirely failed.

[The tentative use of salicylic acid, on the same principle, is justified by analogy. No sufficient experience has, however, as yet, determined its positive value in Pyæmia.—H.]

(*c*) *Treatment of Symptoms and Complications.*—When a case of Pyæmia is under treatment, it always becomes a question of treating, in addition to the general disease, certain symptoms as they arise, and, it may be, certain of the secondary lesions. On this head, again, we fear there is little satisfactory to be said. Some have recommended, on theoretical grounds, that diarrhœa should be encouraged. There seem no valid grounds for this course; and certainly if the diarrhœa became excessive, we should recommend that it be restrained, either by remedies administered by the mouth or by opiate enemata or suppositories. Again, the pulmonary symptoms may become sometimes exceedingly distressing; and then, although probably we have no means of either checking or curing the morbid processes going on in the lungs, we may by opiates or other sedative medicine, judiciously administered, render the symptoms more endurable; or it may be that expectorants, especially ammonia, may be of benefit. Of course if abscesses form in superficial parts, they should be early punctured. As regards other symptoms, and other complications, we have really nothing to say. Many of them will need

[1] See Dublin Journal of Medical Science, vol. xxxiii. p. 367, and vol. xxxvi. p. 470.

no special treatment at all; and generally where special treatment seems to be required, the medical man must be guided by his general knowledge of his profession, and treat them as he would treat such complications arising in the course of fevers and other allied disorders; bearing in mind, however, that where he cannot cure, it is better as a rule to aim at soothing and quieting, than (in the hope of achieving some insignificant advantage over the outposts, so to speak, of his patient's disease) to adopt a fidgeting line of treatment, and so render his few remaining hours miserable.

(*d*) *Hygienic and Dietetic Treatment.*—We come lastly to the general management of pyæmic cases, that is to say, their management as regards diet, stimulus, and hygienic observances. It need scarcely perhaps be pointed out that observance of cleanliness and ventilation, which we insisted on as an important prophylactic measure, should be equally persisted in during the whole course of treatment of a case of Pyæmia; that, further, the patient should never be oppressed unnecessarily with accumulation of bed-clothes—that he should, in fact, be kept cool—and that his comforts should be carefully considered. Pyæmic patients, as has been shown, become at an early period excessively feeble; and in most cases excessive prostration is the most prominent among the symptoms which usher in death. No doubt this debility is functional rather than the direct result of the waste and degeneration of tissue; the consequence and the indication of blood-poisoning, rather than of the want of either stimulus or food; and theoretically, therefore, is to be counteracted by antidotal treatment rather than by nutriment. Whether we possess any mode of treatment that can be regarded as really antidotal we have shown to be in the highest degree problematical; but we have in these cases excessive prostration to deal with, and we must deal with it as best we can. To this end, it is manifestly our duty to administer both food and stimulus, and to administer them as largely as the condition of the patient will admit. The patient's appetite is generally quite annulled; and often loathing of food and vomiting are present. These conditions render it, of course, often exceedingly difficult, and sometimes impossible, to carry out the objects we have in view; and they show the importance of selecting for administration those articles which are least liable to offend the stomach, and of administering these in small and if possible frequently repeated doses, rather than rarely and in large quantities at a time. It is not easy to lay down any rule with regard either to the nature or the amount of food and stimulus to be given. These points must be determined in each case according to its requirements. But it is important to give whatever is to be given systematically. As regards food, that which is in the form of fluid is generally most suitable, such as animal broths, eggs beaten up, milk, gruel, arrowroot, and the like. As regards stimulus, perhaps, considering the irritability of the stomach, brandy, sherry, madeira, diluted according to the patient's taste, are the most generally serviceable. But lighter wines will often be found grateful. We protest against that excessive exhibition (that "pouring in" as it is appropriately termed) of stimulus which it has lately been the fashion to practise.

We need perhaps scarcely add that when pyæmic cases become protracted, and especially when they show signs of convalescence, and during the progress of convalescence, dietetic treatment becomes of paramount importance, and tonics form important aids to that treatment.

MALARIAL FEVERS.

By W. C. Maclean, M.D.

Before entering on the description of the remarkable fevers which are to form the subject of the following article, it is necessary to premise a few observations on the peculiar poison which produces them. This poison, which gives a distinctive name to fevers with periodical returns, is everywhere recognized by the term Malaria. "When a climate is called unhealthy, in many cases it is simply meant that it is malarious." (Parkes.)

In this article it is intended only briefly to summarize the few facts relating to this poison which have been tolerably well ascertained.

No chemist has yet been able to demon-

strate the existence of malaria. We assume its existence from certain observed effects on the organism, just as we do in the case of other poisons which produce certain specific diseases. Malaria is believed to be the produce of organic decomposition in soils, whatever may happen to be their mineral composition; water is indispensable to the process, and a high temperature, although not absolutely necessary, greatly aids it.

It is generated in greatest abundance in marshes, which contain a high percentage of organic matter; hence the name by which it is familiarly known, viz., *marsh miasm.*

It is often found in sandy soils and arid-looking plains devoid of vegetation; but in all such cases the soil will be found to contain a considerable proportion of organic matter, and water will be found not far from the surface, either in the shape of subterraneous streams, or detained by a bed of clay below the sand, preventing its free passage and keeping up evaporation.

Malaria is also generated in hard rocks, such as granite and trap, in a disintegrating state. A notable example is the island of Hong Kong, which consists entirely of weathered and decaying granite. In such soils, so long as they are undisturbed, the existence of malaria may not be suspected. In the case of Hong Kong, for example, it was not until extensive excavations were made into the disintegrating granite for building purposes, that violent and fatal remittent fevers appeared.

Dr. Parkes mentions that the soil of Hong Kong contains less than two per cent. of organic matter, but quotes Friedel to the effect that disintegrated granite, which is highly absorbent of water, becomes often permeated by a fungus, and suggests the possible relation between the development of this fungus and the production of malaria.

The air of marshes known for ages as malarious has been examined by chemists. Watery vapor and carbonic acid are always found in excess; and, under certain conditions, sulphuretted hydrogen. "Carburetted hydrogen is often present, and occasionally free hydrogen and ammonia, and, it is said, phosphoretted hydrogen." (Parkes.) Besides the above, "various vegetable matters and animals, floating in the air, are arrested when the air of marshes is drawn through water, or sulphuric acid, and débris of plants, infusoria, insects, and even, it is said, small crustaceæ, are found." (Parkes.)

Malaria acts with the greatest intensity on the human system in situations which are low and moist, abounding in vegetation undergoing decomposition, *e. g.*, in jungly districts during or immediately after the rainy season, at the bases of great mountain ranges, and in those belts of country in India termed *terrais*, formed by the débris of mountains rich in organic matter, which retain a large quantity of water and are covered with jungle.

It is capable of drifting along plains to a considerable distance from its source, particularly in the direction of the prevailing wind. It ascends mountains, especially when favored by ravines and currents of air. The height to which it can ascend from its source is still matter of dispute. Dr. Parkes thinks that 500 feet is the limit in temperate climates, and from 1000 to 1500 in tropical countries; while others maintain that in the latter we are not safe from its influence until a height of 5000 feet has been reached. It is probable that when men suffer from malaria at elevations above 2000 feet, it is either derived from unsuspected local sources, or it is carried up ravines by currents of heated air from the unhealthy plains.

It is a common belief in India that water is capable of absorbing malaria, and that periodic fevers, dysentery, and even cholera, are produced by drinking water so charged.

This absorbing power of water, and especially salt-water, has often a beneficial effect, when a sufficient breadth of it, not less than from three-quarters of a mile to a mile, is interposed between our habitations and the source of the poison, which is either absorbed or rendered innocuous in its transit.

Belts of trees interposed in like manner exercise a protective influence. [Living trees and shrubbery appear to exercise an influence in preventing the development of the malarial cause. The Great Dismal Swamp, on the southern border of the State of Virginia, being covered chiefly by growing cypress trees, is not malarious. The Eucalyptus globulus, native to Australia, has the reputation of special usefulness in this way. Probably it and the Southern Pine do good mainly by assisting, by their vigorous growth, the drainage of the soil.—H.]

Malaria disappears before cultivation and subsoil drainage, with free exposure of the soil to the action of the air and of living vegetation. When, however, the cultivating hand of man is withdrawn and the old conditions reappear, malaria again resumes its sway. [Nothing is more evident in the history of malaria than its general disappearance before the extension of closely built cities and towns. Suburbs, in which intermittent occurs, gradually become, in that respect, more healthy as lots are drained and occupied by habitations. Exception to this may, it is true, exist, when houses are built upon *made ground*, and the drainage con-

tinues to be very imperfect for a long time. This kind of exception has been observed to a considerable extent of late years in the city of New York.—H.]

It is the cause of intermittent and remittent fevers, and their sequels; it "underlies" the cause of dysentery and cholera; and by its depraving influence on the constitution it often silently undermines the health without the manifestation of any febrile phenomena. Major-General Cotton, in his evidence before the Indian Sanitary Commission, very truly observes "that there are many ailments which the natives of India call fever, but which a medical man does not, which are the effects of malaria."

When a person has for some time suffered from the toxic influence of miasm, a curious impress of periodicity is sure to show itself in all his subsequent ailments, whatever be their nature; and I believe, from extensive observation, that this impress of periodicity is never eradicated.

Casorati, a late Italian physician of eminence, in his "Treatise on Intermittent Fevers," a posthumous work recently published, has given it as his opinion "that miasm is the cause of an extremely small number of intermittent fevers." He says "that there are pernicious intermittents, the origin of which is simply rheumatic." Casorati further dwells on the fact that, within the sphere of his observation, "nothing is more common than to see pregnant women the subjects of tertian fever, under which they frequently abort;" and he gives numerous examples of diseases, such as menorrhagia, cephalalgia, &c. &c., all presenting an intermitting type, due, as Casorati supposes, not to the toxic effect of miasm, but to other causes, such as "humidity," "cold," and the like.

The truth is, that Casorati's sphere of clinical observation was in a malarial region: the stamp of periodicity was therefore deeply impressed on a great number of the diseases that came under his care. The proof of this is not far to seek; for, by his own showing, no treatment was effective until quinine was given. We do not find, where there is no miasm to complicate the case, that "acute rheumatism" or "menorrhagia" or "cephalalgia" derive benefit from anti-periodic remedies, still less that such are indispensable to all treatment.

No sooner is the blood poisoned by malaria than it acts on the stomach and alimentary canal. In all agues, particularly of a severe type, there is from the first great disturbance of the stomach, and in severe remittents this is often the most prominent and urgent symptom. Casorati goes so far as to state that morbid appearances in the stomach constitute by far the most constant post-mortem appearance found in fatal cases of intermittent fevers. In the article Dysentery I have given it as my opinion that miasm is also the cause of that disease. In the present state of knowledge, it is not possible to explain why malaria should in one case cause dysentery, and act with intensity on the glandular structures and mucous membrane of the great intestine, and in another excite an intermittent or remittent fever, with signs of extreme irritation of the stomach and duodenum, going on often to structural changes in those parts. Chemistry may one day reveal to us some difference, at present inappreciable, in the constitution of miasmata to account for the affinities displayed in the different cases.[1]

The structural changes of a more secondary kind induced by malaria are, enlargement of the spleen and liver, to be more particularly described further on.

INTERMITTENT FEVER.

DEFINITION.—A specific paroxysmal fever, the febrile phenomena observing a regular succession, characterized by a cold, a hot, and a sweating stage, followed by a period of complete apyrexia, varying in duration according to the type of the fever.

SYNONYMS.—Periodic Fever, Ague, Paludal Fever.

HISTORY AND MODES OF COMMENCEMENT.—When the human system has been exposed to the influence of malaria, sooner or later, according to circumstances, symptoms of disturbance appear: as already remarked, many have their constitutions silently undermined without suffering from periodical fever at all. It seems probable that in such cases the poison is not presented to the system in a very concentrated form; the blood is so gradually changed that the organs become as it were tolerant of its presence, to such an extent at least that febrile phenomena are not excited at regular intervals for the apparent purpose of expelling it from the blood. On the other hand, people in perfect health may be exposed to the action of malaria in such a noxious form as to be at once completely overwhelmed by it. The late Lieutenant-General Sir Mark Cubbon informed me that many years ago, when on a journey to the Neilgherry

[1] I am informed by Dr. E. Goodeve, late professor of medicine in the Calcutta Medical College, that the cases of dysentery which gave him most anxiety in Calcutta were those in which he was at first uncertain whether the disease was to be remittent fever or dysentery.

Hills, he was compelled to pass a night at the foot of the Segoor Pass, then an uncleared and unhealthy spot. A party of three German missionaries were also detained at the same place, and slept in the same house. These gentlemen were fresh from Europe, and in high health. On the following morning they pursued their journey, and were soon "above fever range." In less than twenty-four hours three out of the four of the party were stricken with fever, and two of them died in a few days.

Most frequently the person who has been exposed to malaria suffers for some days from premonitory symptoms. The toxic influence is evidenced by some degree of nausea and loss of appetite, with muscular pains in the back and lower limbs, with usually a slight feeling of chilliness, soon passing into trifling heat of skin, scarcely marked enough to excite attention. This may recur for several days before a regular paroxysm of ague sets in. Or, without such prolonged warnings, after an hour or two merely of the above symptoms, the patient may be seized with the cold stage, in the manner to be presently described. In such cases there is almost always a considerable amount of urinary irritation, the patient having frequent calls to pass pale-colored, acid, and irritating urine. When this symptom is urgent, a severe paroxysm may usually be expected.

Then follow in succession the three stages which characterize this fever, viz., the cold, the hot, and the sweating stages, at the end of which there is a period of apyrexia, termed the *intermission*, the duration of which varies with the type of the fever. The time occupied by a paroxysm and the period of apyrexia that follows is somewhat incorrectly termed the *interval*. The types of the fever are named according to the length of the interval. These are the *quotidian*, which recurs daily, having an *interval*, in the above-mentioned acceptation of the term, of twenty-four hours; the *tertian*, with a paroxysm every other day, and an interval of forty-eight hours; the *quartan*, every third day, and an interval of seventy-two hours.

These are what have been termed the *regular* types of Intermittent Fever. Physicians recognize others which have been called *irregular:* such, for example, as the *double tertian*, which is said to differ from a quotidian only in having on alternate days fits corresponding in severity, character, and duration; the *triple tertian*, which has two fits on one day, and one the next: the *duplicated tertian*, which has two paroxysms on alternate days, with a fever-free day; the *double quartan*, which has a fit on one day, a mild one the next, the third being a fever-free day; and so on. What is it that determines whether the type of the attack shall be a quotidian, tertian, or quartan? It is probable that this is governed simply by the extent to which the blood has been charged by malaria. The presence of a quotidian seems to indicate a high degree of saturation, requiring a more frequently renewed effort of nature for at least its partial elimination than either a tertian or a quartan.

The tertian is said by many authors to be the primary type of fever, and to be the most common of all. This is certainly not the case in India, where without doubt the quotidian is the most common, and the quartan the rarest of all intermittents.

According to my experience in India and China, a first attack of ague invariably takes the quotidian form.

The duration of a paroxysm of Intermittent Fever varies with the type. It is longest in the quotidian, which lasts from eight to ten or even twelve hours; the tertian lasts from six to eight; and the quartan from four to six hours.

The paroxysms do not always occur exactly at the same hour of the day. In the early days of an attack, when the disease, not having been interfered with by treatment, is "waxing," the cold stage will almost certainly appear an hour or two earlier on the days of the second and third paroxysms than on the first. On the other hand, when the system has been affected by antiperiodics, or the poison has by successive paroxysms been to some extent eliminated, and the disease is "waning," the time of attack will be postponed for an hour or two. I have observed this in my own person, and have noted it in others as of almost invariable occurrence.

The length of time to which intermittents left untreated will run on will depend much on climate, locality, and season, and the extent to which the system has been charged by malaria. Mild quotidians often terminate after ten or twelve paroxysms; quartans last longer, and may run on for months.

When once the system has gone through the phenomena of an attack of ague, paroxysms are liable to recur quite irrespective of fresh exposure to malaria; an error in diet, exposure to wet or cold, any cause that disturbs the balance of the circulation, may bring on an attack, and an impress of periodicity is apt to be given to any ailment from which the person may subsequently suffer. This disposition lasts always for years, sometimes for life.

The type of the disease does not always remain the same; a quotidian may pass into a tertian or a quartan; and an intermittent may, under certain conditions, assume the more grave form of a remittent.

I have repeatedly known sportsmen in India, and officers of the Forest Conservancy department, whose amusements or occupations exposed them only to mild intermittents, so long as they remained in comparatively cool and elevated regions, suffer from severe remittent fever on descending to the plains, a change in many instances apparently due merely to the influence of high temperature, for I have several times observed it when there was no reason to suppose that the sufferers had been exposed anew to the influence of malaria.

The direct mortality from Intermittent Fevers in India is small. Even in Bengal, out of a strength of 344,152, with 111,687 admissions, the percentage of deaths to strength is 0·24, and the percentage of deaths to admissions is 0·76.

But although it is undoubtedly a rare thing to see a person die in the course of an uncomplicated Intermittent Fever, it is nevertheless, indirectly, an exceedingly destructive disease; the fatal results must however be looked for under other heads in the death returns of malarial regions. It is undoubtedly true, as remarked by the Indian Sanitary Commissioners, "that diseases of important organs, the consequences of malarial fevers, occasion much of the subsequent sickness, mortality, and invaliding among British troops serving in India."

Judging from the writings of Casorati and others, Intermittent Fevers seem to be more severe and more fatal in Italy than in India. How far the system of treatment in that country influences the mortality I am not prepared to say. In many cases of simple, and apparently in all complicated agues, Casorati not only highly extols blood-letting, but reprobates its neglect as culpable and dangerous in a high degree. For example, in a case of ague with orchitis, Casorati draws a pound of blood, and does not hesitate to repeat the proceeding; and in all cases where gastric irritation, headache of extraordinary severity, and such like symptoms are present, his treatment is "decidedly antiphlogistic," comprising repeated blood-letting, both general and local, with perfect abstinence from food for many days. In Italy malarial fevers that in the opinion of physicians there demand such treatment are termed "pernicious." In India practitioners of the present day would be disposed to transfer the term from the fever to the treatment, for it is certain, as demonstrated by the experience of the past, that in any of the types of the malarial fevers of tropical regions antiphlogistic treatment such as that urged by the modern Italian school, is not only unnecessary, but most dangerous.

Causes.—Whatever tends to depress the physical or mental powers, and so render the system more liable to the influence of malaria, is a *predisposing cause*.

The *exciting cause* is undoubtedly an exhalation from the soil, given off under the conditions already described, to which the name of malaria is provisionally applied. That specific agues ever arise from other causes of malaria I do not believe, and am satisfied that where they are attributed to "cold," to "moisture," to "irritation," to the "influence of the mind," and such like supposed causes, it will in every such instance be found that the sufferer has at some former period been in a malarial locality, or that this poison, arising, it may be, from an unexpected source, has been in operation just before the attack.

In a most especial manner I desire to express my entire dissent from the doctrine that specific agues are the result of suppressed cutaneous secretions, under sudden impressions of cold; if it were so, we should have agues constantly occurring in temperate climates during the summer months, in places where no miasm exists, which is contrary to all experience. Where this poison has been introduced into the system, suppressed cutaneous secretion under sudden impressions of cold may call the poison sooner into action, perhaps by concentrating it more in the gastro-duodenal mucous membrane during the state of congestion that follows the impression of cold; but to produce a true specific ague I believe the presence of malaria in the blood to be necessary.

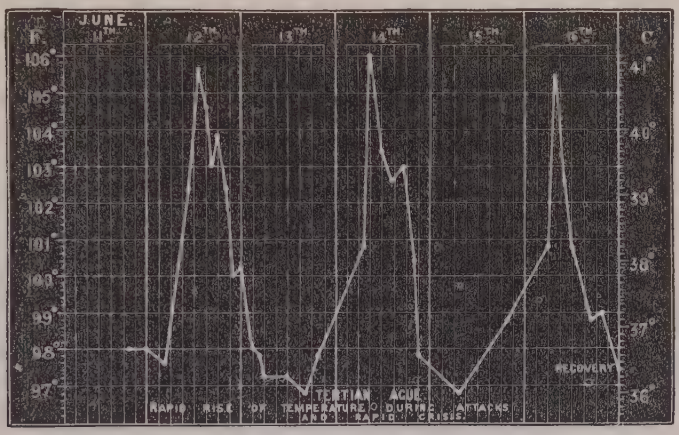
To the question, why the miasmatic poison, unlike that of rheumatism, or variola, or typhus, should produce a periodical, and not a continued fever, no satisfactory answer has yet been given; notwithstanding all the ingenious speculations of scientific inquirers, it remains unexplained.

Symptoms.—After certain *premonitory* symptoms—of which the most prominent are nausea, languor, lassitude, muscular pains in the back and legs—the *cold stage* commences. In this the patient becomes chilly, first in the extremities, then in the back, and soon passes into a most unpleasant sensation of coldness all over the body. The skin shrivels, the nails become blue, and rigors, more or less severe, rapidly succeed each other. In the hottest climate the patient demands to have bed-clothes heaped upon him, although he derives little additional warmth from them. With the above there is often urinary irritation, the patient passing at short intervals considerable quantities of highly acid urine almost devoid of pigment.

This symptom, although not commonly mentioned by authors, I have very often experienced in my own person, and have frequently noticed it in others; when present in a high degree, it adds much to the patient's distress.

The sensation of cold of which the sufferer complains is merely a subjective symptom. (Parkes.) Incredulous as the shivering patient may be, it is certain that the temperature of his blood, even before the rigors begin, is above the normal standard, a fact which is at once demonstrated by placing a thermometer in his axilla, which rises rapidly until it indicates a temperature of 105° to 106·3° Fahr. (Parkes, Ringer, Wunderlich.)

[Fig. 14.



Temperature in tertian ague.]

This sudden rise in temperature is common to all the types of malarial fever. Headache is sometimes complained of, but not always; the mind is inert, and occasionally the patient is drowsy. The duration of the cold stage is variable; it may last from half an hour to two hours and a half, and in rare cases even three or four hours. When the paroxysms have been often repeated, and the poison has been, to a great extent, eliminated, the cold stage shortens, until at last the patient is only conscious of a passing chill.

Hot stage.—Flushes of heat at first alternate with slight rigors. By and by a grateful feeling of warmth steals over the body; the bed-clothes are thrown off; the increase of temperature is now apparent to the patient and his attendants without the aid of a thermometer. The pulse becomes full and frequent; the respiration, although still hurried, becomes more regular. When the hot stage is fully developed, a temperature of 107° or 108° is often noted. The agreeable sensations that accompanied the first feelings of warmth pass away; nausea, and even vomiting, often distress the patient; headache and thirst are complained of; and the patient tosses uneasily in a burning fever.

Physicians in the malarial parts of the Southern States of America look anxiously for the development of what they call "a good hot stage," regarding powerful reaction as conducive to the patient's safety; whereas a quick and feeble pulse, with rapid thoracic respiration and low temperature, are looked on as dangerous symptoms, as indeed they are.

The duration of the hot stage is usually about two hours; in severe cases it may last four or five, and it has known to be prolonged through ten or twelve hours.

Sweating stage.—Perspiration appears first on the brow and face, and gradually spreads over the entire surface, until the patient sweats copiously at every pore. The pulse falls in frequency and strength; the respiration becomes more natural; the temperature rapidly falls to the normal standard; headache first abates, and then passes away.

Captain Burton, the renowned African traveller, writing of the mild Intermittent or Seasoning Fever of East Africa, declares "that there is nothing unpleasant in these attacks. The excitement of the nerves is like the intoxication produced by a plentiful supply of green tea; the brain becomes uncommonly active, peopled with a host of visions; and the imagination is raised almost to Parnassus." This mental excitement I have experienced, and the observant traveller is right when he adds, "the patient pays for it when the fit passes off. These agreeable sensations do not recur with the subsequent paroxysms."

During the intermission the patient is commonly said to be "well," but this is only true in a limited sense; and if the paroxysms be allowed to go unchecked, the sufferer, even during the intermission, soon becomes incapable of much exertion of mind or body.

Condition of the Urine.—As already mentioned, the urine is increased during both the cold and hot stages, and apparently, from the presence of a large quantity of free acid, is sometimes very irritating. Convalescence is ushered in by a remarkable diminution in its quantity; it now becomes scanty, alkaline, or neutral, and of a deep orange color. Intelligent patients soon learn to note this, and intimate to their physicians the occurrence of what they deem a critical discharge, by informing them that no more quinine is required. In the hottest weather, during the active stages of Intermittent Fever, urine always retains its acid reaction for several days. When the fever intermits, the urine then rapidly undergoes decomposition, and changes from acid to alkaline. (Jones.)

A person with ague, not actually suffering from a fit, secretes less urea than a person in health. The moment a fit commences, the urea suddenly increases, although every known cause of increase, as food and exercise, be avoided.

The increase lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermittence. The amount then falls below the healthy average. (Parkes.)

There is a very close connection between the temperature and the amount of urea. (Ringer.) The amount of urea corresponding to a degree of Fahrenheit is greater at a high than a low temperature.

The pigment is lessened in amount. (Jones.) Uric acid is greatly increased during the fit. (Parkes.) The chloride of sodium, according to Professor Ringer, is greatly increased during the cold and hot stages. Albumen is found in an uncertain proportion of cases during the fit, with blood and renal cylinders. I can confirm, from personal observations, Dr. Parkes's remark that chronic Bright's disease is a consequence of ague. Many "old Indians," who have suffered from malarial fevers, die of this disease.

The Blood is changed from the beginning of the attack, and, probably, for some time before. The red globules and fibrin are diminished, the coagulum is larger and more flabby than that of healthy blood, much darker in color—in extreme cases approaching to black—and on exposure to air, instead of the usual bright red, it only assumes a cherry-red color. (Jones.) Its serum is dark and muddy, and it has sometimes an oily appearance.

The skin, after a time, assumes a dirty pale yellowish hue, a change which is often permanent, and which depends, not on bilious discoloration, but on some of the blood-changes above described.

Sufferers from Intermittent Fever are usually depressed in spirits, and are incapable of much exertion of mind and body; their appetites and digestions are bad, and they are prone to diarrhœa from slight causes.

When this anæmic condition is developed, a peculiar cardiac murmur is commonly present, which is prolonged into the great vessels. This is an "anæmic bruit," due apparently to the watery condition of the blood.

If the spleen is much enlarged, the heart is apt to be displaced upwards, and thus to mislead the unwary into a diagnosis of heart disease, when that organ is sound. (Morehead.) The heat-generating power of all victims to malaria is impaired: hence they suffer from atmospheric changes, of which healthy men take no note.

Diagnosis.—Remittent Fever is the only disease which appears to me likely to be confounded with an ague. The regularity of the phenomena, the existence of a distinct period of complete apyrexia, will suffice to determine the diagnosis, and in doubtful cases the thermometer will settle the point, "for all the types of ague present this characteristic peculiarity of a sudden and speedy rise of the temperature up to 105° or 106° Fahr. and of an equally rapid and complete defervescence, till the period of another paroxysm comes about." (Aitken.)

Morbid Anatomy.—Death in an uncomplicated intermittent is so uncommon that few opportunities for post-mortem examination are afforded.

We have seen that one of the earliest indications of disturbed function after the action of malaria is given by the stomach; in the mildest agues this disturbance is present, and in severe remittents intense nausea and urgent vomiting are among the most prominent symptoms. Casorati, who has had many opportunities of dissecting the bodies of those who have died from the "pernicious" agues of Italy, observed "that sufficiently well-characterized morbid appearances in the stomach constitute by far the most constant post-mortem appearance of all those observed." (*Vide* "British and Foreign Med. Chir. Rev." July, 1864.)

Hyperæmia of the stomach and duodenum, then, is one of the most common of the appearances found *post mortem* in intermittents. In some of the cases examined after death at Walcheren, circular ulcers, according to Sir Gilbert Blane,

were found in addition to the hyperæmia above described.

The liver and spleen also suffer, the latter more frequently than the former. In recent cases the spleen is generally found so softened in its texture as to break up under examination; occasionally it is reduced to a dark-colored bloody pulp, enveloped by its capsule. In more chronic cases the organ is found to be indurated, and often so enormously enlarged as to extend downwards into the pelvis. That the spleen acts as a diverticulum in the cold stage of ague there is no doubt; by percussion we can demonstrate that it enlarges with every fit, and contracts again when the paroxysm comes to an end. In time the elasticity of its structure is impaired, and some degree of permanent enlargement results. But there is another cause in operation. Virchow and others have shown that the spleen enlarges not only in intermittent and typhoid fevers, but also in most other morbid processes resulting from the presence of noxious matters in the blood. Irritation of the gland ensues, and the result is increased cell-formation in its structure.

The liver is found in recent cases in various states of congestion, often soft in texture, and of a dark purple or black color. In more chronic cases it is enlarged, the malarial poison acting as a source of irritation, leading, as in the spleen, to increased cell-formation. When the bodies of men who have served long in malarial regions are examined, one of the most common appearances is a deposition of black pigment in the spleen, liver, and kidneys.

Prognosis.—I have never seen a person die from uncomplicated Intermittent Fever.

The prognosis in such cases, under rational management, is favorable.

The danger to life is from the malarial cachexia, and the organic changes to which it gives rise. When the disease is about to yield, not only are the paroxysms less severe, but the time of accession is postponed for some hours. The appearance of a copious deposit in the urine, and an herpetic eruption about the lips, are also favorable signs: so also are the disappearance of præcordial distress, anorexia, and nausea at the commencement of the paroxysm.

[In several localities in the Southern United States, especially in the rice-growing region of South Carolina, and some places near the lower Mississippi, a type of Intermittent Fever prevails, which, in the absence of specific treatment, is often fatal. This is best designated as Pernicious (also called congestive) Fever.

In it all the main symptoms of ordinary Intermittent are intensified; at least in the cold stage. This partakes of the character of the collapse of cholera; except in the absence of the colorless watery evacuations. The skin is cold, pale, or livid; the countenance haggard; thirst is intense; the pulse is small, weak, often rapid or irregular; respiration is oppressed; vomiting is common; the bowels mostly are loose, with discharges often like bloody water.

Sometimes cerebral symptoms predominate; delirium being present, passing into stupor, with stertorous respiration. Tetanic rigidity occasionally exists during the paroxysm.

After an imperfect reaction, lasting from eight to twelve hours or more, another attack comes on, unless prevented by treatment. A third paroxysm is almost always fatal.

The *diagnosis* of such cases is usually not difficult in the localities in which Pernicious Fever is common. In the Northern United States, where examples of the same kind now and then are seen, they may present more difficulty. Especially the variety in which a tendency to stupor exists, may not always be easy to discriminate from apoplexy. The previous history of the patient must then be closely inquired into. The coldness of the skin, and generally the character of the pulse, will aid in drawing this distinction.

In the *morbid anatomy* of Pernicious Fever, the attention of Southern practitioners was long ago called to the frequency of congestion of the brain, liver, lungs, and other organs; giving rise to the title "Congestive Fever." There is no doubt, however, that this condition of the organs is the secondary result of the systemic poisoning by malaria.

Typhoid pneumonia, the winter fever of the South, is, in many cases at least, decidedly a malarious affection, requiring to be treated as such. Dysentery, also, is not unfrequently impressed with the same character, being periodical in the recurrence and remission of its symptoms.

In the *treatment* of Pernicious Fever, the indications are clearly two: to produce reaction from the cold stage, and to prevent its return by specific medication. If the stomach will retain it, quinine may be given as the first, perhaps almost the only medicine. The amount, when it is given, must be larger than in ordinary Intermittent. Five or ten grain doses, repeated at intervals of two or three hours until thirty (some say sixty) grains have been taken, will do all that can be looked for from this remedy. More than sixty grains of pure sulphate of quinia in twenty-four hours cannot be given with safety to the patient; and very seldom will more than thirty or forty grains within the same time be appropriate.

Should the stomach be very irritable,

ice may be given to quench thirst, and, as a stimulant, capsicum, in pills of five grains each. To promote reaction, the patient may be placed for a short time in a hot bath, or bags of hot salt or sand may be placed in contact with the back and limbs; a large sinapism, also, being applied to the epigastrium. Some physicians, instead of capsicum, give camphor, or opium, or ether, during the paroxysm, to promote reaction. A good combination is of camphor, opium, and quinine, each in moderate doses, in pill, bolus, or powder, every half hour during the cold stage. If a comatose condition occurs, the opium should be omitted. When the stomach obstinately rejects everything, quinine may be introduced by the rectum, or by hypodermic injection. Calomel is employed, on traditional evidence, by many practitioners, in this disease. Experience in the North with Pernicious Fever is too limited to afford the means of concluding absolutely upon its value in this affection.

Alcoholic stimulants are more apt to be needful in a second or third than in a first paroxysm; unless in those whose habits have been already intemperate, with corresponding feebleness of system.

Cerebral symptoms, such as low delirium or stupor, may be treated by the application of a blister to the back of the neck, cutting short the hair, &c.; not, however, omitting quinine, if the malarious nature of the attack is evident.—H.]

Treatment.—"Happily for us," says the observant traveller Burton, "the old African treatment is now obsolete. A. B. caught fever—gave him calomel, bled him, blistered him—died on the third day." Happily too for those whose lot is cast in India, the same may be said; the antiphlogistic treatment of malarial fevers is no more heard of there. So completely is this the case, that it seems to me like contending with a shadow to say a word in condemnation of it. In Italy, however, this system not only holds its ground, but appears to be carried out with a higher hand than in the darkest days of African practice. Every perversion of normal function occurring in the course of an ague is still looked on as an inflammation, and treated accordingly by general and local bleedings, and a rigorous system of diet. What Dr. Haldane has recently said on this system of treatment generally is, as it appears to me, peculiarly applicable to every form of malarial fever that has come under my observation. "Formerly, when an inflammation manifested itself, it was regarded as something superimposed upon the organism; as an enemy attacking the fortress of life, which required to be attacked by the most energetic measures. Its supplies must be cut off by the enforcement of a rigorous diet, and it must be attacked with the heavy artillery of bleeding, mercury, and blisters. But it was not kept in mind that by these measures the garrison was weakened in an equal degree with the enemy, or rather in a greater degree, so that even if the adversary were overcome and retired from the contest, the patient often succumbed, owing rather to the severity of the treatment than to the malignity of the disease." (The Modern Practice of Medicine: a Lecture by Dr. Rutherford Haldane, M.D., F.R.C.P.)

Most systematic authors direct us to begin the treatment of all fevers with an emetic. In Intermittents, when the tongue is very foul or the stomach oppressed by food, an emetic does good, giving great relief, and hastening the stage of reaction. The longest cold stage I ever saw was not in India but in England. The sufferer was an Indian officer who was subject to ague. He had partaken freely of pickled oysters; in a few hours he was seized with intense nausea, headache, and epigastric oppression, soon followed by severe rigors. When I saw him he had been nearly five hours in this state, and his condition caused great alarm. I immediately gave him a mustard emetic, which speedily relieved him, and rapidly brought on a short and mild hot stage.

It is hardly necessary to caution even young practitioners against the absurdity of giving an emetic to a patient every time he has an ague, without regard to the special circumstances of his case.

The same rule applies to purgatives. They are useful if the bowels are loaded; the action of a purgative tends to relieve the congested condition of the solid abdominal viscera, and prepares the way for the action of quinine. But great caution is required in the use of purgatives in persons laboring under malarial cachexia, particularly in Asiatics.

If there be much urinary irritation, a few grains of bicarbonate of potash with or without a few drops of tincture of opium will relieve it at once.

During the cold stage the patient should have a sufficiency of bed-clothes, but, beyond this, interference is not often called for, and more sufferers prefer to be let alone. If the cold stage is unusually protracted, or it becomes apparent that the vital powers are so oppressed as to be unequal to the development of reaction, then the external application of warmth and the use of stimulants may be required.

In like manner during the hot stage little interference is called for. The bedclothes should be removed as reaction advances; cooling drinks may be given if they are relished or called for, which I have observed is seldom. If the patient be in a debilitated state from any cause, instructions should be given to watch the

patient when the sweating stage begins, lest symptoms of collapse should appear, in which case support and stimulants should be promptly given.

The paroxysm ended, our utmost efforts should be directed to counteract the poison, and prevent, if possible, a repetition of the attack. In cases of first attacks it is of unspeakable moment to the patient to prevent his system from getting, so to speak, into the habit of going through the phenomena of an ague fit. For, although we cannot doubt that the aim and end of the process is so far salutary that it rids the system of a portion of the poison, it is also true that the organism suffers in the process, and that every paroxysm is a step towards the establishment of those organic changes I have described. In quinine, skilfully used, we have a remedy, particularly in first attacks, which almost deserves the epithet "divine" which has been applied to it.

I always assume that in first attacks the type of the disease will prove to be quotidian, and I take my measures accordingly, making sure that the patient shall have thirty grains of quinine between the termination of one paroxysm and the hour when we may look for another. The first dose of ten grains should be given towards the close of the sweating stage, and looking to the fact that when an attack is, as I have already expressed it, waxing, we may expect the setting in of the cold stage perhaps two hours earlier than on the first day, the last ten-grain dose should be given so as to anticipate that time by at least an hour. Quinine in the treatment of agues should always, if possible, be given in solution, with a few drops of diluted sulphuric acid; its bitterness is best covered by the addition of a little syrup of orange-peel. [Less than thirty grains will suffice for the interval in the type of Intermittent which is usual in the Northern United States; fifteen grains, given between the end of one paroxysm and the time of its expected recurrence, will, in ordinary cases, almost never fail to interrupt the attack. Pernicious fever, in the South, requires larger doses. In ordinary ague, especially when the stomach is irritable, the method of Dr. G. B. Wood is best, in the opinion of many who have tried it, viz., giving one or two grains of quinine at a time, repeated at sufficiently short intervals to get in the amount required for sufficient "cinchonization" during the interval.—H.]

If irritability of stomach be present to such an extent as to lead to vomiting, no time should be lost in giving the quinine by enema. The lower bowel must first be washed out with a little warm water, and fifteen grains should then be given by the rectum in four ounces of beef-tea, if that be at hand, if not, in a little thin starch; and the irritability of stomach still continuing, this should be repeated twice at proper intervals during the apyrexia. I have been in the habit of administering quinine in this way with advantage for the last fifteen years. [Hypodermic injection of a solution of sulphate of quinia is resorted to not unfrequently, when the stomach will not retain it. The amount needed is somewhat in the way of this method of administration, and local irritation is somewhat more frequent than after other hypodermic injections.—H.]

If we are fortunate enough to prevent the recurrence of the paroxysm in this our first attempt, the gain to our patient is great, and it is well to maintain a moderate degree of cinchonism for some days, evidenced by "ringing in the ears." This can be effected by giving three or four grains of quinine in solution every four hours. But our duty to the patient is not yet discharged. We must not dismiss him from our thoughts because we have successfully managed his case so far.

In a lunar month from the date of his first attack, even should he not in the interval be exposed to malaria afresh, there will be a tendency in his system to repeat the same phenomena as before, and this tendency will be strengthened by every successive attack. A day or two, then, before the time, the patient should again be brought under the influence of quinine, which should be maintained until that time is past.

[American experience favors bringing the patient again under the influence of quinine at the end of a *week* instead of a month. Many times the attack will, without that precaution, recur on the eighth day following the last chill.—H.]

In military and naval practice we have great facilities for carrying out this prophylactic plan, by simply keeping the names of the men in a list, and requiring their attendance at the hospital for a few minutes daily at the proper time.

Speaking from a large experience, I promise those who may try it the happiest results, not only to the men themselves, but to the State, whose costly servants they are.

When our object has been attained, and the paroxysms have been broken, it is well, if quinine excites nausea or irritability of stomach, to give what may still be required after food, which will entirely obviate any such unpleasant effect; and, what is of no small consequence, prevent the patient from being disgusted with the remedy.

If we fail in entirely checking the return of the paroxysm, we are nevertheless pretty certain to have made some impression, and it will be a favorable sign if it is postponed for two or more hours, as is almost certain to be the case. Under

such circumstances we must proceed as before, slightly increasing the dose of quinine, if we are not satisfied with the extent to which cinchonism was induced.

Most American authors describe quinine as the *antidote* to the poison of malaria. According to Herapath's experiments, not much of the quinine taken into the system is excreted from it, at all events when disease is present. Out of forty grains given to a man with tetanus, only a fifth part was detected in the urine; the remaining four-fifths were either assimilated in the body, or destroyed in their transit through the vascular system.

When given in ague it does not appear so soon in the urine as in health. (Parkes; Ringer.) "It has no marked effect on the water, urea, and chloride of sodium, though it may at once arrest the rise of temperature;" and Dr. Parkes adds, "After ague has been apparently cured by quinine, there occurs in the next two or three days an increase in urea, chlorine, and water, at the hours when the fit would have occurred but for the quinine. In other words, the quinine dissociates these two symptoms, increased temperature and ureal increase; it stops the first at once, but not the second for some days."

[There is no room to doubt the fitness of the designation of *antidote*, as applied to quinine in the cure of Malarial Fever. How it acts, we cannot know while the nature of the morbid poison is unknown. The "fungus theory" has much to commend it as probable, and quinine is found to be destructive of low forms of vegetation; yet demonstration is here wanting.[1] A remarkable observation of Dr. H. Bence Jones, concerning the existence of a fluorescent substance in human blood, was confirmed in 1867 at the Pennsylvania Hospital by Drs. E. Rhoads and W. Pepper. These observers also ascertained that, in malarial cases, there occurred a notable diminution of this fluorescence, and that it was restored after the administration of sulphate of cinchonia in therapeutic doses.[2]—H.]

We occasionally meet with cases in which quinine appears to have lost its control over the malarial poison, the paroxysms returning with unfailing regularity month after month, in spite of the regular prophylactic use of the "*antidote.*" In such cases it will be found that the sufferers have been long in a malarious locality; and, whether or not we can detect enlargement of liver or spleen by palpation or percussion, the miasmatic and melancholic aspect of the patients, and the presence of bile in the urine, point to the imperfect manner in which the hepatic functions are being performed. In such cases a course of the fluid extract of taraxacum with small doses of podophyline are most useful; and if to the above be added the free use for some days of such blood depurants as the bicarbonate, or acetate, or citrate of potash, considerably diluted, it will be found that quinine, before useless, will soon reassert its power. I have seen this again and again, and by this method have cured agues deemed beyond the reach of art without a change of climate. I have not space to enter into the question of the *modus operandi* of such remedies. It is probable that they act chemically on the effete matters in the blood, which in some way interfere with the due action of quinine on the *materies morbi.*

Next to quinine as a therapeutic agent in this disease comes arsenic. It has been used in the East in the cure of agues and their sequels from remote antiquity. Having always been fortunate enough to have access to an abundant supply of quinine, I have not used arsenic much in the treatment of agues. From motives of economy it is much used in the French army, and in much larger doses than British physicians are in the habit of prescribing. Boudin, acting on the principle that in paludal fevers there is great tolerance of arsenious acid, is in the habit of giving it in divided doses, and, with the watchfulness always required in the use of this powerful poison, to the extent of a grain and even a grain and a half in the intermission. (Morehead, *Clinical Researches.*) Like quinine, its use should be continued for some time after the cessation of the fever, of course in diminished doses. We should carefully look for the earliest signs of its constitutional action; these are watering of the mouth, a silvery appearance of the tongue, redness of the eyes: and the medicine, as a rule, should be given after food. In the brow-ache and hemicrania of malarial localities arsenious acid is very efficacious—often more so than quinine.

I gave an extensive and careful trial of sulphate of bibeerine, prepared by Messrs. Duncan and Flockhart, of Edinburgh, and found it useless in the treatment of all forms of Intermittent Fever. Barberry, a very old remedy in ague, I have also tried in the shape of tincture. I think it possesses some power as a febrifuge, but it is so uncertain in its action, so immeasurably inferior to quinine, that, in my judgment, to use barberry when quinine is available, is to trifle with the constitutions of our patients. Of Warburg's Tincture, as a febrifuge of undoubted efficacy, I shall have to speak when I come to remittent fever.

[1 See, on this topic, Dr. Salisbury, Am. Journ. of Med. Sciences, Jan. 1866, and Dr. H. C. Wood, same journal, Oct. 1868.]

[2 Pennsylvania Hospital Reports, 1868, p. 269.—H.]

[Various substitutes for quinine have been proposed and largely tried. Among these, opium, chloroform (by the mouth, in f℥j doses), and bromide of potassium have had a number of successes. Dogwood bark (Cornus florida), salicin from willow bark, piperin, the leaves of Eucalyptus globulus, and other vegetable materials have been used with variable, often favorable, results.

Other derivatives of Peruvian bark have been much subjected to experimental use of late years. Sulphate of cinchonia has, in doses once and a half larger, equal efficacy with the sulphate of quinia. It less often affects the head, but is sometimes more trying to the stomach. Sulphate of quinidia will act very well in doses not much larger than those of quinine. Similar accounts are given, by physicians practising in malarial districts, of sulphate of cinchonidia, quinoidine, and dextro-quinine. All of these are less expensive than quinine.—H.]

Diet.—I have already shown that Captain Burton has sound therapeutic notions on malarial fevers; I think him equally sound in what he says on diet. He observes: "People will act up to the old nursery saying, 'Starve a fever, feed a cold.' My experience in East Africa long ago untaught me that tenet. I have ever since preferred to support exhausted nature with essence of meat, and beef-tea, and, when such things are procurable, with champagne, brandy cum soda, and y^e^ oldest hock in y^e^ cellar." These may not be available for hospital purposes; but when such wines are used, they should be "the best in the cellar," or they should not be used at all. Dr. Blair, of Guiana, in his admirable Treatise on Yellow Fever, is quite as explicit on this point as Captain Burton. In the adynamic forms of intermittent, or in any form occurring in asthenic subjects, due attention to support during the intermission is the most essential part of the treatment, and it has been very well insisted on by Dr. Morehead, in his great work.

Treatment of Complications. — In the course of an Intermittent Fever, cerebral, pulmonary, hepatic, and gastric complications may occasionally be expected. After no small experience, I unhesitatingly say, that the occurrence of drowsiness, mental confusion, suffusion of countenance, and such like symptoms, should not mislead us into the use of routine remedies directed against them. Let all our efforts be used to cure the fever, to stop the paroxysms, and to the due support of our patients. When these objects are attained, the head symptoms will disappear. In like manner the presence of cough or asthma, or of hepatic congestion, should never induce us to lay aside quinine and the other means recommended above, in order to direct routine treatment to counteract this or that incidental symptom occurring in the course of a malarial fever. Those who do so often put the lives of their patients in great peril.

Practitioners who omit the use of quinine from a groundless fear of aggravating such symptoms, and substitute strong measures of a so-called antiphlogistic kind, applying leeches and cold to the head, giving nauseating expectorants and active purgatives, merely because of the head symptoms already mentioned, the presence of some bronchitic râles and cough, or some tumefaction of the side, will not only have little success in the treatment of malarial fevers, but will aggravate the complications they seek to cure, hasten alarming exhaustion, or bring on sudden collapse.

I have seen epilepsy, long dormant, developed again during the course of a severe Intermittent Fever. The same principle guided me in the management of this case; I redoubled my efforts to extinguish the fever with quinine, and succeeded, and with the fever the epileptic attacks disappeared. Had I interrupted the administration of quinine and directed my efforts against the nervous complication, I feel assured that I should have made no impression on it. With each recurring paroxysm, the epileptic attack would have been repeated, the old habit would have been re-established, and, if nothing worse happened, my patient would have become a confirmed epileptic. It is an old observation that nervous diseases are sometimes remedied or superseded by Intermittent Fever. One case, and one only, lending some support to this supposed fact, has come under my observation. A medical officer in India became subject to epileptic attacks, which resisted treatment. It was feared that he had become a confirmed epileptic. He was attacked by Intermittent Fever, which affected him at intervals for many years; but from the date of his first paroxysm of ague he never had another epileptic attack. I knew this officer in India well; the fits of epilepsy were genuine, and the attacks severe and frequent.

Treatment of Malarial Cachexia.—The first and most essential part of the treatment is removal to a non-malarious climate. If for this end a sea voyage be possible, so much the better; but unless the ship on which the voyage is made be one in which strict hygienic discipline is maintained, much of the good to be expected from this measure will be lost; and if, from inattention to proper dietetic rules, a scorbutic taint be added, the condition of the patient will be seriously aggravated. I have dwelt on this at some length in the *Army Medical Report* for the year 1861-62.

A nutritious diet, abundance of exercise, pure air, and pure water, are powerful curative means. The careful use of some of the means practised by the so-called "hydropathists" for restoring the functions of the skin is strongly to be recommended; a walking tour on the mountains of Scotland, or, better still, a season or two on the moors, will do much to work the poison of malaria out of the system. Great attention should be paid to clothing, which should be warm, particularly on first coming into high latitudes. Sir Ranald Martin has well shown the evil consequences resulting from want of attention to this.

Returns of Intermittent Fever should be met by the prophylactic use of quinine, so given as not to excite gastric irritability. If the patient's appetite be bad, he may take a course of syrup of the phosphate of iron, quinine, and strychnine; a formula for this preparation (now much used at the R. V. Hospital, Netley) is given below,[1] for which I am indebted to my colleague, Dr. Aitken. Some of the other preparations of iron may be, after a time, substituted; a very suitable one is the tincture of the acetate, from the old Dublin Pharmacopœia.

[The great value of iron in the treatment of recurrent or chronic Intermittent, which, in certain cases, although interrupted, is not cured by quinine, is far from being appreciated by the profession generally. Few cases will resist the effect upon the system of Vallet's Pil. Ferri Carb.,[1] given for three or four weeks, after "breaking" the chills in the usual way with quinine.—H.]

Treatment of Enlarged Spleen.—This "ductless vascular gland" enlarges, as has been already stated, under the influence of malaria, without, as well as with, the intervention of fever. The treatment advised above, exercising, as it is calculated to do, a favorable influence on the constitution of the blood, often acts beneficially also on the enlarged spleen. Should this organ, however, continue undiminished in size, we have a powerful remedy in the ointment of the biniodide of mercury, applied over the gland. The experience of many practitioners in India having demonstrated the extraordinary efficacy of this remedy in cases of goitre, it has of late years been successfully used in some parts of India as a remedy in solid enlargements of the spleen.

When used in goitre, a portion about the size of a nutmeg is applied over the swelling with a smooth spatula, the patient is directed to expose his goitre to the sun's rays as long as he can bear the smarting which quickly follows. A second application is immediately made, and it rarely happens that any further treatment is necessary.

Within the last six months we have been very successful in the R. V. Hospital, Netley, in treating enlarged malarial spleens and livers with this ointment. I have pursued the same plan, substituting the heat of the fire for that of an Indian sun. In some cases, where the spleen has extended down into the pelvis, it has, after several applications, been reduced almost to its normal limits; and in only a few cases has it failed to reduce the size of the organ most signally. It has acted just as energetically on enlarged malarial livers.

The mode of treatment is worthy of extensive trial, and I am the more induced to recommend it to the notice of the profession in England because in not a single example has it induced any unpleasant constitutional action.

I am so impressed with the unsuitableness of mercurialization in cases of mercurial poison, that I have watched nar-

[1] The following is Dr. Easton's formula for the preparation of the syrup of the phosphates of iron, quinia, and strychnia (syrupus ferri, quiniæ, et strychniæ phosphat.):—

"℞. Ferri Sulph., ℥v; Sodæ Phosph., ℥vi; Quiniæ Sulph., grs. cxcii; Acid. Sulph. Dil., q. s.; Aquæ Ammoniæ, q. s.; Strychniæ, grs. vi; Acid. Phosph. Dil., ℥xiv; Sacchar. Alb., ℥xiv.

"Dissolve the sulphate of iron in one ounce of boiling water, and the phosphate of soda in two ounces of boiling water. Mix the solutions, and wash the precipitated phosphate of iron till the washings are tasteless. With sufficient diluted sulphuric acid, dissolve the sulphate of quinia in two ounces of water. Precipitate the quiniæ with ammonia water, and carefully wash it. Dissolve the phosphate of iron and the quinia thus obtained, as also the strychnia, in the diluted phosphoric acid; then add the sugar, and dissolve the whole, and mix without heat.

"The above syrup contains about one grain phosphate of iron, one grain phosphate of quinia, and one thirty-second of a grain of phosphate of strychnia in each drachm. The amount of phosphate of quinia might be increased according to circumstances, and if eight grains of strychnia were employed in place of six, as in the above, the phosphate of strychnia would be in the proportion of the one twenty-fourth of a grain in every fluid drachm of the syrup. I would scarcely venture on a much larger dose. In cases of delicate children, with pale countenances and deficient appetites, I have given, with great benefit, a combination of equal parts of the above syrup, and of that prepared by Mr. Edward Parrish, often called 'Chemical food.' To children between two and five years of age the dose of this combination may be a teaspoonful three times daily." (*Vide* Aitken's Science and Practice of Medicine, vol. ii. 657.)

[1] Three or four grains thrice daily.—H.]

rowly for any ill effects from the use of this compound, without observing anything to justify my fears; on the contrary, in every case where it has acted on the spleen or liver in the manner above described, an immediate improvement in the patient's general health has been the invariable result.

REMITTENT FEVER.

DEFINITION.—A specific paroxysmal fever, with exacerbations and remissions, characterized by a slight and ill-defined cold stage, which does not recur at every exacerbation; an intense hot stage, with violent headache and gastric irritation; and an almost imperceptible sweating stage, which is sometimes wanting.

SYNONYMS.—Bilious Fever, Bilious Remittent, Endemic Fever, Marsh Remittent, Gastric Malarious Remittent, Jungle Fever.

HISTORY.—This, the gravest form of true miasmatic fevers, has been observed wherever malaria is generated in sufficient concentration, both in hot and in temperate climates, but it is most prevalent and fatal when high temperature and malaria act in combination.

Our armies have suffered from it in Spain and Portugal, at Walcheren, and in the Mediterranean; it prevails with great intensity on the western shores of Africa, and in the course of its great rivers. In the East Indies it is most common as well as fatal in the delta of the Ganges, in the terrais, in the jungles at the base of mountain ranges, and, at certain seasons, in many uncultivated and jungly plains.

It is found in America, North and South, prevailing chiefly, with varying degrees of severity, in the vast region between the northern lakes and the Gulf of Mexico, and it is a formidable endemic in the West India Islands.

Remittent Fever is a more serious disease than any type of intermittent, and the direct mortality from it is much higher.

In all intermittent fevers there is a period of complete apyrexia; in remittents, the defervescence is not complete. The more urgent symptoms between one exacerbation and another abate; in some cases this abatement is well marked, in others it is so slight that the period of so-called remission may escape the notice of all but a wary and experienced observer.

A remittent fever may, after a time, pass into one or other of the types of an intermittent; and conversely, an intermittent may assume the graver form of remittent, either under the influence of a fresh charge of malaria, or as I have frequently observed under the stimulus merely of exposure to a high temperature.

Remittent Fever, as we have seen, has many synonyms, but there is one, the use of which I earnestly deprecate, viz., Inflammatory Remittent, still used by many authors, although not always in the same sense. Some employ the term merely to denote a high degree of intensity in the febrile phenomena, without wishing to imply the existence of inflammatory action; while others use it in the belief that the terrible disturbance of the vascular and nervous systems is due to a genuine phlogosis. In either view the term is objectionable; one is based on false views of the true pathology of the disease, and both are calculated to mislead in treatment.

Modes of Commencement.—As in all paludal Fevers, so in this, symptoms of gastric irritation are the first evidence of approaching disorder. The patient complains of præcordial anxiety, anorexia, and nausea, with weariness, languor, and lassitude. Uneasiness, sometimes amounting to great oppression, at the epigastrium, is perhaps the most constant, as it certainly is the most distressing, of the signs of an approaching attack of Remittent Fever; it is sometimes present for twenty-four or thirty-six hours before the setting in of the cold stage.

The time of day at which the first paroxysm takes place is not uniform, but when the disease is established a morning remission is almost an invariable rule. (Wood; Hunter; Morehead; Balfour.) It is a point of great practical importance to observe in each case the periods of exacerbation and remission, and the duration of each. If the exacerbation begins at noon it will usually decline about midnight, or a little before, and the remission will last till noon next day. Such cases are usually comparatively slight and manageable. Or the exacerbation may begin at midnight, continue all night, and remit in the morning, the remission lasting till midnight. Or, in severe cases, there may be a double exacerbation, at noon and at midnight, the remissions being in the evening and morning, the last being invariably the most distinct.

When the disease assumes this type, the evening remission is sometimes so slight as to escape notice: but for the invariable morning remission it might be mistaken for a continued Fever. Treatment will of course have an effect on the periods of exacerbation and remission, just as it has on the paroxysms of an intermittent.

Duration.—This may be stated at from five to fourteen days. Like all forms of miasmatic fevers, the duration of the disease is much affected by treatment and the action of antiperiodic remedies.

Modes of Termination.—The Fever terminates in recovery, or by passing into some type of intermittent, or in death. When the disease ends in recovery, as it usually does, the fever generally culminates in a profuse perspiration, and so ends. Sometimes the amendment is gradual, the exacerbations become less severe, the heat of skin is less pungent, the vomiting and oppression at the epigastrium subside, the pulse diminishes in force and frequency, the tongue becomes moist and clean, the remissions are longer and more distinct, and they are ushered in by free action of the skin; and in this gradual way the Fever subsides.

In some remittents, in which the paroxysmal character has been from the first well marked, if antiperiodic remedies have not been skilfully used, the disease is apt to become chronic, and to pass into one or other of the intermittent types.

I have never seen death take place earlier than the eighth day. This event appears to be determined by the destruction of the vitality of the blood by the agency of the morbific cause.

When death occurs at a later period, the patient sinks, partly from the cause just mentioned, and partly from exhaustion, consequent on the excessive and prolonged excitement of the system. Here again treatment exerts a powerful influence, and may determine not only the time but the mode of death.

Cause.—On this subject I have nothing to add to what has already been said under the head of intermittent fever. I believe that all forms of Remittent Fever are due to the same miasmatic cause as intermittent, aided probably by high temperature. When a Remittent follows exposure to malaria it is reasonable to suppose that the morbific agent has been absorbed in a high degree of concentration.

I attribute some effect to the agency of high temperature, because remittents are more common in hot malarious countries than where the same poison exists in temperate regions, and because, as already mentioned, I have, in a great many instances, observed intermittents pass at once into a severe type of the remittent form, apparently from no other cause than the descent from comparatively cool and elevated regions into the heated plains, and this under circumstances when there was no reason to believe that the sufferers had been again exposed to malaria.

The period of incubation is also, I think, much influenced by temperature—being shorter in hot, longer in cold climates—and doubtless also by the degree of concentration of the poison. I have already given an example of a fatal miasmatic fever being developed within a few hours after exposure to the cause in a notorious malarial locality. From a week to ten days or a fortnight have in my experience been the most common periods of incubation.

Symptoms.—These in an uncomplicated Remittent Fever, occurring in an adult of a tolerably vigorous constitution, are as follows: The most constant, as well as the most urgent, of the premonitory symptoms, already described, is oppression at the epigastrium. The cold stage is neither so complete nor so long continued as in ague; in some of the worst remittents I have ever seen, no rigors were observed at all, a passing sensation of chilliness, alternating with flushes of heat being all that the patients complained of, as regards temperature. In other cases rigors are present, but they are seldom severe; here, as in ague, these sensations of cold are merely subjective, the thermometer indicating already a temperature perhaps 2° above the natural standard, which, as the hot stage develops, mounts to 7° or 8°, and, according to some observers, even 10° above that standard.

As the hot stage advances, vomiting often begins, and continues throughout the disease a distressing and embarrassing symptom. The sense of fulness and oppresion at the epigastrium is not relieved by the vomiting, although the amount of fluid thrown off is out of all proportion to the quantity taken. The tongue is furred, and, as the temperature of the body rises, dry. The pulse, which in the premonitory stage was slow, small, and irregular, now rises rapidly to 100 or 120; in men of sthenic habits it is full; in adynamic cases from the first, although very frequent, it is small and compressible.

The countenance is flushed, the eyes suffused, and the patient complains of rending headache, with pains in the limbs and loins; the skin is red and distended; the heat ardent and stinging; the sufferer is restless, and tosses in his bed in the vain search for an easy posture.

When the above symptoms have lasted for from six to twelve hours, they begin to abate; a slight degree of moisture breaks out on the brow and neck, and gradually spreads over the body; the pulse comes down in force and frequency; the heat of skin diminishes; there is some relief of headache; vomiting ceases; and the patient obtains some sleep.

This is the period of remission.

In severe fevers it is sometimes hardly possible to distinguish any but the morning remission, so slight is the abatement. Keeping this in mind, it should be anxiously looked for. The pulse will often give the only indication, and the physician who knows how precious for treatment such golden moments of remission are, will diligently watch for the earliest

signs of lull in the midst of this tempestuous action of the system.

After a respite of some hours, varying from two hours to eight or twelve—seldom so long—the fever returns, often without a chill, or so slight a one as hardly to attract notice ; all the symptoms above described are renewed, and always in an aggravated form. This is technically called the exacerbation, which in due time passes into the remission again.

I have already pointed out the usual course of the periods of remission and exacerbation, but I repeat once more that the attack is dangerous just in proportion as the disease resembles a continued and not a paroxysmal fever.

Of all the symptoms *nausea* and *vomiting* are the most constant and the most exhausting : the vomited matters at first consist of any food that may be in the stomach, then of a watery fluid, often in surprising quantity. Soon bilious regurgitation takes place into the stomach, and the rejected matters become of a greenish-yellow color, then brown, and finally in extreme cases, black, resembling the "black vomit" of yellow fever. The resemblance will be more striking if, as sometimes happens, the skin assumes a yellow tinge, and a hemorrhagic tendency be evinced. I have seen two cases at Madras, both in officers of the Forest Conservancy department, in which the hemorrhagic range was most extensive, the patients passing blood from the stomach, bowels, and kidneys.

In one of these cases I had the advantage of the assistance of Dr. Cornish, the able Secretary of the Principal Inspector-General of the Madras Army, and we both agreed that the symptoms in this case came nearer in their *ensemble* to those of true yellow fever, than any we had ever seen in the whole range of our experience in the east.

Headache is a prominent symptom. It is described as throbbing at first, passing, in subsequent paroxysms, into a constant pain, with some degree of tension of the brow, sometimes said to be "rending."

Delirium.—Some confusion of thought is common enough, but violent delirium is rare. In very adynamic cases, or where depletion has been freely used, low muttering delirium precedes the coma in which cases of this description often close.

Hiccough is often troublesome where gastric irritability is severe, but it usually appears as the disease is passing off. In one of the cases alluded to above, it lasted for days.

The Bowels are usually constipated in the first instance, sometimes large watery evacuations appear early in the disease, at other times when it is passing away.

Practitioners whose choicest weapon against "bilious" remittents is calomel, are but too familiar with the dark brownish-black evacuations of cadaverous odor, the appearance of which too surely indicates that an unfavorable termination of the case is at hand.

The state of the *urine* is deserving of the closest study.

Many authors of great authority write of it as always scanty, high colored, and of high specific gravity. But in severe Indian Remittents I have noticed the opposite condition. Dr. Cornish, I am sure, must remember that in the case we saw together there was even diuresis throughout, until convalescence was established. I have notes of three other cases, in which the same condition prevailed ; in all four the urine was bloody. It is invariably acid, seldom contains albumen, so seldom that Dr. Parkes has proposed this as one of his points in the diagnosis between yellow fever and malarious Remittent, on the strength of Ballot's observation, who found the urine albuminous in 300 cases of yellow fever. According to Jones of Georgia, the urea is increased in severe Remittent Fever ; and the uric acid lessened till convalescence, when it increases. The pigment also is lessened. (Parkes on Urine.)

The older authors describe, and very graphic some of their descriptions are, what they called *putrid* Remittents. These, as Sir Ranald Martin has well shown, were all complicated with scurvy, the inevitable result of protracted sea voyages to India in overcrowded and otherwise unsanitary transports, not supplied with antiscorbutic remedies. Soldiers treated in this way were landed on the malarious shores of Bengal, and when smitten with the endemic Fevers of the country, the symptoms presented "putrid" phenomena, and the mortality was shocking. The medical officers of the first expedition to China in the year 1840, had an opportunity of observing, under the unsanitary arrangements which characterized so conspicuously the first occupation of Chusan, the terrible effects of malaria and scorbutus combined. An entire regiment, 900 strong, was almost destroyed by malarious Fevers and bowel complaints in a few weeks, and such of us as survive can bear testimony to the truthfulness of the description of "putrid" Remittent Fevers given by the writers above alluded to.

Without, however, any scorbutic taint, we may have Remittent Fever presenting from the commencement an adynamic character. I was very familiar with cases of this kind when serving in the immediate vicinity of the great native city of Hyderabad in the Deccan. From the malarial quarters of that densely populated and most unsanitary city, I used to receive into the Residency Hospital, during the autumnal months, a number of

cases of this kind, presenting from the first signs of great depression, the fever after the second or third exacerbation becoming almost continued, the skin being yellowish and covered with petechiæ, the pulse exceeding 120, small and compressible, the tongue dry and black, the teeth covered with sordes, the respiration quick, and sometimes irregular, the abdomen distended, the bowels loose, and a disposition to hemorrhage from nose, mouth, and bowels, and almost invariably delirium, with a tendency to coma. Such cases, unless energetically treated, hasten rapidly to a fatal termination by exhaustion and coma.

Remittent Fever with *cerebral affection* is usually met with in India in sthenic Europeans lately arrived in the country, who have been living imprudently and at the same time exposing themselves to the sun.

In such cases the exacerbation is severe, with ardent fever, a full and firm pulse, mental confusion, and even delirium.

Hepatitis is a rare complication. This is Morehead's experience ; in twenty-two years' service in India I never saw a genuine case of this complication. I have repeatedly alluded to gastro-duodenal irritation as a prominent symptom in malarial fevers ; tenderness, proceeding from this cause, is often mistaken for hepatic inflammation.

Jaundice.—Some degree of yellowness of the skin is common in remittents, both of an ardent and adynamic type ; but completely developed jaundice is more rare.

It occurred in twenty-eight out of 114 of Dr. Morehead's select clinical cases ; I have seen a few examples of this complication, but never had an opportunity of examining one after death. Ten of Morehead's cases proved fatal.

Pressure on the common biliary duct by the head of the pancreas was found in one ; in another the hepatic and common ducts were obstructed by a lumbricus ; and in two there was constriction of the cystic duct. Traces of inflammation of the mucous membrane of the stomach and duodenum was observed in six cases. In almost all there was enlargement of the lymphatic glands situated near the entrance of the common biliary duct into the duodenum, but not to such an extent as to cause pressure. (Morehead.)

Splenic enlargement is found, as in other types of malarial fever, but not so frequently as in Intermittents, probably because the cold stage in remittents is not so prolonged, and also because the whole duration of a remittent is shorter than that of an intermittent fever. When found after death it may, as in some of Morehead's clinical cases, be traced to the influence of previous attacks of an intermitting character.

Diagnosis.—After what has been said, it is unnecessary to enter into the points of difference between an intermittent and Remittent Fever.

There can be no doubt that enteric fever and adynamic forms of remittent have often been confounded in India, and yet the diagnosis to a careful observer is not difficult. In the former, if the characteristic eruption appears, it will settle the point ; but, in India, this is more frequently absent than in this country. Gastric and duodenal irritability are present in all remittent fevers, absent in enteric.

The fever is more or less paroxysmal in the former, continued in the latter. In Remittent the skin is frequently yellowish ; not so in enteric fever. In the former, apart from the epigastric and hepatic regions, abdominal tenderness is generally absent ; present in the latter. In Remittent, the stools are dark colored and bilious ; in enteric fever, they are brownish yellow, with whitish yellow-colored flocculi floating in them.

If hemorrhagic tendency evinces itself in the course of a Remittent, the blood proceeds from the mouth, nose, urinary organs, and bowels ; while in enteric fever, it is from the intestines only, at an advanced stage, during the separation of the glandular sloughs. Lastly, careful examination into the history of the case, with an eye to causation, will materially assist the diagnosis.

Specific yellow fever being unknown in India, no question as to diagnosis between it and Remittent Fever can arise there. Elsewhere, the diagnosis will be established, as it appears to me, by attention to the following points :—

Yellow fever is continued ; all Malarial Fevers are paroxysmal.

Albuminous urine is the rule in yellow fever—a rare exception in Remittent. The spleen enlarges during the paroxysm of malarial fever, but is not affected in the other. Hemorrhage from the stomach and other viscera is the rule in yellow fever ; comparatively rare in malarial fevers. Death is common in yellow fever on the third day, but seldom occurs in the most malignant remittents before the seventh.

Over yellow fever quinine has no power, save when there is a malarial complication ; over true malarial fevers of every type, its power is beyond the reach of question. Convalescence after yellow fever is rapid and agreeable ; slow after malarial fevers.

Pathology.—Having gone fully into the pathology and *post-mortem* appearances of Malarial Fevers in the previous article, it is unnecessary to enter again into a description which would involve

much useless repetition; such points as more especially belong to Remittent Fevers I have incidentally considered in connection with the symptoms.

Prognosis.—Death from an uncomplicated Remittent Fever ought to be a rare occurrence, and, under good management, so it is. Even in ardent cases in sthenic constitutions, the prognosis is favorable; and, if skilfully treated, recovery may be confidently looked for, in a large majority even of adynamic cases, if seen sufficiently early.

The early subsidence of gastric irritability, headache, and frequency of pulse, are favorable signs. Distinct remissions, with action of the skin, and postponement of the exacerbation, are also evidence of an approaching favorable change. On the other hand, if the fever is more continued than paroxysmal, with a pulse failing in strength while it gains in frequency: if there be a tendency to collapse at the close of the exacerbation, profuse hemorrhage from the stomach or bowels, cold sweats, delirium, and coma; in a word, signs of extreme exhaustion, these all point to danger, and indicate the necessity for restorative treatment of an energetic character.

Treatment.—The disease is not an inflammation; therefore, bleeding or antiphlogistic remedies have no legitimate sphere of action in the contest. I freely admit that a young physician has no more difficult lesson to learn on being brought face to face with his first case of tropical Remittent Fever, in the person of a young and vigorous adult, than to restrain his hands from the use of such means. When he feels the burning heat of skin and full pulse, sees the flushed face and congested eye, the tongue furred and dry, and listens to complaints of a head racked with pain, and limbs vainly seeking relief in incessant change of posture; and, in addition, sees his patient distressed by an intolerable sense of oppression at the stomach, which is not only not relieved, but is even aggravated by almost incessant vomiting;—when, I say, all this is seen for the first time, an inexperienced man is apt to say this is the "inflammatory remittent of so and so; here antiphlogistic treatment must be used, or my patient is lost."

I believe there is another and a better line of treatment available.

The best possible hygienic arrangements should be made, and care taken to secure careful ventilation of the room or ward.

The history of the case should be inquired into, particularly with a view to ascertain the hour of first accession, so that the period of probable remission may, as nearly as possible, be calculated, in order that the visits of the attendant should be so timed as to insure the patient being seen, particularly at the morning remission.

Cold stage.—This, in Remittent Fever, is so transient that patients are seldom seen at this time, and no special treatment is required.

Hot stage.—It is seldom necessary to order an emetic in fulfilment of the time-honored practice of beginning the treatment of all fevers with one. Vomiting, in a great many cases, requires rather to be checked than encouraged: where, however, there is much nausea and oppression, or sense of fulness at the epigastrium, without vomiting, a few tumblers of tepid water may be given, which will answer the purpose. It is well, also, that the bowels should be thoroughly cleared as soon as possible. For this purpose, from three to five grains of calomel, extract of colocynth, and scammony should be given, with a few drops of any aromatic oil. I have found this combination very effectual; it rarely, if ever, gripes or causes nausea; it appears to act on the whole tract of the intestine, and seldom requires a nauseous draught to aid its operation.

If the hot stage be mild, without much headache, ardent heat of skin, epigastric or hepatic tenderness, not much interference is required, beyond giving the patient small quantities at a time of iced or well-cooled water, soda-water, or lemonade, if any of these are relished.

If, however, the reaction be very vigorous in a sthenic adult, with severe headache, pungent heat of skin, severe pain in the loins, and much restlessness, something must be done for his relief. The hair should be cut close, and cold should be assiduously applied to the head, taking care that this is not done, as it often is, so as to act as a fomentation.

The inordinate heat of the skin may be reduced (*a*) by cold affusion; (*b*) by sponging the surface with tepid water; (*c*) by enveloping the body in a sheet, wrung out of cold water, and changed two or three times; (*d*) by packing the patient in a wet sheet, after the manner used in hydropathic establishments. Each method has its advocates; and in such cases as I have described above, I do not think there is risk in any of them. Where, however, there is marked epigastric or hepatic tenderness, with a tendency to extreme hepatic or splenic congestion, as pointed out by Morehead, the use of the wet sheet *alone* is objectionable, as tending to aggravate that condition. The wet sheet-packing acts powerfully on the skin, and is more likely to relieve, than to increase, congestion of internal organs. This method, however, must be used with caution, if at all, in cases of an adynamic

character, as exhaustion and even collapse might follow the powerful sudorific action it generally excites. The sponging of the surface with tepid water is always safe and grateful to the patient.

Vomiting in severe and so-called "bilious" Remittents is often one of the most distressing symptoms. I have seen it induce great exhaustion. It is best combated by giving small quantities of ice-cold water, or by sucking small lumps of ice in the folds of a handkerchief, by the repeated application of sinapisms to the epigastrium, by inhaling the vapor of a *few drops* of chloroform, or by the addition of the same to an effervescing draught. I have seen the wet sheet-packing most effectual in restraining vomiting and relieving the distressing sense of oppression at the stomach, which the sufferer vainly endeavors to get rid of by incessant retching.

Hepatic and splenic tenderness should be relieved by fomentations, stupes, or sinapisms. Sometimes a pad of lint well sprinkled with chloroform covered with oiled silk, and applied to the epigastrium, is very soothing. By treatment of this kind, diligently used, the necessity for abstracting blood by leeches is obviated.

Treatment during the Remission.—On the first signs of this, as soon, that is, as moisture appears, and the heat of skin abates, and the pulse comes down in force and frequency, quinine should be given in an effective dose of not less than ten grains. I have given fifteen often, twenty sometimes; I am no advocate for excessive doses, and any quantity over twenty grains I deem to be excessive. I am never deterred from giving quinine merely because, even in the remission there may be headache or foul tongue. Experience has taught me that the best remedy against those conditions is the one that acts on the toxic agent that is the primary cause of all the disturbance.

I am never deterred from giving quinine, because the remission is slight; so soon as I am satisfied that there *is* an abatement of symptoms I proceed to give the remedy, in the full belief that if I make a skilful use of these golden moments of remission, however slight this may be, I shall be rewarded at the end of the second exacerbation with one more distinct and perfect than the first, knowing also that if through timidity I suffer the first remission to pass unimproved, the next may be more faint, unsatisfactory, and difficult to recognize. I by no means wish to imply that there is always so much difficulty in recognizing the remission. I desire only to guard the inexperienced against expecting to find the remission always so distinct as we find it described in books.

If quinine is rejected, and the irritability of the stomach is such that a second dose is also vomited, twenty grains ought at once to be administered, in any bland fluid, by enema. If the stomach retains the medicine, it should be repeated every second hour until thirty or thirty-five grains have been taken before the hour of expected exacerbation.

Suppose the purgative has not operated, are we to wait for its operation before giving quinine? I have done so, but finding that I lost more ground by delaying quinine than I gained by the action of the purgative, I abandoned the practice. At the same time I am fully alive to the necessity of securing free action of the bowels at as early a period as possible.

As soon as the second remission appears, quinine must be given as before, and continued until full saturation of the system is evidenced by cinchonism, or by a distinct abatement of the disease. Ringing in the ears and deafness are unequivocal signs of cinchonism. So soon as this state is brought about, in a vast majority of instances, the exacerbations will become milder, and terminate in a copious sweat, and the patient will enter on a state of convalescence.

Having ventured to write with so much confidence on quinine used in this way, I am glad to be able to adduce the testimony of Dr. Davy to the safety of the measure. This high authority says, "That in the Remittent Fevers of the West Indies, during the first quarter after the practice was introduced of giving quinine in full doses to cinchonism, out of 165 cases only two proved fatal, and the record of the post-mortem examination in the two fatal cases shows that they were rather instances of latent phlegmasiæ than of fever of the remittent type."

To this unexceptionable testimony I shall only add the evidence of one other observer of equal reputation and experience. The late Dr. David Blair, Surgeon-General of British Guiana, thus expresses himself on the question of the safety of quinine: "It has been prescribed by me to patients of both sexes and all ages, and, where ascertainable, almost invariably to cinchonism, during thirteen years, and probably to the extent of several thousand ounces of the sulphate, and during that time I have seen no danger from its effects, with the exception of three or four cases of imputed abortion."

During the remission the patient should have mild farinaceous diet, milk, chicken tea, and such like. As soon as the gastric irritability subsides, beef-tea should be given, and on the first sign of exhaustion, nourishment and stimulants should be given at short intervals.

Should quinine ever be given during the exacerbation? In the adynamic form of the disease, such as I described as coming from the malarial quarters of the

city of Hyderabad, I never waited for a remission, but gave it at once by mouth or rectum, or both, combining with it the assiduous use of support and stimulants at short intervals.

The American physicians led the way in this practice, and demonstrated its safety.

Again, when called to cases which have been mismanaged in their early stages, either by the neglect of quinine, or the too free use of antiphlogistic means, we must act in the same way. There is no time to wait for a remission: the low form of exacerbation then present will hourly assume more and more a continued type; the remissions, if they appear, will be of short duration, and it will soon be hardly possible to recognize them at all. Such cases can only be saved by energetic means, by quinine, support, and stimulants, given in quantities regulated by their effects. In this way most hopeless-looking cases may be snatched from the jaws of death, as I have seen in a great many instances. What I wrote on the treatment of the complications of ague, I repeat here emphatically. Practitioners who relax in their efforts to stop the exacerbations, who pause in the use of quinine while they apply routine remedies for this or that symptom, now applying leeches to the head because delirium or headache is present, to the epigastrium because there is some tenderness there, will have little success in the treatment of the worst form of Indian Remittents.

My experience has satisfied me that such symptoms are most effectually met by the means which directly tend to counteract the poison which is keeping up the excitement, and disturbing the functions of the organs to which it is conveyed by the circulation; at the same time, active stimulation of the skin over affected organs should not be neglected. It is surprising how much relief may be given by sinapisms, turpentine-stupes, or stimulating embrocations, without having recourse to so double-edged a remedy as depletion.

Mercury has been largely used in the treatment of Remittent Fever. When I first went to India, calomel, chiefly in combination with James's Powder, was used to an extent that, to practitioners of the present day, is hardly credible. "Inflammation" was thought to play an active part in this fever, and as calomel, next to the lancet, was regarded as the most direct antiphlogistic remedy, it was used with the intention of subduing this inflammation.

The end sought was to "affect the mouth," and quinine, when given at all, was only ventured on when this desired consummation was attained, and then timidly in utter ignorance of its real value and true therapeutic action. A practitioner of this school in India in the present day would be an object of terror to all educated men within reach of his prescriptions.

Beyond measure miserable is the spectacle of a man whose system, already saturated with malaria, is still further depraved by the mercurial cachexy.

This was the system which, introduced into India by the late Dr. James Johnson and some of his followers, superseded the admirable treatment recommended by Lind, and others of his day. The consequences were not creditable to our art, and the return to more rational treatment, based on a sounder pathology, has been attended with a signal diminution in the mortality from all forms of malarial fevers.

Before concluding, I must add a few words on a febrifuge, which, in Southern India at least, has attained a high reputation. I mean Warburg's Tincture.

This is a remedy the exact composition of which is kept a secret in the family of its inventor.

In common with the whole profession, I deprecate the use of secret remedies, and, as a general rule, decline to use them.

But it is no secret that quinine enters largely into this combination, and is its most active ingredient. Whatever be the exact nature of its composition, I am bound to say that in Remittent Fever this tincture is, without doubt, an efficacious remedy. It is the most powerful sudorific I know. A person under its influence sweats until he saturates not merely his linen and bedding, but also the mattress on which he sleeps; and his breath and skin for days exhale a powerful and peculiar aromatic odor. I have seen it, in at least thirty cases treated by myself, cut short severe remittents after one or two exacerbations.

It has been largely used for many years by officers in the Mysore Commission, whose duties often expose them to fevers of a dangerous type, in the malarial districts of that province. Major-General Frederick Cotton, of the Madras Royal Engineers, in his evidence before the Indian Sanitary Commission, thus expressed himself regarding this remedy: "Those engaged in opening the passes through the fatal jungles of the western coast of India found that medicine invaluable. In the Parambaddy Pass, especially, a trial of it was made on a very extensive scale, and with admirable results.

"When its efficacy became known, the coolies employed would work in the most dangerous parts of the forest, if they were sure that the medicine was at hand." The tincture is sold at a high price, in small bottles containing two doses. After

the bowels have been opened by an aperient, one-half a bottle is given, all drink is withheld, and at the end of three hours, the remainder is administered.

It has a fine golden yellow color, apparently from tincture of aloes, and its taste is persistently bitter. Sometimes in three hours after the first dose, more frequently in an hour after the second, profuse diaphoresis sets in, and this goes on until the bedding is saturated.

In a great many cases there is no exacerbation after this sudorific action. So great is this action of the skin, and in some cases so exhausting, that this remedy, used in the manner above described, would be highly dangerous in adynamic cases—a fact not adverted to in the printed directions which accompany every bottle. If used at all in such cases, it should be in much smaller quantities than those ordered; and support should be given as soon as the skin begins to act.

My friend Dr. Morehead speaks slightingly of this tincture, but after many trials of it, and a full knowledge of the success that has followed its use in many formidable types of Malarial Fever, I do not hesitate to recommend that careful trials should be made of this remedy in regions where these fevers prevail. But, I repeat, it requires caution; if given indiscriminately in the manner recommended by Warburg, I am confident its use must often be disastrous.

[The approval above expressed of the American practice of giving quinine early, during the exacerbation, is confirmed by the experience of a large number of practitioners in the Southern States. In the Northern and Middle States, however, where the malarial poisoning is evidently less intense in degree, with a less marked adynamic tendency, it certainly answers well, in most cases, to put off giving quinine until the fever has at least *begun* to remit; and then to give it in more moderate doses than are usual in the South.—H.]

DYSENTERY.

By Professor W. E. Maclean, M.D.

Definition.—A specific febrile disease, characterized by nervous depression; by inflammation and sloughing of the glandular apparatus of the mucous membrane of the large intestine (sometimes extending into the small gut); by tormina and tenesmus, with scanty, mucous, and bloody stools of a peculiar odor, changing, as the disease advances, to serous, and giving off a gangrenous effluvia.[1]

Terminology. — Δυσεντερια, δυς difficulty, and εντερον an intestine; Dysenteria, Tormina, Fluxus Dysentericus, Colonia, Colonitis; Dysentery, Flux, Bloody Flux; Dyssenterie, Flux de sang, Fr.; die Ruhr, die rothe Ruhr, German; Dissenterie, Ital; the Looseness (vulg.).

History.—Dysentery was well known to the most ancient writers on medicine, and has largely occupied the attention of modern physicians, particularly of those who have served in fleets and armies. It has been seen in all climates, in the temperate as well as in the torrid zone. No country has been exempt from it; sometimes appearing alone, at others as a formidable complication of malarial fevers or scurvy, often treading in the footsteps of devastating wars, for in all ages it has been the scourge of armies, and one of the chief causes of mortality in unsanitary camps and garrisons.

In the pre-sanitary age it was as common and nearly as destructive to human life in Britain as it is now in unhealthy tropical regions as yet unvisited by the sanitary reformer.

In Ireland "the looseness," as it was called, was a common endemic disease, causing great mortality throughout the seventeenth century, and prevailing occasionally in an epidemic form down to the year 1818.

Dysentery has ceased to be a destructive disease in this kingdom; it has disappeared before a higher civilization, and what it brings in its train, viz., improved agriculture and drainage, more particularly subsoil drainage, the removal of filth from the vicinity of dwellings, the

[1] Most physicians will agree, however, that all the characteristic dysenteric symptoms sometimes occur so as to constitute it a local affection; a colonitis, instead of a specific systemic disorder.—H.]

supply of pure water to our cities, in a word, increased attention to hygiene. Just in proportion as Malaria, the product of moisture and organic decomposition in soils, has been banished from our midst, so has Dysentery ceased to be a prevalent and fatal disease.

In India, among Europeans of all classes, this disease comes next to fevers in frequency, but the direct mortality caused by it is greater than from all the forms of fever known in that country.

"Out of an aggregate British force of 25,433 men of Her Majesty's Army serving in periods of eight and ten years respectively in the stations of Calcutta, Chinsurah, and Berhampore, all in Bengal Proper, there occurred 8499 cases of Dysentery and diarrhœa.—In the presidency of Madras, again, out of an aggregate British force of 82,342 men serving there from 1842 to 1848, there occurred 10,531 cases of Dysentery, and 9189 cases of diarrhœa, making a total of 19,720 cases of bowel disease exclusive of cholera."[1] Nor is this all, for most of the casualties which occur amongst sick soldiers on the voyage homewards from India are from chronic Dysentery.

The Naval Medical Reports show that of late years, except on the East India, China, and West Coast of Africa Stations, bowel complaints do not cause a high mortality. In the Report for 1860 it is said that the "intractable flux" of China was, as usual, far more destructive of health and life than any other disease that attacked the force. The death rate from this cause was in the ratio of 13·6, and the invaliding 25·9 per 1000 of mean strength.

Modes of Commencement.—Dysentery in all its varieties commences in one or other of the following ways. After the presence for a longer or shorter time of signs of constitutional disturbance, the patient is severely griped, with frequent calls to stool, the motions becoming scanty, mucous, and bloody. Or, there is a burst of unhealthy semi-feculent biliary discharges, which soon give place to the characteristic stools first described. In either case the odor is offensive and peculiar, becoming gangrenous as death approaches, after extensive sloughing of the intestinal tissues.

The Course of the disease will depend on a variety of conditions, such as its type, whether it be mild or sthenic, malarial, typhoid, or scorbutic; also on the stage at which treatment has commenced, and still more on the nature of that treatment. In *mild acute Dysentery*, under favorable circumstances and rational treatment, the intestinal lesions are generally moderate in extent and degree; the duration of the attack seldom exceeds eight or ten days, sometimes not more than half that time; healthy alvine discharges, a copious lateritious sediment in the urine, and a free perspiration proclaiming restoration to health. Under less favorable circumstances, injudicious treatment, or, it may be, some imprudence on the part of the patient, the issue may be less fortunate, and the case may pass into the chronic form. In *sthenic Dysentery*, the constitutional symptoms are more urgent, the local lesions are more severe, more rapid, more destructive, leading, if not checked by early treatment, to changes incompatible with life, or, after much destruction of the glandular and mucous tissues of the great intestine and much suffering, to an imperfect recovery. Still, this form of the disease, when early seen and well treated, gives a high ratio of speedy and complete recoveries. The duration of the disease is from nine days to three weeks. It may prove fatal on the eighth or ninth day, or death may not take place until the end of the third week; this termination is ushered in by sudden cessation of abdominal pain, increased fluidity, and gangrenous effluvia of the stools, and delirium.

In the *malarial form*, the course of the disease partakes of the periodic nature of the complication. There are remissions and exacerbations of the febrile symptoms, the solid viscera of the abdomen are apt to be deeply implicated, and hepatic, splenic, or renal symptoms may seriously complicate the case, and determine an unfavorable issue.

The *typhoid or malignant form*, often with a scorbutic taint grafted on it, is the most hopeless and fatal type of the disease. This is the form seen in unsanitary camps and besieged garrisons, in beaten armies after great privations, and sometimes in armies that have not sustained any military disaster, but have the misfortune to be commanded by generals who set at nought the laws of health, as was the case notably in the first wars in Burmah and China, and more conspicuously, in the Crimea.

In this form the lesions are not confined to the colon, but, passing the ileo-colic valve, extend into the small intestine. The disease runs its course in about fifteen days or less; nervous depression is extreme, and the tendency to death is by exhaustion.

Causes.—When we consider the variety of causes to which this disease has been attributed, it is impossible to admit that agents so many and various can give rise to an affection which in all climates has presented so much uniformity in its symptoms and anatomical lesions.

[1] Sir R. Martin.

It appears to me that many of the so-called "causes" of Dysentery must be regarded more as active agents of *propagation* than of *causation*. For my own part, I believe Dysentery to be caused by the action on the blood of a poison having a peculiar affinity for the glandular structures of the large intestine. This poison I believe to be a malaria generated in the soil by the decomposition of organic matter. Once a common and fatal disease in this country, it is now so rare that a London hospital physician rarely, if ever, sees a case of genuine specific Dysentery, save such as have been imported from malarious countries. How comes it that a disease with which our predecessors were so familiar has become so rare? Many of the commonly received "causes" are as much in operation now as then; *e. g.*, the combined action of cold and moisture, the action of irritants on the mucous membrane, unripe fruit, unwholesome and indigestible food of all kinds, feculent and other accumulations in the larger intestines, yet Dysentery does not result. Is it not that for the reasons already assigned, less malaria is evolved from the soil?

It seems that just in proportion as we have banished malaria, so have we got rid of Dysentery. For a long time the prisoners in the Penitentiary at Millbank were subject to visitations of Dysentery at those seasons, and in those states of atmosphere, which most favor the decomposition of organic matter in the soil.

The late Dr. Baly, then physician to the prison, in the Gulstonian Lectures for 1847, has given a most instructive account of an outbreak of this kind. Dr. Baly has shown that the disease which prevailed in Millbank prison was precisely the same in its symptoms, course, and lesions, as that described by Sydenham, and by writers on tropical diseases of the present time. He investigated its cause with much care, and was led to the conclusion that it was "due to a poison introduced from without, viz., a malaria rising from the soil," and that all the conditions required for its production abounded in the close vicinity of the prison.

In India, Dysentery prevails most and is most fatal in moist alluvial soils containing organic matter in a state of decomposition, as for example, very notably, in Calcutta.

It is no doubt true that the disease is sometimes seen, and that in a malignant form, in places which are not alluvial. I cannot give a better example of this than the old infantry barracks at Secunderabad in the Deccan of dysenteric notoriety; but there, as at Millbank, the conditions necessary for the production of malaria were only too abundant.

The sad but instructive history of those barracks has been given by Staff-Surgeon Crawford and the writer of this article. in the Army Sanitary Report for 1860. The barracks stand (for notwithstanding their dreadful history they are still in use) on low ground, swampy on one side, and overshadowed by rocks on the other, exposed to the malarious influences of the marsh in the southwest monsoon, while the rocks on the other side shut out the invigorating northeast wind. A graveyard, now closed, is placed close to the buildings, on a higher level, and in the direction of the natural drainage; another, at a greater distance and in a less objectionable site, is on the south side. The surface and subsoil are thoroughly saturated with organic matter, the removal of which is impracticable. The neighboring soil until recently, when something has been done to fill them up, was furrowed by ravines, in which ordure was deposited by natives; the privy accommodation was of the worst possible construction and badly placed, the barracks were invariably overcrowded, and surrounded by a high wall. Here were all the conditions for the production of *malaria*, and the causation and propagation of disease.

For half a century the loss of life in these buildings, *chiefly from malignant Dysentery*, was shocking. For some years it was nearly one in three of *strength;* so late as 1826 it was nearly one in every five. Between the years 1837 and 1858, out of the annual strength of 834·44 occupying those buildings, the admissions into hospital were 1529·40, and the deaths 37·20. In 1858 the "Royals" occupied the barracks, with an average strength of 1098; there were 2497 admissions into hospital and 104 deaths, being nearly ten per cent. of the strength.

Here we have two examples, one in England the other in India, of the local existence of malaria with a like result, the production of malignant Dysentery; the symptoms and anatomical lesions being alike in both cases, with this exception, that in the barracks, hepatic complications were common, due probably to the greater intensity of the cause, combined with high temperature and intemperance among the soldiers. It is quite true that the provisional term malaria is a vague one. In the present state of knowledge we cannot isolate it, and must here take it to mean a poison resulting from the decay of organic matter in the soil, which, when conveyed into the body, is capable of causing a disease, of which certain anatomical lesions of the great intestine are a characteristic and invariable incident.

The following are usually named as "exciting" causes, but, as already remarked, it is more than probable that some of them are merely active agents in

propagating a disease that has a specific cause.

Impure Water.—"There is," says Dr. Chevers, "the strongest reason for believing that much of the cholera and Dysentery which occur on board the vessels in the port of Calcutta is caused by drinking the always muddy and filthy and often brackish water taken up in buckets over the ship's side. Nearly every person, native or European, who comes to Calcutta suffers, more or less, just at first, from some kind of bowel complaint, but none suffer so much as seafaring men;" and no wonder; for the same authority informs us, "that opposite Calcutta the water is frightfully impure. There it receives some forty tons of excreta daily"—(and we may confidently assume that this enormous mass of impurity contains no inconsiderable proportion of cholera and Dysentery stools, for these diseases are always present in that most unsanitary city)—"a multitude of bodies of dead cattle, and some 15,000 corpses yearly." Dr. Rose makes precisely similar observations as regards seamen frequenting the port of Shanghai in China, and attributes the heavy mortality among them from Dysentery to the same cause, viz., drinking river water loaded with organic impurity, and still further polluted by the excrements of an immense population.

Miss Nightingale, in her summary of the Indian Sanitary Report observes, with too much truth, "there is no reason to hope that any station (in India) has what in this country would be called a pure water-supply, and at some it is to be feared that, when men drink water, they drink cholera with it," and we may add, Dysentery also.

Exposure to Cold.—Dr. Mackay, R. N., describes the mode in which this cause operates among seamen serving in the malarious rivers of China. The men, when they lie down on the deck to sleep, pull up their frocks and coarse under flannel jackets, so as to expose the abdomen. When the cool night wind sets in, the exposed skin of the sleepers, from being bathed in perspiration, becomes dry and finally chilled, and in a very short time they awaken griped, and perhaps sick, and so commences very frequently an attack of what Dr. Mackay calls "Sporadic Dysentery."

Impure Air.—Dysentery once established is propagated by the effluvia from the evacuations of those affected. In most Indian barracks a few years ago, the latrines were so badly constructed, so injudiciously placed, and so ill-kept, as to aid materially in propagating both Dysentery and cholera, by exposing the healthy to the effluvia arising from the evacuations of those affected. I affirm from frequent observation, that barrack-rooms most exposed to the effluvia of latrines always furnish the largest number of dysenteric cases, and the heaviest mortality.

In like manner I have seen the disease propagated in hospitals by the practice of preserving the evacuations of large numbers of dysenteric patients, for the inspection of medical officers at morning and evening visit. No single measure of a preventive kind yet tried has exercised a more beneficial effect on the health of troops in India, than the improvement which has been introduced in the position, construction, and conservancy of barrack and hospital latrines.

SYMPTOMS.—These will of course vary with the type of the disease. It is not pretended that in practice we can always expect to find the line separating one type of Dysentery from another to be sharply defined. The distinctions are not, however, fanciful, or adopted here merely for the sake of convenience in description; for those who have seen much of the disease in tropical countries must be familiar with the different forms about to be described. One form, it is true, often passes into another by insensible gradations, and a strong family likeness runs through them all.

Mild Dysentery.—Careless exposure to cold night air, or to a chill after exertion, is, in malarious regions, the most common exciting cause of this form of the complaint. The chill is succeeded by slight heat of skin, loss of appetite, and sometimes nausea. Griping pains in the belly, technically termed *tormina*, follow, with frequent calls to stool, the evacuations consisting of semi-feculent mucus, with or without an admixture of blood, passed with painful straining, called *tenesmus*. There is seldom much abdominal tenderness on pressure. The tongue is white and moist, thirst is not urgent, but even where the appetite is not much impaired, the griping which soon follows all but the mildest articles of diet makes the patient averse to solid nourishment.

The course, duration, and termination of every form of Dysentery are so much influenced by the patient's prudence or the reverse, and by the treatment to which he is subjected, that it is not easy to describe them. If he is reasonably prudent, abstaining from stimulants, unsuitable food, and the use of irritating purgatives, the disease will probably subside in from seven to fourteen days: under good treatment I have seen it do so in forty-eight hours, or less. The skin resumes its action, and the more this is the case the more rapid and complete is the restoration to health. Feculent evacuations return, griping and straining cease, and the urine deposits a copious red sediment.

It is not always that the disease runs so happy a course, and has such a fortunate termination. Too often young and inexperienced sufferers seek delusive ease from stimulants, strong port wine, or brandy, or both, while the more ignorant and needy try to quench their sufferings in the poisonous and fiery spirits of the nearest bazaar. Under such management the disease may soon assume a more formidable aspect, passing into the acute form, to be presently described, or, after a longer or shorter period of suffering, varying according to circumstances, it may become chronic.

Acute Dysentery.—The disease begins with a chill or well-marked rigor, soon followed by more or less heat of skin, with a quick but usually compressible pulse, and nervous depression. Tormina is much more urgent than in the mild form described above, and the calls to stool are more frequent and pressing. At first the evacuations may be semi-feculent and watery, but this state does not last long; soon they become scanty, muciform, and bloody. If the rectum is much implicated, tenesmus will be a distressing symptom. If the seat of the disease be higher up, not only will tenesmus be less urgent, but the nature of the stools will be different. In the latter case, depraved biliary secretions will be more abundant, and will be intimately blended with mucus, epithelium, and blood. If the rectum be deeply affected, the bladder will often sympathize, being either irritable or so paralyzed as to require the use of a catheter, while the stools will be more muciform and the blood less intimately incorporated with them.

From the first the stools are very offensive; they give out an odor which is peculiar to Dysentery, an odor justly said by Dr. Parkes to be "the most offensive of all the organic effluvia." It is difficult to describe it, but once experienced it is never forgotten, and is in itself conclusive evidence as to the nature of the disease; and not only so, but an experienced physician can in some degree form an opinion from it as to the nature and extent of the mischief going on in the tissues of the affected bowel. The absence of tenesmus, although a great relief to the patient, is by no means *per se* evidence of a slight amount of disease; it merely indicates that the rectum is less involved than other parts of the colon.

Soon the calls to stool become more urgent and frequent, the patient is hardly in bed ere he desires to rise again, each time convinced that he is about to pass something that will relieve him. The abdomen becomes tumid, and, in the region over the parts most implicated, tender. Often this tenderness is well marked at one point and absent in others. At last the patient can hardly be induced to leave the close stool; he desires to remain on it, and strains involuntarily. His patience and temper give way, he becomes irritable, nervous depression increases, and the countenance indicates suffering and despondency. The stools, which at first consisted of a little feculence with bloody mucus, now contain portions of shreddy, granular exudative matter, often resembling washed raw meat. The disease still advancing, the countenance becomes more anxious and depressed; irregular febrile paroxysms come on; the pulse rises in frequency, while it diminishes in force; the abdomen grows more tympanitic; the tongue, still continuing foul in the centre, becomes red at the edges and dry, then dark brown, and finally black. If no improvement takes place, another change in the stools follows; they become serous, of a brownish color, and very copious, exhausting the patient terribly. The effluvia become insupportably fetid, even cadaverous, pervading the whole ward, and penetrating into neighboring rooms, unless the nursing is good. The abdomen becomes more tumid, but pain ceases. In this state the patient often flatters himself that all is well, deeming the cessation of pain a favorable symptom; by and by his mind begins to wander, and, as if in some degree to compensate for past sufferings, his delirium takes pleasing forms, and he dies exhausted without more pain. Under happier circumstances and good treatment, amendment begins before destruction of the affected tissues has gone to such a degree as to be incompatible with life. The stools improve, become feculent, and lose the dysenteric odor; blood and mucus disappear, the abdomen becomes less tumid and tender, the countenance improves (a most favorable sign), the febrile paroxysms disappear, the pulse gains in volume and loses in frequency, and the state of nervous depression passes away. There is still a third termination; the case may pass into the chronic stage; or this state may result after convalescence from the acute attack has been well advanced, in consequence of some imprudence on the part of the patient or those about him; errors in diet; or, in military life, from exposure to hardships before health has been sufficiently restored.

Chronic Dysentery.—As the patient sinks into this unhappy condition, he continues to lose flesh. The discharges, still maintaining much of the dysenteric odor, are for the most part fluid, but they vary from day to day, and even on the same day, being muciform, serous, and bloody. Sometimes they are tolerably natural, and even formed; more frequently they consist of thin feculence, of a reddish-brown color and most offensive smell. Often

they are pale and frothy, and in this condition are usually voided with violence. Power over the sphincter is impaired, often lost entirely. The sufferer does not gain flesh, the appetite may be tolerably good, or capricious, or bad. In any case the food is hurried along the intestine, and the patient is poorly nourished; the tongue is red and glazed, sometimes deeply fissured; night sweats are frequent, the hair drops off, boils are common in various parts of the body, and the patient looks older than he is. This is the general condition, but it is liable to be modified by any complication that may be present, the malarious, or syphilitic, or scorbutic cachexia, or by hepatic disease. The condition of the patient will also depend on the extent of injury done in the acute stage to the mucous and glandular structures of the intestine, the presence or not of unhealed ulcers, the condition of the intestine as regards thickening of its coats, or the opposite state of atrophy: whether or not the lesions are bounded by the ileo-colic valve, or pass into the small intestine; and, in no small degree, on the extent to which the solid viscera, liver, spleen, and kidneys are implicated.

Thus it will be seen that chronic Dysentery is not, as many suppose, merely due to abortive cicatrization of the ulcers left by the acute stage, but is often a much more complex state.

I believe, and shall presently demonstrate, that Acute Dysentery is in a high degree amenable to early and judicious treatment. On the other hand, speaking from a large experience, I affirm that complete restoration to health *by the unaided efforts of nature*, is an extremely rare occurrence; one of two things happens, either the disease destroys the patient, or it passes into the chronic form just described.

Malarious Dysentery.—By this term I mean to indicate those cases in which *malaria* acts with a high degree of intensity on the system, developing, in addition to the symptoms already described, those which are characteristic of malarious fevers.

It is in this form that we find hepatic complications most frequently present—complications which add greatly to the gravity of the disease, and unfavorably influence the prognosis: such cases will be recognized by the periodicity of the febrile paroxysms, the presence of gastric irritability, such as we see in remittent fevers, and by the peculiar nature of the evacuations, which from the first are serous and contain little blood, but have the characteristic dysenteric odor. There is also a therapeutic aid to diagnosis; ipecacuanha is not well borne, and quinine acts powerfully in checking the febrile paroxysms.

Malignant Dysentery.—I have already described the conditions under which the disease is generated; it has been described as "Dysentery plus the typhoid condition." From the first, the patient has a cachectic aspect, the countenance soon becomes sunken, listless, and sodden, a burning heat in the belly is complained of, with great oppression and sinking; the voice is weak, the stomach irritable, and pulse frequent, small, and compressible. The evacuations are serous and bloody, attended with tormina and tenesmus, and the dysenteric odor is intense from the beginning. For a short time after the first burst, the stools may be muciform and bloody, but this condition does not last long; they again become abundant, serous, and bloody, consisting of dissolved blood, serum, epithelium, and gangrenous portions of semi-dissolved mucous membrane, exhaling an insupportable gangrenous odor. The urine is scanty, scalding, and fetid, and in extreme cases the secretion is suppressed entirely, as in cholera. The skin is in general cold and clammy, and immense discharges of blood often take place from the bowels, determining the fatal issue, and also from the mouth and nostrils.

Scorbutic Dysentery is met with when the conditions which induce that cachexy are superadded to any type of Dysentery. I had an opportunity of seeing the ravages worked by this form of the disease during the first occupation of Chusan in 1840, one of the most disgraceful episodes in our military history. Here we have the usual signs of the scorbutic cachexy—pallor, emaciation, extreme lassitude, pains in the loins and limbs, a spongy condition of the gums, which bleed on the slightest pressure. Soon livid and purple-colored spots appear on the legs, and ulcers of a weak and unhealthy kind follow the least injury to the surface, the pulse is feeble, the appetite bad, and the debility extreme. To the above are added the symptoms of Dysentery, only the evacuations are, from the first, fluid, bloody and nearly as offensive as in the malignant form. Scorbutic Dysentery does not run so rapid a course as the sthenic or malignant (typhoid) form, rarely proving fatal in less than three weeks, and, in the more chronic form, it may extend over as many months.

[In the U. S. army *Chickahominy* experience, in 1862, a considerable number of cases of Scorbutic Dysentery and diarrhœa occurred. Great emaciation and debility existed, with loss of digestive capacity and purple blotches upon the trunk and limbs. Several cases were complicated with pneumonia, found, *post-mortem*, to have involved chiefly the lower and posterior portions of both lungs. Death resulted in a number of instances; but the

larger number recovered under antiscorbutic treatment.—H.]

Diagnosis.—This ought not to present any difficulty. In acute cases the tormina, tenesmus, muciform and bloody stools, and above all, their peculiar odor, will distinguish Dysentery from diarrhœa. If seen in a more advanced stage, when the stools have become more fluid, the history of the case, the nervous depression, the blood in the discharges, and once more, their cadaveric odor, will establish the diagnosis.

The author once saw a case in which a grave error in diagnosis had been made. A lad between thirteen and fourteen years of age, the son of European parents in India, was seized with frequent calls to stool, with much gastric irritation, nausea, vomiting, restlessness, and oppression. The practitioner who first saw the case, seeing that the stools were scanty and muciform, without feculence, and passed with griping, pronounced the case to be one of Dysentery, and proceeded to treat it with calomel and opium. Under this treatment the stools, still presenting the same characteristic appearance, became of a greenish color, and the gastric symptoms were aggravated. Seeing the patient at this stage, the first thing he noticed was the entire absence of the peculiar dysenteric odor in the evacuations. On inquiry, it was found that before the appearance of the so-called dysenteric symptoms, the patient, after slight febrile and catarrhal symptoms, had an eruption on his skin, which, after imprudent exposure to a cool sea-breeze, suddenly receded, the recession of the eruption being followed by the symptoms mistaken for those of Dysentery. The patient was placed at once in a hot bath; soon the characteristic rash of measles appeared, nausea and vomiting subsided. Under simple treatment the disease ran a mild course, and the lad soon recovered.

Pathology.—From what has already been said, it will be seen that I regard Dysentery to be as much the consequence of a specific poison as any of the recognized miasmatic diseases. Why this poison should, as Baly has expressed it, have such an affinity for the glandular structures of the large intestine, we can no more explain than we can tell why the poison of enteric fever should evince a like affinity for the glands of the small intestine.

Looking to the whole history of the disease, its mode of propagation and its characteristic lesions, it seems impossible to regard it as a simple inflammation of the mucous membrane, a mere colonitis, but rather as a specific disease, of which inflammation of the glandular structures and mucous membrane of the great gut, and its consequences, are characteristic and invariable incidents.

Morbid Anatomy.—It is difficult to give a perfectly accurate description of the Morbid Anatomy of the complex structures involved in this disease. The descriptions of authors are at variance with one another. The confusion has arisen partly from the ignorance of some of the observers of the healthy structure of the parts described; and also, in some instances, from not tracing the change of structure from its earliest manifestations to its ultimate results, a fault due in many instances to lack of opportunity.

So great is the confusion in the descriptions, that my colleague Dr. Aitken has asked (1) "Whether distinct epidemics are characterized by distinct local lesions? (2) Whether two or more distinct diseases have not been confounded under the one name of Dysentery? Or (3) Whether the various local lesions described by different writers are only so many varieties, forms, or types of the same diseased process—a process modified in particular cases by constitutional peculiarities, or by other circumstances." This latter view Dr. Aitken is of opinion is the one most consistent with observation, and in accordance with what we know of the history of other miasmatic diseases.

In the Dysentery of temperate climates, it is seldom that more than one or two of the anatomical divisions of the great intestine are implicated. In tropical countries, not unfrequently, the morbid process affects the whole tract of the intestine from the cæcum to the anus. Except in Scorbutic Dysentery, it is rare to find the lesion extending into the small intestine.

I propose to describe the morbid appearances under the three heads of Congestion, Ulceration, and Exudation.

In the rare instances in which the affected bowel has been seen before ulceration of the glandular apparatus has commenced, it has been observed to be swollen and somewhat softened, the color of the affected parts being rosy red, purplish, or of a brownish and sometimes a leaden hue. This discoloration of the mucous membrane is by no means uniform; in the non-febrile forms of the disease I have seen the membrane between the ulcers unchanged, and even paler than natural, and there is as much diversity in the degrees of softening of the tissues.

Ulceration.—It is mainly to the careful observations of Drs. Parkes and Baly that pathologists are indebted for an accurate description of this process. At the very commencement of the morbid action, Dr. Parkes has shown that the solitary glands

are enlarged in various degrees, "from the size of a millet-seed to a small shot" (Baly). They are seen to be distended with a white exudation, some with a dark central spot, and all surrounded by a vascular ring. It is on and about these glands that the ulceration, or, as Baly prefers to call it, sloughing process, begins, spreading from them into the mucous membrane. Dr. Parkes was led from his dissections to the belief that ulceration almost always begins in the glands themselves, very rarely around them, and only occasionally, in very rapid cases, by effusion of fluid beneath the mucous membrane. This last is the "circumscribed sub-mucous suppuration," leading to the formation of circular and other shaped ulcers, described by Morehead, Haspel, and Bleeker. This commencement of the ulcerative process must be familiar to all who have seen the rapidly destructive type of Dysentery which so long prevailed in the old infantry barracks in Secunderabad, before described. The ulcers are at first circular and have rounded edges, but as they enlarge the edges become flat, and they spread out into irregular shapes, having for the most part their long diameter in the direction of the plicæ of the mucous membrane round the intestine. In the more advanced cases the whole tract of the gut is studded with ulcers of all shapes, sizes, and degrees of development, presenting in different cases a great variety of appearances—some deep and irritable-looking, others more superficial and pale, some having their floors covered with lymph of various shades, others having their floors formed by the muscular coats. In the rapidly destructive sloughing Dysentery, I am of opinion that the process begins by sub-mucous purulent effusion, detaching the mucous membrane, which rapidly becomes gangrenous. I have seen the whole tract of the mucous membrane, from the rectum to the transverse colon, presenting a black gangrenous mass, in which it was impossible to distinguish a shred of healthy tissue.

As soon as the diseased action implicates the other coats, lymph is effused between them. In chronic cases, in emaciated subjects, the colon can be grasped through the parietes, as a patient of mine once expressed it, "like a ruler." Sometimes the coats of the intestine themselves become thickened and fleshy-looking.

Cicatrization of Intestinal Ulcers.—The process has been well described by Drs. Parkes and Baly. A fibrinous exudation takes place on the floor of the ulcer, which becomes organized. The edges, after being rounded, are drawn down to meet the floor, and from them a delicate lamina shows inwards till the whole surface is covered (Baly).

Exudation.—I have already described an exudative process as being seen at an early period of the disease in the solitary glands, to which, however, it is not confined; it soon fills the neighboring tubular glands, and spreads over portions of the mucous membrane of the colon and rectum, sometimes, but in India rarely, and then only in chronic cases, extending over the entire surface of the intestine, and even passing into the ileum. Usually of a yellow or gray color, it forms a thin granular layer: when this is detached, the subjacent membrane is found to be vascular and red. This exudation, when microscopically examined, is composed of epithelium and fibrinous granules, but in the severer forms, Dr. Aitken describes it as consisting of "fine germs with nuclei, mixed with elongated cell-forms" (connective tissue cells).

This exudation is either detached in fragments with much griping and distress, or becoming to a certain extent organized, it is finally, by a process of ulceration, detached in tubular portions, a process, as shown by Morehead, to be sometimes attended with hemorrhage.

In Chronic Dysentery. — There is no more common error than to suppose that the symptoms in Chronic Dysentery are invariably due to the presence of unhealed ulcers in some portion of the mucous membrane. We constantly see cases in which not a single breach of continuity is found. There is, in such examples, abundant evidence of previous ulceration, and the whole surface of the mucous membrane will be found thickened by a deposition of black granular matter, the result, as Dr. Aitken thinks, "of excessive vascular action and of subsequent changes in the extravasated blood." In other cases abundant ulceration is found, particularly in the rectum and sigmoid flexure of the colon, the ulcers being in every condition, some cicatrized, others undergoing that process, most without vitality enough for healing. Sometimes the whole of the alimentary canal is in a state of atrophy, the glandular structures having disappeared, and the tissues being so attenuated that they are quite transparent. In such cases the general emaciation of the sufferer is extreme. In more recent cases great thickening of the connective tissues, and even of the coats of the intestine will be found. In this condition, the exudation is usually found to have undergone the process of organization already mentioned, and it may be found in a state of ulceration like any part of the natural tissue (Aitken).

The coincidence of Dysentery and hepatic abscess is one of the most familiar facts in pathology. But it is often assumed because no abscess is found at a *post-mortem* examination in a case of Dys-

entery, that the liver is normal. Now, in a case of tropical Dysentery it is extremely rare to find the liver healthy. This need not excite surprise, for when to the predisposing, or exciting causes of the disease we add a high temperature, a diet too stimulating for the climate, and habits of intemperance, we have enough to explain the various hepatic diseases, functional and organic, which so often in tropical climates seriously complicate Dysentery. And this without having recourse to any theory of blood poisoning by the absorption of discharges from the sloughing intestine. This view is strengthened by a consideration of the fact that liver abscess is exceedingly rare in the Dysentery of temperate climates. In all Dr. Baly's cases in the Millbank Penitentiary there was not a single case of hepatic abscess.

Prognosis.—The grounds for a favorite prognosis are, (1) The original slightness of the attack. (2) The absence of much nervous depression. (3) A natural countenance, with a pulse of good strength and moderate frequency. (4) The absence of gangrenous odor in the stools. (5) The early appearance of feculence in the stools; and lastly, the absence of any signs of serious hepatic complication.

On the other hand, rapid failure of the nervous and circulatory systems; a pulse increasing in frequency and failing in strength; an anxious and sodden countenance; extreme restlessness; sudden subsiding of pain with increasing fetor of the stools of a gangrenous character; hemorrhage from the bowels, mouth, or nose; hiccup; black and dry tongue; suppression of urine, and delirium, all point to extreme danger.

Treatment.[1]—It is impossible to overestimate the importance of early treatment in tropical Dysentery. So rapid is the progress of the disease, so terrible the lesions it causes in a space of time apparently insignificant, that no pains should be spared, more particularly by military and naval surgeons, to impress on the minds of those committed to their charge the necessity of seeking medical aid on the first manifestation of the symptoms of the disease.

Mild Dysentery.—In this form I have much confidence in the use of the hot bath as a powerful means of restoring the suppressed action of the skin. The bath should be brought to the bedside of the patient and should be maintained at a high temperature, and the patient should be kept in it until he feels faint. After being quickly and carefully dried he should be put to bed, and a dose, not less than from fifteen to twenty grains, of ipecacuanha should be given in the manner to be presently explained. This may require to be repeated in eight or ten hours.

If the patient abstains from all fluid for some hours after taking the medicine, it is seldom that much nausea or vomiting is produced, provided the horizontal position is maintained, which it ought to be.

The result generally is free action of the skin, rapid subsidence of griping, and the appearance of feculent motions. In some cases it may be necessary to give a few drachms of Fresh castor-oil, guarded by a few minims of tincture of opium, or a few drops of chloroform. And it is well also to stimulate the abdominal surface by the application of a few turpentine stupes.

The above simple treatment will suffice in a great many cases of that mild form of disease which follows chills without much charging of the system with malaria, provided it be had recourse to sufficiently early.

Morehead speaks slightingly of the hot bath in the treatment of any form of Dysentery, but I have found it to be a most useful remedy, used with the precautions and in the manner advised above.

Acute Dysentery.—In this form it is even of more importance than before that the patient should be early seen and treated. He should at once be ordered to bed, and as quickly as possible brought under the influence of ipecacuanha in large doses. Some insist on the propriety of first giving a full dose of Battley's sedative, tincture of opium, or a few drops of chloroform, with the intention of making the stomach tolerant of the remedy, and restraining nausea and vomiting.

I believe that the sedative in some cases is useful, and acts in the manner just described. On the other hand, I have often seen ipecacuanha do its work well, and with little disturbance of the stomach, without opium. Should it be determined to premise opium, thirty drops of the tincture should be given, and in half an hour followed by from twenty-five to thirty grains of ipecacuanha, which should be given in as small a quantity of fluid as possible; a little syrup of orange-peel covers the taste as well as anything else. As already advised, the patient should keep perfectly still, and abstain from fluid for at least three hours. If thirsty, he may suck a little ice, or a teaspoonful of cold water at a time may be allowed.

It is seldom that under this manage-

[1] In every form of Dysentery, both in public and private practice, the stools ought to be received in glazed vessels containing some disinfectant solution, instantly removed from the house or hospital, and carefully and deeply buried. I cannot too earnestly impress this caution on the minds of military and naval surgeons.

ment nausea is excessive, and vomiting is rarely troublesome, seldom setting in for at least two hours after the medicine has been taken. The abdomen should be covered with a large sinapism, or a sheet of spongio-piline sprinkled with a little turpentine after being wrung out of hot water.

In from eight to ten hours, according to the urgency of the symptoms, and the effect produced by the first dose, ipecacuanha in a reduced dose should be repeated, with the same precautions as before. All who have had opportunities of trying this mode of treating Dysentery can bear testimony to the surprising effects that often follow the administration of one or two doses of ipecacuanha given in this manner. The tormina and tenesmus subside, the motions quickly become feculent, blood and slime disappear, and often, after profuse action of the skin, the patient falls into a tranquil sleep and awakens refreshed. The treatment may require to be continued for some days, the medicine being given in diminished doses, care being taken to allow a sufficient interval to admit of the patient taking some mild nourishment suited to the stage of the disease.

This, in a few words, is the system of treating acute Dysentery now almost invariably followed in India. It has been long enough in use on a sufficiently large scale to enable us to appeal to statistics and bring it to the test of figures. It has almost entirely superseded the old plan of general and local bleeding, with mercurialization, either by calomel in scruple doses, or in smaller quantities at short intervals, in combination with opium. Here are the results, derived entirely from official sources :—

Under the old system in Bengal the mortality among Europeans during the forty-two years, from 1812 to 1853-54, amounted to 88·2 in the thousand.

During 1860, when large doses of ipecacuanha were give to the almost complete exclusion of all other methods of cure, the mortality was 28·87 in the thousand.

In the Madras presidency under the old treatment the mortality from the disease during seventeen years was 71 per thousand treated, but when ipecacuanha was largely used, as above described, it fell to 13·5 in the thousand.

In the 44th Regiment, quartered at Fort St. George, Madras, Surgeon Mee treated 68 cases "in the ordinary way" —with a mortality of 6, or 8·8 per cent. Subsequently 59 were treated with large doses of ipecacuanha, and all recovered.

Mr. Docker, surgeon of the 2d Battalion of the 7th Royal Fusiliers, to whom is undoubtedly due the honor of recalling us to a more rational and successful way of treating Dysentery, after he had recourse at the Mauritius to the plan of using large doses of ipecacuanha, lost only one out of fifty-three cases.

I have not space to enter into the complete history of ipecacuanha as a remedy in Dysentery. I must refer my readers to an excellent and exhaustive paper on the subject by Dr. Blacklock of the Madras army in the Madras Quarterly Journal of Medical Science, and to a paper on the same subject by Dr. Ewart of the Bengal army, in the sixteenth number of the Indian Annals of Medicine. Known from remote times in Peru as a remedy for Dysentery, it was introduced into this country as the *Radix Anti-Dysenterica*. It appears to have been used in Europe in the treatment of the disease more as an adjuvant to other remedies than trusted to alone. In process of time ipecacuanha came to be used almost exclusively as an emetic and expectorant, and, in combination with opium, as a diaphoretic in the form of Dover's powder. In the Madras presidency for many years before the publication of Mr. Docker's paper, it was used as a remedy in Dysentery, chiefly after the example of the late Drs. Geddes and Mortimer, and, as I can testify from twenty-two years' experience of its use, with a marked superiority over the plan of treatment by mercurials. The great merit of Mr. Docker was the introduction of the plan of using it in the large and effective doses of which I have spoken, with the gratifying results given above.

It is probable that ipecacuanha owes much of its usefulness in this disease to its action as an evacuant. It is a blood depurant of an effective kind. It appears to increase the secretion of the whole alimentary canal, as well as that of the liver and pancreas ; under its use tormina and tenesmus disappear, and feculent evacuations are more quickly restored than by any other known remedy.

It also promotes free action of the skin, and exercises a sedative action on the circulation. "In fine," says Dr. Ewart, "ipecacuanha in large doses may be said to fulfil many indications.

"It produces all the good effects that have been ascribed to blood-letting without robbing the system of one drop of blood, of mercurial and other purgatives without their irritating action, of antimonials and sudorifics without their uncertainty, and of opium without masking the disease." I may add, that it is the most simple, the most successful, the most conservative, and the least distressing mode of treatment I have ever seen used in Dysentery. Year by year under its use the number of chronic cases is becoming smaller, and hepatic abscess as a complication is less frequently seen. Although most effective in the early stage of the acute form, it may be given at a

much latter period with advantage; and even in chronic cases, where from any cause subacute symptoms have set in, I often give it with the best results. Some authors caution us against its use in large doses in adynamic cases, and doubtless in such cases very large doses are not advisable. Still, I have used it even where the powers of life were very low, and with the best effect. I well remember the case of a lady, sent to Madras from Calcutta, who landed in such a state of exhaustion that it was with difficulty I could hear her voice. With some misgivings, the symptoms being urgent and the case critical, I gave twenty-grain doses of ipecacuanha at intervals of eight hours, interposing support between the doses; after the third dose this lady was out of danger and made a rapid recovery.

If unmanageable vomiting follows the use of ipecacuanha, hepatic complication of a serious kind may be suspected—or the vomiting may arise from the system being overcharged with malaria, severe gastric symptoms of this kind being extremely common in remittent fever.

As soon as the disease abates, the dose of the remedy should be abated also. It is well, however, for some days to administer ten or twelve grains at bedtime, for a night or two after the stools are to all appearance healthy. Fomentations [flaxseed or hot mush poultices.—H.], stupes with turpentine, or the application of strong chloroform liniment to the abdomen, help to lessen tormina and diminish suffering. If a little diarrhœa, without the dysenteric odor remains, it may be checked with a little astringent mixture such as the compound chalk powder, with or without opium. Astringents in any shape during the acute stage are not only useless but dangerous. [After the excitement of the first period has abated, however, the obstinate persistence of dysenteric discharges may often be advantageously met by the use of an *enema*, containing three grains of acetate of lead with thirty or forty drops of laudanum, diffused in a small amount (f℥ss) of starch. This may be repeated after several hours, if necessary. Small injections of starch, with laudanum, without lead or other astringent, sometimes give important relief in dysentery.—H.]

In Malarious Dysentery quinine in full doses should be given, not less than a scruple in solution some time before the ipecacuanha, and repeated until cinchonism, as evidenced by ringing in the ears, is induced. Ipecacuanha and quinine should be given in alternate doses until the characteristic effects of both are produced. In the malignant Dysentery of camps our utmost efforts must be directed to improve the hygienic conditions of the sick, and in addition to the means already mentioned, carefully modified to suit the condition of our patients, the solution of the pernitrate of iron should be given in full and frequent doses. In some cases I have given ten drops every hour with advantage, and it may be combined with quinine, while the patient is at the same time sustained by milk in small quantities and at short intervals, with beef tea when it can be retained, with wine and brandy when required.

In Scorbutic Dysentery our utmost efforts must be directed to improve the condition of the patient's blood. It is in this form of the disease that fresh Bael fruit has been found so successful in Bengal and other parts of India. I have had occasion to prescribe this frequently in Madras to officers and others who had contracted Scorbutic Dysentery in the province of Pegu, and often with the best effect. Bael fruit has often fallen into disrepute as a remedy in Dysentery, simply from its indiscriminate use. My conviction is that where there is no scorbutic taint it is without efficacy. The Bael fruit is used in India in many forms, as a sherbet, a conserve, a marmalade, or an extract. The former is probably the most efficacious. Sir Ranald Martin in the *Lancet*, and Dr. Alexander Grant of the Bengal Medical Service, in the first volume of the *Indian Annals of Medicine*, have both written interesting accounts of the various modes of preparing and using this remedy. It is doubtless in the same form of the disease that the "Grape cure" has been found so efficacious. One caution I cannot help giving. I remember the case of a young officer at Secunderabad, who, while convalescing from Acute Dysentery, partook freely of grapes. Shortly after, he was seized with all the symptoms of peritonitis from perforation, and rapidly sunk. After death it was found that a grape stone had become entangled in one of the half cicatrized ulcers in the colon, where it acted like a pea-issue; a minute perforation resulted, causing the death of the patient.

Many of the invalids from India, suffering from chronic Dysentery, arrive at Netley in a more or less scorbutic state; all are benefited and some cured simply by causing them to use whatever fruits are in season. [The same was true of the U. S. A. *Chickahominy* cases, before alluded to.—H.]

Chronic Dysentery.—Whenever the disease falls into this stage and resists treatment, the patient should as soon as possible be sent to a better climate. If the locality be malarious, this should be done at once. Often moving him to the sea-coast suffices. More frequently a voyage to Europe is essential to recovery; many lives are lost by delaying this measure until it is too late, and many men are

embarked in such an advanced state of disease that they die after being a few days on board. As already stated, the chief mortality among Indian invalids on the voyage home is from Dysentery.

I have elsewhere (Army Medical Reports) insisted on the necessity of extreme care in the management, diet, and clothing of men suffering from the disease at sea. To men in this condition the salt ration is simply destruction, and unless they be warmly clothed on entering high latitudes, they are certain to have their sufferings miserably aggravated.

Whenever the symptoms assume an acute or subacute form, the patient ought at once to be placed in bed, and ipecacuanha should be given as recommended in the acute stage, in doses suited to the condition of the patient, and the violence of the symptoms. Gentlemen who have done duty in the clinical wards at Netley have often seen me use this remedy under such circumstances with the happiest effect.

I recommend the use of a water-belt over the abdomen for some hours daily. This acts as a fomentation, and the steady, uniform pressure it maintains seems to favor the absorption of the fibrine effused between the intestinal coats. If there be much uneasiness about the fundament, a water compress over the anus often affords more relief than opiate enemata.

The cold hip-bath should be used daily for a few minutes. I have seen this most serviceable. The greatest attention should be paid to diet. The proper nutrition of the patient is often the most difficult part of the treatment. In those cases where atrophy of the small intestine is much advanced, with perhaps fatty or amyloid degeneration of the liver, or both, it is almost, if not quite, impossible, and the patient dies starved. If the state of the gums point to a scorbutic taint, the diet must be regulated on the principles already laid down under that head. In extreme cases milk must be our chief resource, sometimes with a little lime-water, or beat up with egg, and good sherry or brandy; a teaspoonful of curaçoa added is often highly relished. Rice flour, sago, arrowroot, or stale bread may be added, and changed so as to suit the capricious appetite. When solid food can be taken without suffering, it should be given; tender mutton should be broiled "quickly and not too much," and eaten with bread and butter. These rules are quite as applicable to the acute stage, where food, while the acute symptoms last, should be simple and farinaceous, and the return to solid food should be gradual.

With regard to medicine, where astringents are indicated, they should clearly be of such a kind as do not tend to lower the already sufficiently low state of the patient. Acetate of lead, sulphate of copper, and suchlike remedies, I use with a sparing hand, and only under the pressure of necessity. My favorite remedy, particularly in men returning from tropical regions, anæmic from loss of blood and the depraving influence of malaria, is the solution of the pernitrate of iron, which I use at Netley very freely, and often with the happiest effect. Under this remedy the whole system often rallies wonderfully, the condition of the blood improves, color returns to the blanched cheek, the stools become more natural and less frequent, the appetite improves, and digestion is more perfectly performed. The citrate of iron and quinine may after a time be substituted.

When astringents of a more direct kind are necessary, the decoction of logwood with lime-water, catechu, gallic acid, and suchlike may be used. Pain must be allayed by opiate enemata, gentle douches to the anus are often most soothing, and the use of liniments containing chloroform often allays irritation. [In obstinate chronic Dysentery, with evidence of ulceration of the intestine, enemata containing metallic astringents are sometimes very useful. In the Pennsylvania Hospital, in 1847–48, a number of soldiers returning with severe chronic Dysentery from the Mexican war, were cured by the use of injections of sulphate of zinc. Even large doses (gr. v to x in f℥iv of flaxseed infusion) were well borne in those cases. Strong solution of alum sometimes produces a similar beneficial effect.—H.]

In Conclusion.—It will not fail to be remarked that I have not only not advised but by implication have deprecated the use of mercury in all stages and forms of the disease.

My first objection to the use of mercury, particularly in military practice, is one that was forced on me more than twenty-two years ago by observing this fact. Whenever soldiers find that in addition to the misery inseparable from an attack of Dysentery they are to be subjected to that of ptyalism in its mildest degree, they will not present themselves at the hospital gates until further concealment of the complaint is impossible. Secondly, because in ipecacuanha we have a remedy that effects all that mercury can do as an evacuant, without its irritating effects. Thirdly, because experience has shown that men "cured" by mercurial treatment are as a rule cachectic, ex-sanguine, prematurely old-looking, extremely sensitive to atmospheric changes and to relapses from trivial causes. Fourthly, because chronic Dysentery is more frequent after mercurial treatment than

when the disease is treated by ipecacuanha. And, lastly, "because men actually under the influence of mercury are very predisposed to the disease." (Morehead.)

In many text-books and works of high authority, blood-letting is still insisted on as an essential part of the treatment of acute Dysentery.

I respectfully dissent from this doctrine, first, because, although from the violence of the symptoms there is an appearance of *power*, this is deceptive, for alarming depression often follows free depletion. Secondly, even where the measure appears to relieve symptoms, the heart's action is weakened to such an extent that congestion of the affected mucous membrane remains, a condition which Dr. Blacklock has shown to be nearly as destructive to the tissues as the more acute action. Thirdly, because convalescence after bleeding is tedious. Fourthly, because although bleeding has fallen into disuse the mortality from Dysentery has decreased. Lastly, it appears to me that the authors who still urge it in this affection and malarial fevers take no account of the state of public opinion on this question. Whatever may be the case elsewhere, a generation has certainly arisen in India that knows not the lancet. Men know, on the other hand, for they see it daily, that those who are not bled recover in a larger proportion than in the days when bleeding was the rule of practice. In short, out of a military hospital where patients have no option but to obey, I assert that it would be simply impossible to carry out the rules for blood-letting which still stand in some of our text-books. I cannot help thinking that treatment of this kind is much in the position of certain penal statutes, which, although still in the statute-book, have become obsolete by the force of enlightened public opinion. I am confident that were it possible even for Robert Jackson, the most sagacious and far-seeing physician the British army ever produced, to reappear once more on the scenes of his former labors, it would be impossible for him, with all the influence of his great name, and all the authority of his vast experience, to induce the men and women of the present day to submit to treatment which they believe to be mischievous, and know to be unnecessary.

[Acute inflammatory Dysentery occurs, however, sporadically, in climates free from endemic or epidemic influences, so sthenic in character, that those who are willing to resort to local, and, in a few cases, general blood-letting, may find advantage in it. Application of leeches, rather freely, to the lower portion of the abdomen, has been known to give very important relief and aid in treatment.

In the same class of cases, some practitioners have reported excellent effects from the use of small but repeated doses of a solution of sulphate of magnesium. By its diffusive action over the intestine, promoting secretion from the congested mucous membrane, and also by its sedative influence upon the circulation, we may explain the benefit which, empirically, has been obtained from it.—H.]

EPIDEMIC CHOLERA.

By Edward Goodeve, M.B.

Definition.—An epidemic, and in some places an endemic disease of great mortality. Typical Epidemic Cholera is characterized, in its developed stages, by vomiting and purging of watery fluid; by rapidly causing a state of the body called collapse, in which there is extreme depression or diminution of nearly all the functions of life; by terminating in death—often within twenty-four hours from the first symptoms of the disease—or in healthy reaction, or in various dangerous sequelæ, mostly of a typhoid nature.

Synonyms.—Cholera Morbus—Epidemic Cholera—Asiatic Cholera—Algide Cholera—Cholera Asphyxia—Cholera Spasmodica—Blue Cholera—Malignant Cholera.

The disease called by the above names is a severe epidemic affection, well known in India, and which has at intervals ravaged different parts of Europe, Africa, and America. In Calcutta and Bombay it prevails so constantly that it may now be said to be endemic there. It appears frequently in our camps and stations in India, and its occurrence therein, in violence, is a signal for unwearied toil and labor to our regimental and other surgeons, and of anxiety to all. And it may well be so, so widespread is its diffusion,

so destructive are its effects, and so resistant is it to treatment. It demands, indeed, the careful study of all who practise medicine, and most especially of those who pursue it in our Indian territories.

HISTORY.—In a practical sketch such as this, it would be vain to occupy space and time with inquiries as to whether Cholera was, or was not, known to ancient writers. There is little room for doubt that our forefathers were acquainted with it in Europe. In India, Cholera has been observed several times since the English have had possession there. There is evidence of its having been known in Madras in 1769, 1770, and 1774. It attacked the artillery commanded by Colonel Pearse, marching to join Sir Eyre Coote, in 1781, and the troops under Colonel Cockerell, in 1790. Its occurrence in three or four places in different parts of India, in the beginning of the present century, is well authenticated. The epidemics, though apparently sometimes severe, do not seem to have been very frequent. Independently, however, of old records, we have a prominent starting-point for the history of Cholera in 1817, since which time it has been frequently prevalent. In 1818 the Western world was startled with the intelligence of the appearance in India of a disease which was ravaging Lower Bengal, and had also attacked the camp of the Marquis of Hastings, then engaged in the Mahratta war, and who was at that time halted on the banks of the Sind, in the Upper Provinces. A new disease, or at least one unknown in such a terrific form, was carrying destruction through all the ranks of the army, both European and native. The scourge appeared in Lord Hastings' camp on the 6th November, 1817, and in five days destroyed 5000 men. In it, in all, 9000 deaths occurred. But not in the camp of war only did it cause surprise and terror. After having shown itself during the previous months in Mymunsing, Patna, Kishnaghur, Chittagong, and some other places, it burst out in August, 1817, in the agricultural province of Jessore, among the peasants and laborers of the rice swamps and palm groves. Many thousands were swept away by the pestilence in the course of a few weeks. There might have been such a disease in the mist of past ages, but the memory of living man possessed no vivid or substantial knowledge of it. It burst upon the suffering generation with the violence of an unheard-of plague; impressing all with dread and consternation.

From this starting-point in India, Cholera spread east and west, far beyond the bounds of Hindostan. Its appearance in other lands may be traced with tolerable accuracy. From Bengal it spread eastward and southward in the following chronological order. We find it in

1818, in Burmah, Arracan, and Malacca;
1819, in Penang, Sumatra, Siam, Ceylon, and the Mauritius;
1820, in Tonquin China and China;
1822, '23, '24, in all China;
1827, in Chinese Tartary.

Turning to the west, we find it, in

July 1821, at Muscat and the Persian Gulf; in
1822, in Persia, and prevailing there during 1822, '23, '29, '30; and in 1823, at Astrachan,

without spreading further westward for some years, *i. e.* until 1829, when it reached Orenburgh through Tartary, revisited Astrachan in 1830, and from thence started on its course through Europe.

The westward course continued slowly. In May 1831, it was very severe at Moscow and Warsaw; in July of the same year at St. Petersburgh and Cronstadt; in October, at Berlin and Vienna. In England the first cases showed themselves at Sunderland, in October 1831, and the epidemic prevailed in the British Empire for fourteen months. It crossed the Atlantic and reached Quebec in 1832. This fatal malady ravaged the whole of Europe, and left that quarter of the globe in 1837, the last place affected being Rome. Since 1817, epidemics of Cholera have been frequent all over India, so that the disease may be said to have been naturalized there; causing a large mortality among all classes.

Besides the first great epidemic above mentioned, the western parts of the world have suffered from two severe visitations of Cholera, viz., in 1848–49, and in 1853–54. These appear to have travelled from the East much in the same manner as that of 1832.

[In 1866, Europe and America were again visited. In 1868 it was particularly severe in S. America. In 1872, and again in 1873–4, it was destructive in Hungary, Poland, and Prussia. In 1873 it was the cause of great mortality in several towns in the Mississippi Valley. Yokohama, Japan, and Canton, China, were severely visited by it in 1877.—H.]

Thus Cholera seems to have spread east, south, west, and north from its first birthplace in Bengal, which became but the centre of an epidemic area comprising nearly all the world. It travelled slowly at first, and not continuously but in irregular waves, checked sometimes, but not destroyed, by winter cold, Neither climate, nor season, nor earth, nor ocean seem to have arrested its course, or to have altered its features. It was equally destructive at St. Petersburgh and Moscow as it was in India; as fierce and irresistible

amongst the snows of Russia as in the sunburnt region of India; as destructive in the vapory districts of Burmah as in the parched provinces of Hindostan.

Etiology.—The predisposing causes of Cholera are doubtless common to some other epidemics. The exciting cause is, probably, an aerial, or at all events an airborne poison, and probably of the zymotic class. We do not know, however, whether it is of an organized, organic, or inorganic nature. There are many circumstances in favor of its being of organic composition, some of its being organized. It has hitherto eluded all chemical and microscopic research. We are much in the dark as to its origin, preservation, multiplication, or diffusion. The poison is generally supposed to be some addition to the ordinary atmosphere, but some writers have speculated upon there being merely some modification of its ozone or electricity, or upon some dynamic change. Others have sought for an origin in telluric influences. In considering the causation of Cholera, we must bear in mind that we have to account for a disease spreading quickly over large areas, often preceded by epidemic diarrhœa; frequently developing itself with little warning in the places attacked, and often disappearing suddenly and returning to them after brief intervals, remaining absent for many years; visiting with great regularity the same places on each return; sometimes limiting itself with singular abruptness in certain localities, passing over places in its route with strange capriciousness, and afterwards returning to them, spreading sometimes with and sometimes against the direction of the prevailing winds.

It will be well, apart from all theory, to consider the various circumstances which appear to have some connection with the development of Cholera. These may be divided into those which act upon the individuals in masses or groups, and those which belong to the individual only. To the first belong meteorological conditions, climate, soil, purity of atmosphere, food, water, &c.; to the second class, sex, age, occupation, &c.

Atmospheric Conditions.—No solution of the question of the cause of Cholera can be found in ordinary atmospheric changes. The opposite states of heat and cold, humidity and dryness, high and low barometric states, &c., have prevailed or been excluded without banishing the disease. A certain amount of heat seems to favor the spread and severity of the epidemics. In Bengal the hot seasons, including the hot and dry and rainy seasons, have witnessed the worst epidemics. In Bombay, Dr. Ewart's tables (Vital Statistics of the Armies in India) show that for eight years the most fatal cholera months for European troops were from April to September; the admission to strength, and the mortality to treated, being much higher: the percentage of fatal cases to treated giving 50·710 per cent. from April to September, and 19·510 per cent. from October to March: and in 3676 admissions during seventeen years, April to September gave 2918 cases, and October to March 758 cases. Dr. Morehead shows that the greatest number of admissions into the Bombay European General Hospital took place in the months from April to September inclusive. He mentions, however, that Dr. Leith gives rather different results for natives. In his tables of mortality in Bombay for the three years 1848–52, he reports 7112 deaths from October to March, and 5110 from April to September. Dr. Hugh Macpherson has shown that, in Calcutta, Cholera is always most rife in the hot months, both for Europeans and natives; the severity of the disease generally rising from January, through March and April, and descending from that month to August, when it is at its lowest. In the recently published Report of the Commission appointed to inquire into the Cholera Epidemic of the Northwest Provinces of India, in 1861, it is shown that the curves of the disease for the Northwest Provinces do not quite agree with those of Bengal, as will be seen presently. In Europe, temperature appears to have had an influence. In England, in 1848–49, the highest point of the curve line was in September. In 1853–54, Mr. Glaisher and others record that the greatest violence of the disease was during the months having the highest temperature. Although the greatest mortality in India is in the hot weather, there is a considerable amount of it in the cold seasons; much less, however, in the cold seasons of the Northwest Provinces than in those of Bengal. In Europe also it prevailed somewhat severely in the winter of 1848–49, and very severely in Russia during the winter of 1830–31. The circumstance of the Russian houses being kept very warm inside hardly accounts for this. If Cholera were extinguished by cold, it is difficult to see how this action could have been neutralized in entire districts by the warming of the insides of the houses. It appears, from the above, that Cholera exists under very wide ranges of temperature, but that nevertheless it is probable that a certain amount of heat does contribute to its intensity.

More persons are attacked in the early morning than during the rest of the day. The temperature of the individual may be lowered during those hours, and this may have the effect of predisposing him to suffer. Possibly the lower temperature of the air may cause a greater concentration of the poisonous atmosphere near the

ground at that time. The tendency of attacks to commence in the early morning has been noticed in Scotland by Dr. Adams, and in India by Mr. Twining, Dr. Morehead, Sir R. Martin, and others.

Rain and Moisture.—These do not prevent the spread of Cholera. It prevails most in Calcutta in the dry season. It does not do so in the Northwest Provinces of India, in Bombay, or in Madras. The Northwest Provinces have at times suffered severely in the hot and dry months, but the majority of the epidemics have been in the wet months, as shown by the Report of the Commission of 1861. Singularly enough, though the dry months are now the most fatal in Bengal, the great and desolating outburst, which startled the whole world, ravaged Jessore in August, 1817, in the midst of one of the wettest seasons known. The combination of heat and moisture, when the air is moving slowly, or not at all, seems very favorable to the spread of Cholera. Thus a prevailing hot, moist, and stagnant atmosphere during these epidemics has been recorded by many writers. This is mentioned by Mr. Thom in his report of the memorable outbreak of Cholera at Kurrachee in 1846. He states that the dew-point was very high, 83°, with thermometer at 96° in the shade; and there was induced a sense of languor and oppression, a stifled feeling about the respiration, and inability to undergo the least fatigue. This sense of languor and oppression is often felt during the lulls in the rains in India, independently of Cholera times, and therefore must not be considered causative of the disease. A warm, moist, stagnant atmosphere in Bengal, at any time of the year, is often followed by sporadic cases of Cholera, or by an increase of cases where the disease is endemic. Cholera has been known to cease after heavy falls of rain, and, on the other hand, to set in immediately afterwards, as in the Meerut gaol in 1861. In considering the influence of rain, we must recollect the temperature prevailing, and the time that has elapsed after the said fall. A very few hours after a fall of even heavy rain, if there be no wind, is sufficient to produce just the stagnant, hot, and moist atmosphere which is so oppressive to the feelings, and favors so much the spread of Cholera. One must not be misled by the name of dry months. The hot months in Calcutta are called the dry months, and are indeed the driest of the year, yet a great deal of moisture exists dissolved in the warm atmosphere; absolutely a greater amount of moisture in the air there than in England. In 1853–54 the atmosphere in England was drier than usual, for every month except May and December, the rainfall being 18·62 inches, or 5·93 less than the average.

Winds.—The only way in which these seem to influence the spread of Cholera is when they blow over places charged with miasma. It has appeared to prevail in certain situations when the air came across foul places, as privies, cesspools, &c., and shifts of wind have been known to have been followed by subsidence of the disease. It declined after a hurricane which took place in Madras in 1818. In Europe there was no fixed relation between the quarter from which the wind came and the intensity of the disease. Though Cholera appears to travel with the wind in many instances, it does not always do so. Orton reports that it travelled across a great variety of country from the Nerbudda to Bombay, directly opposed by a strong wind blowing night and day for half the year, at the same rate of progress that it passed from Madras to Cape Comorin, with the breeze in its favor. Absence of horizontal movement of the air, or a stagnant atmosphere in combination with heat and moisture, has already been alluded to.

Electricity — Ozone — Barometric Pressure.—These have not been found to exercise any decided influence, or to have existed in different conditions during different epidemics. Most extended observations on some of these points, by Mr. Glaisher, are to be found in the appendix to the Cholera Report for 1853–54, and in the Report of the Indian Sanitary Commission of 1862.

Much has been written on the influence of meteorological states upon the spread of Cholera, and there is much confusion and apparent contradiction in the results and on the inferences drawn. Some connection between atmospheric states and the epidemics does exist, but it is not that of cause and effect. It is very probable that heat and moisture, which are so favorable to most organic actions and to chemical changes, may promote the multiplication of the Cholera poison, and that a calm stagnant atmosphere may allow of its concentration. We do not look upon heat and moisture as the cause of a tree or plant; but we find them very influential in their developments. Mr. Glaisher's observations in concluding his report on the Meteorology of London in relation to the Cholera epidemic of 1853–54, allude to those of 1832 and 1848–49, and show that the atmospheric conditions during the prevalence of Cholera are well worthy of attentive study. He says: "The three epidemics were attended with a particular state of atmosphere, characterized by a prevalent mist, thin in high places, dense in low—during the height of the epidemic; in all cases, the reading of the barometer was remarkably high and the atmosphere thick. In 1849 and 1854 the temperature was above its average, and

a total absence of rain, and a stillness of air amounting almost to calm, accompanied the progress on each occasion. In places near the river, the night temperatures were high with small diurnal range, a dense torpid mist, air charged with many impurities arising from the exhalations of the river and adjoining marshes, a deficiency of electricity, and, as shown in 1854, a total absence of ozone, most probably destroyed by the decomposition of the organic matter with which the air in these situations is strongly charged."

"In 1849 and 1854 the first decline of the disease was marked by a decrease in the readings of the barometer and in the temperature of air and water; the air, which previously for a long time had continued calm, was succeeded by a strong southwest wind which soon dissipated the former stagnant and poisonous atmosphere. In both periods at the end of September the temperature of the Thames fell below 60°; but in 1854 the barometer again increased, the air became again stagnant, and the decline of the disease was considerably checked. It continued, however, gradually to subside, although the months of November and December were nearly as misty as that of September."

Climate.—We are not in a position to estimate the precise value of climate on the intensity of the disease. The statistics of the native populations of India and of nations of the Eastern part of the globe are not sufficiently well known to enable us to compare the virulence of their epidemics with those of Europe. For India, we have only the military statistics on which we can rely, but they are of too exceptional a character to serve the purpose of the inquiry. Whatever may have been the virulence of separate epidemics, there is a much greater frequency of the epidemic in India. Whereas Cholera has visited Europe only twice severely since 1832, the epidemics are of constant occurrence in some part or other of India. It is doubtful whether there is any relation between the marsh malaria of a country and Cholera. The question of the influence of climate will be again referred to in the section on mortality and susceptibility.

Nature of the Soil.—Cholera has prevailed so severely nearly all over the world, that it is not likely that the soil exerts much influence. Some writers have thought that it has been less severe on the Laterite foundations, in Madras and other places. Dr. Maclelland states that stiff clayey soils have had more mortality than loose sandy and easily drainable ground. Dr. Lorimer found that nearly half the epidemics examined took place on the black cotton soil. The volcanic formations of Auvergne escaped in 1842, although surrounded with Cholera fields. Dr. Farr states that in England it was less fatal on the primary geological formations than on others.

Elevation above the Sea Level.—The most favored seats of Cholera all over the world are places not high above the sea, along the banks of rivers, and the estuaries of great streams. In London, the lower elevations were most fatal; and as the height of the locality increased, mortality decreased. Doubtless this is not from any difference of barometric pressure, but because these situations generally combine so many unfavorable sanitary conditions, as the most moist subsoil, the worst drainage, the least ventilation and air movement, the most impure air, and the most dense populations. In London and its neighborhood the mortality was at the rate of 156 per 10,000 in the lowest districts, viz., Newington, Rotherhithe, St. George's Southwark, and Bermondsey, about the level of the Thames; and 15 per 10,000 in the highest, viz., Hampstead, Islington, Marylebone, and St. Pancras. There were some exceptions in places in which all sanitary conditions were perhaps worse than some of the places of a little lower level. Cholera is less prevalent in mountain elevations than on low levels. This is the case in the Indian hill stations. They have not, however, been exempt from severe visitations. Dr. Chevers mentions, in his "Review of the Means of Preserving the Health of European Soldiers in India," that it prevailed in 1845 at Kussowlie, 6000 feet above the sea, and at different periods at Murree, Dhurmsala, Darjeeling and Jackatalla, 6000 to 7000 feet high and in lower elevations at Soobathoo, Hazeerabagh, and Mabaleshwur, 2000 to 3000 feet above the sea.

Impure Air.—There is abundant evidence that a foul atmosphere promotes the severity of Cholera. For copious illustration of this and the numerous sources from which the atmosphere may be vitiated, the reader is referred to the various reports of the Board of Health on the occasions of the English epidemics. Among others, the evil influence of privy emanations has been frequently noticed; and the same evil influence is obvious in the Report of the Indian Cholera Commission for 1861, in the instance of the epidemic in the cantonment of Meean Meer. On the other hand, it is found that in many epidemics some very foul and filthy places escape altogether. Thus, the filthiest parts of Lahore and Gwalior escaped, while the cantonments of Meean Meer and Morar, close to these cities, were ravaged by Cholera. The immunity of the filthy Ghetto at Rome is another instance. In some instances places with

apparently the purest atmosphere did not escape, as, for instance, in 1849, the healthy parts of Middlesex, Hertford, Buckinghamshire and Kent. Nevertheless, in spite of exceptions, the places in which the air is most vitiated from privies, cesspools, drains, decaying animal and vegetable refuse, or overcrowding and concentration of human emanations, are those in which Cholera has generally been most fatal and most widely spread.

Impure Water.—This, doubtless, plays an important part in Cholera epidemics, either as a predisposing, or, as some think, as an exciting cause. Dr. Snow thought that the poison was produced in the alimentary canal, and existed in the Cholera evacuations, and that these, through leakage of drains, cesspools, &c. contaminated water, which when drunk communicated the disease. Whether this be the manner in which Cholera is caused by impure water or not, it is certain that this promotes the spread of the disease. Strong evidence of this has been collected by Dr. Snow and Mr. Simon. The latter, in his report on the last two epidemics of London as affected by impure water, gives us a statement of the mortality among the consumers of water, supplied by two companies drawing their water from distinct sources, but distributing it in the same district, at the same time, and among the same class of people; the pipes of the two companies being laid pretty evenly in the same areas, in many places running side by side in the same streets, and the houses supplied pretty equally distributed. The Water Companies were the Lambeth Water Company and the Southwark and Vauxhall Company. The first drew their supply at Ditton, above the influence of the London sewage and tidal flux, the last from the river near Vauxhall and Chelsea. The Lambeth supply was tolerably pure, the Vauxhall Company's very impure. The deaths in the houses supplied by the Lambeth Company were at the rate of 37 to every 10,000 living; in those supplied by the Southwark and Vauxhall, at the rate of 130 to every 10,000 living. The population drinking the foul water appears to have suffered 3½ times as much as that drinking the pure water. This seems to be an experiment as free as possible from error; the population submitted to the test being 400,000—500,000; the only ascertainable differences of circumstances in the two classes being in the kind of water supplied. The well-known instance of the sickness following the use of the water of a pump in Broad Street, Golden Square, affords strong evidence of the evil influence of water contaminated with cesspool drainage. Dr. Routh attributes the severity of Cholera in the winter in Russia to the drinking of water from melted snow collected from the immediate neighborhood of dwelling-houses and on which the Cholera discharges had been thrown. Impure water alone will not necessarily produce Cholera. There are instances of freedom from the disease with bad water supply in the same way as there are with vitiated atmospheres already mentioned.

Bad Food.—Mr. Granger, in his Report on the Epidemic of 1848–49, says, "Several marked examples were brought under my notice where violent attacks of Cholera were distinctly traceable to the use of putrid fish, bad pickled pork, decayed cheese," &c. Dr. Carpenter quotes Dr. Brittan as authority for the fact of an outbreak of Cholera and Choleraic diarrhœa among a number of school children, who had eaten plentifully of spoiled oysters, and by which eleven of the sufferers lost their lives. In the earlier days of the appearance of the disease in India it was thought that many attacks were caused by the eating of diseased rice. I remember the case of a gentleman who was attacked a few hours after eating hermetically sealed fish. In this class of causes may be included the noxious effects of purgative medicines given during Cholera. Numerous Indian writers recognize the mischief produced by these. I believe that this is not confined to the saline and hydrogogue purgatives only. I have seen milder purgatives followed by Cholera. Sir R. Martin and Twining caution us against administering during Cholera times any purgative medicines likely to operate in the early morning; *i. e.*, about the time that the first symptoms of Cholera generally commence.

Regiments marching.—In Madras these seem to have been very liable to Cholera, as shown by Rogers, Lorimer, and Balfour. From Dr. Rogers's Report on the Asiatic Cholera in the regiments of the Madras army from 1828–44, this is shown both for European and native corps. Large bodies or regiments or parties of European recruits *en route* to join their corps have suffered greatly. On the other hand, treasure parties, consisting of smaller numbers of men, from ten or twenty to a maximum of 100 or 200 men, have suffered little. Thus in the European corps the ratio of marches attacked to marches unattacked has been 23·92 per cent.; in the small parties 2·72 per cent.; the officers being comparatively exempt. Dr. Lorimer's reports go to prove that long marches were much more fatal than short ones, and that there is a ratio between the distances marched and the attacks. In Madras, in the native troops, the attacks were 46 per cent. between 600 and 800 miles, and 75 per cent. between 800 and 1250 miles marched. Dr. Rogers attributes the difference of susceptibility to fatigue, exposure, crowding, and other

predisposing causes which act more severely for reasons shown on large than on small bodies of moving troops. The adherence of Cholera to troops moving seems at variance with the beneficial effects so often experienced on moving troops out of infected cantonments in Cholera epidemics. The difference is probably due to there being less fatigue, less crowding, better conservancy, and more hopefulness and cheerfulness on the part of men leaving infected cantonments for a short time, than in the case of ordinary marches. It has been known that a regiment suffering severely from Cholera in camp on the march has lost it on getting into barracks. A remarkable instance of this occurred in the case of H. M. 63d Regiment, which suffered extremely during the greater part of its march from Poonah to Bellary, but entirely lost the disease in two or three days after getting into barracks in the unhealthy station of Bellary. In this case probably extreme heat in tents, overcrowding, and fatigue in the month of April acted as powerful predisposing causes. H. M. 86th Regiment, which suffered so severely at Kurrachee in 1846, had come off a long and fatiguing march from Upper Scinde.

Influences belonging to the Individual.—*Sex.*—This has but little influence. In London in 1849 the percentage of deaths to living was ·67 in the male and ·65 in the female; in lunatic asylums 6·4 for males, and 6·6 for females; the attacks 8·9 for males and 11·6 for females. In India we can depend on the returns of the European corps only, and the numbers are probably too small to be relied on to show the comparative liability of the sexes. Ewart's tables give privates 1·74 per cent., and women 1·58 per cent. admissions to strength, and among privates ·70 per cent., and women ·50 per cent. of deaths to strength. *Age.*—Mr. Grainger states that the liability to fatal attacks increases after the age of fifty in both sexes; the ages from five to forty-five having the lowest comparative mortality. In India in 1861 the influence of age was hardly perceptible. According to Dr. Gull, in England it was most fatal to those under one year and over fifty-five, corresponding, indeed, with the general tendency to mortality in England.

Residence in India.—It has been thought that Europeans of short residence in India were more liable to suffer than those of long residence, but the opinion does not seem to have been formed upon any well-ascertained facts. The Committee on the epidemic of 1861 investigated the matter for that outbreak, but they consider their data too imperfect for forming opinions. They say, "We can base no conclusions upon the figures shown in this table. The soldiers whose period of service was shortest apparently suffered the most. The proportion of deaths to strength falls from 7·7 per cent. among men of less than two years' Indian service to 3·5 per cent. among men who have served more than ten years. . . . The proper investigation of this subject must be left to future observers. It will probably be found that the influence exercised by length of residence in India upon the liability to Cholera is as little important as that of the other predisposing causes that have been already noticed."

Previous Health.—Both the strong and the weak are susceptible of Cholera. Mr. Thom states that some of the most robust men of the 86th were struck down at Kurrachee. There are often seen in the Calcutta hospitals, cholera-stricken, the stout, rosy, muscular Affghan, as fine specimens of men, in bone and muscle, as can possibly be conceived. Doubtlessly previous debilitating disease influences the mortality. It is not so certain that it greatly increases the susceptibility, though Dr. Morehead mentions that cachectic and debilitating diseases appeared to have had a strong predisposing effect on the attacked in the hospital at Bombay. Scurvy and diarrhœa have probably some predisposing influence. In Dr. Gull's report it appears that little predisposition was caused by previous disease, and Dr. Gairdner mentions that the post-mortem examinations in Edinburgh showed very little disease in the bodies of those who died there of Cholera. In India, in 1861, the sick in hospital furnished 13·5 per cent. of cases to strength, while the men in barracks afforded but 4·7 per cent.; but there were probably other causes than mere predisposition at work in this instance.

Habits.—I believe that these have less influence upon the susceptibility to Cholera than has been supposed. Persons in whom habits of intemperance have established organic disease of liver or kidneys may be less liable to recover when attacked, but the real degree of susceptibility engendered by intemperance has not been completely ascertained. In India in 1861 the intemperate were more subject to attacks than the abstemious, but their chance of recovery in the attack was better. However, the numbers tested were small.

[In the United States, if not elsewhere, it was well proven in 1832–3, 1849–50 and 1854, that, whether more or less susceptible than others to Cholera, drunkards were more likely to die from it than any others.—H.]

Occupation.—This seems to produce no special liability; but of course those which expose the individual to unhealthy influences and residence may increase his risks. Thus the privates and non-commissioned officers of regiments suffer more than the

officers. Fatigue, want, grief, fright, have doubtless some degree of predisposing influence, though it would be difficult to estimate the amount in figures. Of these, fatigue is probably the most injurious.

Probably most of the causes mentioned above may be considered as predisposing causes. Impure air and water may convey the exciting cause, but this will be presently referred to. None of these in themselves appear to be sufficient to produce Cholera, though they may all render it more fatal. The existence of all the conditions mentioned, both before and after Cholera epidemics, without producing the disease shows that some special agent or cause must be present to give rise to it. Before entering upon the question of this specific cause it will be well to mention some points in the natural history of Cholera not yet spoken of.

Health of Communities before and during Cholera Epidemics.—In Europe a prevalence of several zymotic diseases has been noticed in these periods; particularly typhus fever, influenza, and diarrhœa. The increase of fluxes was so great previous to the outbreak of 1848 that Cholera in England was looked for long before its arrival. There was also an increase of typhus fever. Dangerous and fatal influenza preceded the epidemics of 1832 and 1848. Diarrhœa is constantly present during Cholera. In London, for the quarter ending September 30, 1849, notwithstanding the large mortality from Cholera, the deaths from typhus fever were nearly equal to those of the four preceding corresponding periods; themselves of remarkably high mortality. In India, diarrhœa constantly prevails during epidemics, but the precedence and concurrence of other zymotic diseases is not so well made out. Cholera, however, does not seem to banish them.

From the above we seem justified in inferring that circumstances similar to those which favor Cholera promote also the spread of the other zymotic diseases.

Limitation of Areas of Cholera. — This is sometimes singularly abrupt. It has been known to attack persons on one side of a street, of a camp, or a town, only. In 1848–49 it attacked one side of a small village in Argyleshire, and confined itself exactly to one side of the town, which consisted only of one main street, divided into two equal parts by a toll bar, beyond which to the west not a single case occurred, though the inhabitants were in constant intercommunication. Dumdum, seven miles from Calcutta, often suffers severely, while Calcutta does not, and *vice versâ*. One part of a ship may suffer and the other not. A wing of a building may be ravaged while the rest escapes. The limitation to districts is sometimes remarkable, and was shown in the case of the 9th Lancers on its passage up the Ganges in 1842. The regiment travelled in boats, by wings, and at separate times. When the left wing reached Monghir, although there had been no communication with the shore, Cholera broke out in it, and continued with the detachment for about twelve days, until it had got beyond the infected districts. When the right wing reached Monghir, it, also, was attacked, and lost the disease exactly at the same place that the other did. The limited area of infection has been tested by the good effects of moving troops into camp when suffering from the disease. Numerous instances of this are on record; among the latest some are afforded in the Cholera Commission Report for the outbreak of 1861. A move of a few miles is generally sufficient.

Mode of Invasion of Localities.—We find that both in India and Europe it is often preceded by diarrhœa, sometimes for months, as in England previous to the outbreak of 1849. Then a few cases of Cholera appear, and in a short time the epidemic sets in severely. At other times the extreme violence shows itself rapidly and suddenly after the prevalence of a few cases for a few days, as at Kurrachee. Of this outbreak Mr. Thom says: "It suddenly burst forth in a few hours in every European regiment, whether in camp or in barracks, in every tent or in every house, and it was at its acme in forty-eight hours afterwards, when instead of spreading further it gradually and steadily declined. Now, it appears that for some days or even weeks a few cases had appeared in the native town of Kurrachee, but there also, at the same period, the malady became *suddenly* general over the whole place." In Paris, in 1832, Dr. Baly relates, that in eighteen days from its commencement it had reached its climax, and had already extended to all the quarters of Paris, and had been fatal to 7000 people. In Lord Hastings's camp in 1817, 5000 people died within the first five days of its appearance.

Departure of Cholera Epidemics.—These often leave a place rapidly, sometimes after sudden atmospheric changes. At Dumdum, in 1859, Dr. Hugh Macpherson reports that a violent outbreak occurred and carried off one-sixth of a detachment of recruits and their families in ten days, confining itself entirely to the barracks; it then disappeared as suddenly as it came. In the 2d Madras Europeans, as before mentioned, the disease continued for a month in hot and sultry weather, and disappeared, also, as suddenly as it came. The occasional sudden departure after high winds and storms has been already alluded to. Generally the decline of Cholera is more gradual, the intensity

of the mortality diminishes, the recoveries to attacks are more numerous, and finally the disease disappears. In England in 1848–49 the epidemic was about three months from the commencement of its decline to its final disappearance from the country; having prevailed about sixteen months. Cholera is apt to leave a place for a time, and then return to it, and be as severe on its second as on its first visitation. In 1848–49, Cholera prevailed slightly at Sunderland from October to January, was absent for a month, returned with severity, subsided for two months, and returned a third time with still greater violence. The epidemics do not break out simultaneously all over a country, but there is, nevertheless, often the beginning of the disease in several distant places at the same time; thus, the epidemic of 1848–49 first showed itself on the same day in Edinburgh, Sunderland, and Hounslow. It broke out at Malta, Palermo, and Gozo at the same time. It appears that Cholera epidemics have a tendency to reach their climax over large areas at about the same time. Dr. Baly states that the period of the greatest intensity of the epidemic of 1848–49 was, in the majority of cases, in August and September; thus, of 226 places affected, 61 suffered most in August and 118 in September, or 179 out of 226, in those two months. Also that in large towns or cities there was the same general approach to simultaneousness, as appeared in a comparison of different counties and of different towns one with another. Occasionally in epidemics a smaller climax is noticed, and there also is a simultaneousness in the period of this; that for 1848–49 was in January, 1849, when a decline in the intensity of the disease took place pretty generally.

Cholera does not spread uniformly over a large area or country; it seems rather to spread from certain centres first attacked, often passing over places in its apparent course, and perhaps subsequently invading them. Some places indeed, although in the track, escape altogether. It has a tendency, even after long intervals, to return to certain haunts in the same towns; thus, certain places which suffered greatly in 1832 were equally attacked in 1848–49, and some remarkable coincidences occurred in illustration. It has happened that the very first house invaded in 1832 gave the first cases in 1848.

Protection by previous Attacks.—These do not appear to confer any immunity; there are numerous cases on record of persons who have had Cholera more than once. Some writers have speculated upon the susceptibility of individuals being diminished by long exposure to Cholera atmospheres without being attacked. The opinion does not seem to rest on any good foundation.

Channels of Introduction into the System.—Being ignorant of the nature of the poison, we cannot well say how it enters the blood; it probably enters through the respiratory or intestinal surfaces.

Incubation.—It seems that this may be very short for many cases; perhaps two or three days, or even less. Of course if we include the Choleraic diarrhœas and those cases which show other preliminary disturbances, mentioned hereafter, we must allow a much longer time. In the case reported by Dr. Barry, and referred to in the paragraphs on Contagion, the period of incubation does not seem to have exceeded forty hours. When Cholera breaks out in ships at sea several days after leaving port, the attacks are probably due to freshly acting causes. [Especially when (as happened with a number of vessels in 1849–50 between Europe and America) the outbreak commences upon a vessel which has been as much as ten days or two weeks from land, and when no Cholera existed at its port of departure, the only rational explanation is that the Cholera cause, being migratory independently of human transportation, was present in the atmosphere through which the vessel passed in its voyage. One of the Copes' line of packet ships between Liverpool and Philadelphia was so attacked at sea in 1850. On the day on which the greatest number of new cases and deaths occurred, a large iceberg came in sight, lowering the temperature many degrees. From that time the Cholera on board ceased; no new cases occurred.—H.]

Mortality to Populations.—This varies for different epidemics and in different districts. In 1848–49, in Dr. Baly's report, we find that the deaths were 30 to every 10,000 living in England and Wales. This mortality was not evenly distributed: the inland districts giving 17 per 10,000, and the coast districts 50 per 10,000, and 404 districts 7 per 10,000, and 85 districts in which there were none. The denser the population to the square mile, the greater the comparative mortality; thus a population of 915 to the square mile gave 65 deaths per 10,000, and 235 inhabitants, 7 per 10,000. In India among the European troops, according to Ewart, the deaths to strength of the men for 7 years was annually 0·70 per cent., or 70 per 10,000; of the officers, 0·12, or 12 per 10,000. It is not possible to ascertain what is the mortality of the native population of India from Cholera or any other disease. The susceptibility of populations is very difficult to ascertain, because a large number of attacks not fatal are never reported, and therefore, except in the case of troops, no reports are to be depended on. In India, Dr. Ewart places

the liability of European troops to attack at 174 annually per 10,000 for the men, and 73 annually per 10,000 for officers. In India the percentage of attacks is much higher for European than for native troops. The Cholera Commission has shown that the native prisoners in gaols have a liability nearly the same as the European troops.

Diffusion of Cholera.—This is of the greatest interest. Setting aside for the present the question of contagion, it will be well to consider the facts of the diffusion of Cholera by human intercourse. From the mass of evidence on this point, it is impossible to doubt that in many instances it has been spread, in some manner, by such means. Dr. Barry, in the Indian Annals, for 1854, relates that Cholera made its appearance in the military hospital, at Gowalparah, on the 27th April, and that several cases afterwards occurred. The first case was that of a sepoy who had just arrived with a detachment from Gowhatty. Now, there was no Cholera at Gowhatty when he left, and none at Gowalparah when he arrived; but the whole detachment had, forty hours before, passed through a place called Palasbarree, where it was raging. It is supposed that the sick man brought the disease with him from thence. It spread, but not fast, and the first cases which occurred at Gowalparah were from those who passed through Palasbarree, their comrades who waited upon them in hospital, and the sick soldiers in the hospital into which they were received. The dispersion of the children of the Tooting school on account of the fearful outbreak of Cholera among them, in 1848, was attended with attacks and deaths among the children removed, and seizures among the inmates of some of the establishments into which they were received, although there was at the time no Cholera in the institutions or surrounding neighborhood. In the Report of the Indian Cholera Committee on the epidemic of 1861, some remarkable facts are stated with reference to the introduction of Cholera into Gurwhal and Kumaon in 1852. The Report says, "The districts of Kumaon and British Gurwhal lie entirely within the Himalaya mountains, on the borders of Rohilcund. They cover an area of 12,000 square miles. The population is very scanty, scattered for the most part in small villages, which are often separated from one another by vast mountains and tracts of forest. These districts are cut off from the plains of Northern India by an uninhabited belt of forest, and by the swampy and almost deserted region called the Terai. These tracts, some twenty miles in breadth, effectually cut off the inhabitants of the mountains from those of the plains. The intercourse between them is at all times very little, and confined to a few particular lines leading to places of pilgrimage or trade. Cholera is generally as completely absent from these mountains as from any part of Europe, but it has occasionally spread among their inhabitants epidemically with great violence." Mr. J. Strachey, C.S., the able president of the Cholera Commission, formerly in civil charge of the Hill District of Gurwhal, mentions the following facts, which occurred under his own immediate observation: "In the early part of 1852, extensive works of irrigation were in progress at the foot of the mountains in Kumaon, in the Bhabur" (the strip of forest land which divides the mountains from the plains), "of which Mr. Colvin has spoken in his note. Several thousand workmen were collected there from all parts of the neighboring hill. Cholera broke out among these people with great virulence, and they fled panic-stricken to their homes, which were generally at a distance of several days' journey in the interior of the hills. Up to this time Cholera had been unheard of in Gurwhal, or in any part of the neighboring mountains. This is a fact which was carefully inquired into and thoroughly ascertained. Many of the workpeople who had fled from the Bhabur died upon the way to their homes; many others were attacked when they reached their villages. Then Cholera broke out among the other inhabitants of the villages, commencing in very many instances in the families of the men who had brought the disease from below. For a considerable time Cholera was entirely confined to places which had been in direct communication with persons suffering from the disease, but in the course of a few weeks it had become impossible any longer to trace such connection, and Cholera became generally epidemic in the hills. Many of the first cases were carefully investigated; it appeared to be proved, beyond the possibility of a doubt, that in many instances Cholera had never been heard of in the villages until the arrival of the men from the Bhabur, who were the first attacked by it. . . . . There were no other circumstances that could be discovered which appeared to throw even the possibility of doubt upon the fact that Cholera was brought by human intercourse into a district which, up to that time, had been perfectly free from every sign of the disease."[1] There are other instances brought forward by Mr. Strachey, communicated by Mr. B. W. Colvin, C.S., and Dr. F. Pearson, Superintendent of Vaccination in Kumaon, and to these the reader is referred.

[1 Yet, since the epidemic *migrates*, why should it not, *per se*, reach "the hills" later than the lower villages?—H.]

Although it is probable that Cholera is spread by human intercourse, it is indisputable that it originates in places without it being possible to trace any previous communication with infected persons. This has happened over and over again in towns and large establishments in which the outbreaks, after the greatest perseverance, could not be accounted for by any previous exposure to infection. Its appearance at St. Kilda, in the Western Islands of Scotland, is as strong an instance as can be given. This island was cut off from all communication with the mainland, and yet the disease appeared in it suddenly without a trace of importation. In 1848, Dr. Parkes could not trace any contagious origin for the first cases occurring in London.

Contagion of Cholera.—The fact of the diffusion of Cholera by human intercourse leads us to inquire how this operates, and to the question of the Contagion of Cholera. Diffusion by contagion does not negative the possibility of an origin independent of infection. The majority of medical men in India, accustomed to see Cholera year after year, to be in constant intercourse with the Cholera sick, and to see the general immunity of hospital attendants and of themselves, doubt the contagiousness. Some physicians, however, think differently. It does not seem proved that contagion can exist in the shape of a volatile poison emanating from the sick and rapidly infecting the health. It would not be so easy to disprove that there may not be some form of poison which may not be volatile, or which may require time to become so, or to develop its poisonous properties and capability of infection. A volatile poison, at all strong in its action, would be most dangerous to all about the sick, and yet in India the medical men, nurses, hospital coolies, sweepers, and others who are constantly engaged about the sick, do not appear to be more liable than the rest of the population. The disease seldom spreads from bed to bed in a ward; on the contrary, when people are attacked in hospital they lie generally in a distant corner, or in another ward. I have noticed this over and over again; and though I have been connected with the large hospital of the Medical College at Calcutta for many years, I do not recollect any spreading to the nearest or neighboring patients. I should, as far as my own experience goes, say that Cholera does not spread from the sick to the whole by any rapidly acting emanation. Dr. Morehead's observations support the view of the non-spreading of Cholera in hospitals through contagion. They were carefully conducted through three epidemics in Bombay; and though he refrains from drawing positive conclusions, his facts are not in favor of contagion. His observations will be found in his valuable Clinical Researches on Diseases in India. Sir Ranald Martin states, that of the five native keepers and washers of clothes of the Calcutta European General Hospital, who during twenty-five years had kept and washed all the hospital clothing, not one had Cholera, nor had those who assisted them. The same may be said of the dressers and sweepers. In England, in spite of some suspicious instances, the washers of soiled linen and bedding do not seem to have suffered out of proportion to others. In Dr. Baly's report, he analyzes the reputed cases of contagion from this cause, and shows that out of thirty-five reported cases seven only seem to support the contagion theory. It is remarkable that the washers of large collections of linen, soiled by Cholera patients, in many large hospitals, did not suffer seriously in 1849. Dr. Waller Lewis's report to the Board of Health, in 1849, shows the error of the assertions of contagion as regards the case of a number of washerwomen reported to have caught the disease by washing soiled linen, and whose cases he personally investigated.

Against the foregoing may be quoted the cases reported by Dr. Barry before referred to; also the Report of the Committee of 1861, which records that, as far as that epidemic was concerned, the patients in hospital did suffer more than their proportion of attacks. Out of hospital the cases to strength were 4·7, the deaths to strength 2·9, and deaths to treated 62·7 per cent. In hospital, among patients, the cases to strength were 13·5, the deaths to strength 10·6, and the deaths to cases 78·5 per cent. This applies to the whole of the troops attacked with the epidemic in thirteen stations, omitting Meean Meer and Morar, in which the proportions were higher than the above. Including these stations, the cases to strength were 14·7, the deaths to strength 11·6, and the deaths to cases 79·2 per cent. So that it is remarked by the Commission that "the virulence of the disease among hospital patients was clearly more than twice as great as it was among the healthy strength of the regiments." The medical officers escaped entirely, and subordinate medical establishments, which in such epidemics may be said almost to live in the hospital, and the hospital servants, who must have been much more in immediate contact with the sick than the patients were, suffered but slightly; and of thirty men of the 31st Native Infantry sent daily to attend the sick from the 24th of August, when the epidemic was at its height, not one was attacked. It is not very easy to explain these discrepancies and the difference between the spreading in hospital in this and other recorded epidemics. As regards the comparison with the men out

of hospital, there may have been something in the situation of the hospitals themselves to account for the disease spreading so much in them. It appears that in several instances the epidemic broke out first in them. It is possible that the number of patients exposed was too small to render the inferences drawn from their suffering free from fallacy. The returns of the particular regiments show that the greatest excess of difference was in those in which the patients in hospital were small, say 3 to 20. The ratio in those in which were the two largest numbers, viz., 113 and 117, was different; one being above and one below the numbers attacked in the lines.

Infection does not seem to have arisen from the dead body, if one may judge from the impunity with which an immense number of post-mortem examinations have been made in most places, and the contents of the intestines submitted to all kinds of examination. Drs. Mackintosh, Aitken, Gairdner, and others bear testimony to this.

Portability of the Cholera Poison.—A strong instance of this is related by Dr. K. Mackinnon in his work on Public Health, &c. "A regiment proceeding by water down the country had the disease badly. It met a corps coming up the country, with which it exchanged boats: the disease stuck to the boats, left the corps it first affected, and attacked the new regiment which had a clean bill." The breaking out of Cholera in ships many days at sea seems also to show that the poison may be carried.[1] This does not necessarily imply that it was thrown off by a diseased individual. For aught we know, the poison in any district may adhere to surfaces of any kind, and be carried about or remain attached to walls, &c., until fitting circumstances call it into action. When attached to movable surfaces, it may be transported to distant places, and be one of the means of diffusion through human intercourse. We have no knowledge of the period of the disease at which contagious emanations from the sick, if there are such miasms, are evolved. Those who believe that the poison is to be found in the evacuations would consider it to be given off with the rice-water stools. Dr. W. Budd informed me of a case which, he thinks, bears upon the question of the possibility of infection in the convalescent stages. A gentleman was attacked with Cholera in London, and in early convalescence came down to Bristol, and was nursed by his mother, with whom he lived in a very airy, open, and elevated part of Bristol. In three or four days the lady was attacked and died. There was no Cholera in the neighborhood at the time, though there was some in distant parts of Bristol.

Of late years Dr. William Budd, of Clifton, has advocated the contagiousness of Cholera through the medium of the intestinal discharges. His propositions are these: 1st. That the disease is essentially contagious or communicable; and 2d. That it is disseminated, as he believes, exclusively by the liquid discharges from the intestinal canal of Cholera patients. Dr. W. Budd believes that the poison is rapidly multiplied in the human body, and that the rice-water discharge contains the product of this multiplication and becomes a source of infection, so that from one Cholera patient virus enough may arise to propagate the disease to numerous persons. He considers that the poison may be disseminated in the following principal ways: 1st. By the soiled hands of attendants on the sick, a mode of communication which is probably very common within the limits of the family circle; 2d. By means of bed and body linen, and other articles tainted with the rice-water discharges; and 3d. Through the medium of the soil, which, as the discharges are liquid, necessarily receives the great bulk of them.

Dr. W. Budd thinks that from these places of deposition the poison may spread itself by rising into the air with the products of evaporation, by percolating into drinking water, or by atmospheric dispersion, in the form of impalpable dust, after it has passed into the dried state. *He considers that it is a disease which infects the ground.* He contends that the poison may be preserved for months or years in a dry state, in the same way that the germs of fungi or infusoria may be, and that they may in favorable conditions be brought into activity, and being received into the human organism, after a very short incubation produce the disease, and by multiplying with enormous rapidity provide sufficient material for extensive infection. On the other hand, he thinks that in unfavorable circumstances the germs may as rapidly decay and become extinct, and that thus an epidemic may end. In short, Dr. W. Budd imagines germs capable of preserving a permanent dormant vitality with susceptibility of immense power of reproduction in the living body, and liable to rapid decay when placed in unfavorable circumstances. These views are very simple; they are by no means far-fetched or beyond the analogy of other epidemic diseases, and have been received with great respect in England. They are supported by strong evidence, mainly of this nature: Cholera has been known to rage violently among, and to be confined to, one sex, in

[1 Reason has been given, on a previous page, for a different interpretation of these facts.—H.]

establishments containing both male and female inmates under the same roof, separated from each other merely by walls or partitions, and breathing the same air, eating the same food, and drinking the same water; the only difference between them being in the use of separate privies; the contamination of one of these privies by discharges thrown into it from the first case explaining the reason of the difference of suffering in the two sets of persons.

Although, according to Dr. W. Budd's view, Cholera is contagious, it is so in a very different form and manner than if it were so by means of a volatile or gaseous emanation proceeding from the sick. Doubtlessly it explains the diffusion of Cholera in a more satisfactory way than the ordinarily received notion of a contagious poison, and after the facts brought forward by him it would be wrong to neglect the practical application of their teaching. I think, however, that more evidence is required before it can be held to be proved that the specific exciting cause of Cholera is to be found in the discharges only. The theory will hardly explain all the phenomena of the spread of the disease in all cases. Take, for instance, the rapid appearance of the disease in the Second Madras Europeans, mentioned by Rogers; its rapid diffusion at Kurrachee, in 1846, and in Paris, in 1832, as before stated; or such a case as this, mentioned in Jamison's report; in a cantonment perfectly free from disease, ten men of the same regiment were attacked in a single night, and every case proved fatal—no fresh case happening in the corps or any other. It is not to be supposed that these men were the only susceptible persons in the whole regiment. It may be difficult to account for the outbreak on any theory, but not less so on Dr. W. Budd's than on any other. It appears unable to account for the manner in which Cholera becomes milder in the decline of an epidemic. The deaths to cases are much diminished. We should expect that as the epidemic advances the virus would be more in quantity, more concentrated, more virulent; but such is not the case. The susceptibility in the population remains to a considerable extent, because numerous cases occur, but they are milder. We should expect that the virulence of the disease would go on increasing until the susceptible people were quite exhausted. Some other explanation than decay or partial decay of the Cholera germs seems needed. The regularity with which, over wide areas, Cholera epidemics advance, and the simultaneousness with which they attain their maxima and their decline, seem to imply some general law, and not a diffusion dependent upon the chances of fecal contamination only. I think that those who have been in the habit of seeing much of Cholera will not be inclined to think that there is much potency at all events in the *fresh* Cholera discharges. It has been my lot, in numerous epidemics, to see many cases at a time, and for a long time in my own wards; to see the beds, the sheets, the hands, of attendants, the floors, frequently—nay constantly—soiled with discharges: to see the utter impossibility of providing fresh beds and fresh blankets, &c. for every case; to see abundant opportunities for the diffusion of the poison from the discharges; and yet, as mentioned under the head of contagion, not to see Cholera spread in the wards. If fresh Cholera discharges were so virulent, every bed next to a cholera-bed would be a bed of the disease; every bed and metal bed-pan would be a source of Cholera to every succeeding patient. My own observation is that no such evil results. The hospital sweepers, whose special duty it is to remove all the discharges, do not suffer out of proportion to others. Dr. Baly's report on this point has been already referred to. It may be that decomposing Cholera discharges may be more mischievous, as has been supposed by Pettenkofer. If, as supposed by Dr. Budd, the poison is multiplied so rapidly in epidemics as to suffice for the rapid diffusion which takes place in severe epidemics, it ought to be equally powerful when under observation in a sick ward. The mode in which Cholera spreads in wet weather in India seems adverse to its diffusion by the discharges. In dry weather, with a strong wind, it is easy to suppose that the Cholera dust may be spread to a considerable distance; but in the rains of the tropics and bordering countries we cannot trace this mode of diffusion. All matter infecting the ground must be swept away in the violent falls, to which English rain is but a trifle; it would, one would suppose, be diluted and swept away into rivulets and streams, and ultimately into great rivers. Sometimes it might overflow tanks, but in many parts of Upper India the drinking water is taken from deep wells; and where there are tanks, the Hindoos especially are careful to keep them pure, so that it is not so probable as may be supposed that they are often contaminated. And yet it is in the rainy seasons of the Northwest Provinces that the severest Cholera epidemics have raged. Although the specific poison of Cholera may not exist in the rice-water stools, it must be admitted that Dr. W. Budd has brought forward strong evidence in support of his views, and the facts which he adduces can, in the present state of our knowledge, be explained only upon the supposition that the evacuations of Cholera patients have, in some state or

other, an influence upon the spread of Cholera. There are yet many points that require elucidation to prove that the Cholera poison multiplies in the human body, though perfectly in accordance with the cases of many other epidemic diseases, so that the specific agent is contained *pur et simple* in the rice-water stools. It would seem that they are the congenial soil for the multiplication of the poison, rather than the direct source. It would be a good thing for mankind that Dr. W. Budd's views should prove correct. It would then be a comparatively easy thing to prevent the spread of the dire pestilence.

[In the absence, however, of any conclusive proof of the correctness of those views, it is unfortunate that they should have met with so large an acquiescence as they have, in England and America, East Indian medical authorities do not sustain them; in Germany Pettenkofer has opposed them, and in France they are, at least, not generally accepted. The disadvantage of maintaining, without proof, that the stools of Cholera patients *only* convey the "contagium" of the disease, is, that the attention of sanitarians and the general public is thus diverted from the needful care of other excretions and material of organic decomposition, *all* of which is promotive of the spread of Cholera.—H.]

Cryptogamic Theories of Cholera.—Chemical analysis of the air has thrown no light on the cause of Cholera. It is possible, however, that prolonged microscopic examination of strained air, as in the method of Pasteur, might be more successful. Dr. Cowdell has advocated the cryptogamic origin of the disease. Dr. Britton made some careful examinations of air collected by an apparatus from Cholera rooms, and discovered therein some bodies which resembled others found by Dr. Swayne and himself in the rice-water discharges, and by Dr. W. Budd in the water of districts infected with Cholera. These were afterwards shown by Mr. Busk to be starch-granules, and a species of uredo or blight. Mr. Rainey, Dr. R. D. Thomson, and Dr. Hassall examined the air in the epidemic of 1853–54. The two former made an extensive series of observations at St. Thomas's Hospital. Although all these observers found organic forms in abundance, they did not discover any special cause for Cholera in those organisms. Nevertheless, there seems much that is attractive in the cryptogamic theory of the disease, and the whole subject derives additional interest from the late researches of Pasteur. Valuable information on this subject might be afforded by investigations made in India during the presence and absence of Cholera epidemics. The writer of this has often regretted that he never had leisure to undertake such investigations. Many of the phenomena observed during the march of Cholera epidemics might be explained much more satisfactorily upon the supposition of the exciting cause being masses of organisms moving in obedience to atmospheric impulses and currents, than by most other theories. They might multiply wherever they found a fitting nidus, which might be in privy atmospheres, or in air abounding in emanations from decaying and putrefying matter, or in crowded rooms, and indeed, all vitiated atmospheres. They might appear to impart an infecting character to the choleraic discharges by multiplying enormously in them.

[Klob of Vienna, Thomé of Cologne, and Hallier of Jena, have asserted the discovery of peculiar fungi in the stools of Cholera patients. Hallier ascribes the Cholera fungus, which he calls *urocystis*, to the rice plant as its first *habitat*. Assistant Surgeon T. R. Lewis, of the British army in India, in a careful investigation of the subject, failed to find confirmation of Hallier's theory. It has been opposed, also, by Berkeley, the eminent cryptogamic botanist. Loesch of St. Petersburgh, observed multitudes of cercomonads in Cholera discharges. Possibly, the presence in extraordinary numbers, of minute organisms not specific or peculiar, may become a morbific cause: as trichinæ, which are almost or quite innocent while few in number, produce fatal derangement of the system when they accumulate in millions.

Even if specific organism causative of Cholera be not discovered, since it is conceivable that it may be ultra-microscopic in minuteness, we may still hold the view that, for this and analogous diseases, the "germ theory" has in its favor a large preponderance of probability.—H.]

After allowing all possible latitude to the dissemination of Cholera by human intercourse, which may include the diffusion by gaseous emanations acting directly through the atmosphere, or through fomites, or the diffusion through the Cholera evacuations, it appears that we are still unable to explain all the facts of its origin in a place, and its spread over the land. Summarily we may say that we shall still want an explanation of those facts which show that Cholera will arise without the possibility of tracing any communication with the sick, or with fomites or choleraic discharges. It cannot be said that the supposition of a poison, cloud, or miasm, spreading through the land, will account satisfactorily for all the phenomena, either such as its strange limitation to certain areas, or its passing over a town on its apparent track, leaving it unscathed, or nearly so, to show itself violently in some place beyond the town so spared. The only way in which this ca-

pricious action could be accounted for, would be on the supposition of clouds of matter or of fungi or infusoria, limited in their spread by atmospheric currents, sometimes compressed into vertical strata and touching the earth in a thin line or belt only, or lifted upwards by some upward atmospheric movement, and thus being above the places passed over. But though it would be possible to conceive this mode of progression of a mass of organisms obeying the impulses of the atmosphere, yet no such organisms have been hitherto demonstrated, and, indeed, the progress of the disease against strong periodical winds would seem to oppose the view. May it not be a mistake to consider the specific cause at all as a simple body, either generated from without, and air-wafted to a particular spot, and then multiplying itself indefinitely, or as a locally-generated agent, and spreading over certain areas? Might it not be more in accordance with facts to suppose that neither a miasm from without, nor a miasm from within, exclusively contains the specific poison? Might it not be that two factors are needed, the one some airborne material or some dynamic modification of atmospheric elements coming from without, the other some local element: neither being potent unless united? The peculiar atmosphere sweeps along hither and thither, and it is only when it meets with the other peculiar substance that the poison is generated. It may be that a Cholera evacuation is the most prolific of the peculiar local agents. Some general, not local law, seems to govern all Cholera epidemics. Contagion from Cholera discharges may operate, but there must be something beyond this. Contagious diseases are not epidemics at all times, even in the same places. In Lower Bengal every year the native smallpox inoculators proceed to inoculate patients. The disease in these cases rarely spreads. For years successively this may go on without evil results, and there is no uncontrolled diffusion; but every few years a wide-spreading epidemic of smallpox, which nothing seems to resist, sweeps over the land. There is nothing discoverably different in the epidemic and the common years—the same people, the same habits, the same places, the same filth, or the same absence of it. In all the seasons there is the poison. In all the seasons there is the susceptible population; and yet how different the results! It is not to be supposed that the mere amount of poison generated in times of confluent smallpox makes the difference; but even if so, how is it that in one year all the cases are confluent, in others discrete? Why has the inoculated poison such a limited influence in favorable years? If there be poison in the Cholera stools, it may bear the same relation to Cholera epidemics that the inoculable virus of smallpox in healthy years bears to the destructive epidemics of the exceptional ones; in certain circumstances a sufficient cause—not the only one.

Though it must be confessed that we do not know what is the exciting cause of Cholera, we may hope that we are grasping some of its laws. It is impossible to deny that it riots far and wide, independently of contagion; but we must admit that it may be spread in some way by human intercourse. This is perhaps more satisfactorily explained by the views of Dr. W. Budd, than by other modes of contagion. The influence of predisposing causes is certain, and it is undoubted that they may be rendered less active. It is to be hoped that the Sanitary Boards now permanently given to India by the recommendations of the Royal Commission on the Sanitary State of the Army in India, will do much to diminish the mortality of Cholera. While grateful to them for their labors and recommendations, we must in justice to the officers of the Royal and Indian Medical Services state, that many of them had earnestly striven in the cause of preventive medicine long before the committee was formed, and that most of the facts published by it were well known and appreciated by the said services. Among those who have worked in the spirit of the Sanitary Committee, and who have done much to advance the cause of sanitary science in India, either by their writings, by their position about those high in power, or by their official standing or appointments, Kenneth, Mackinnon, Martin, Bedford, Chevers, Parkes, Balfour, Morehead, Rogers, Lorimer, Maclean, Alexander Grant, Forsyth, Hugh Macpherson, Ewart, Maclennan, Leith, Mouat, and P. Walker may be mentioned with special honor.

Symptoms.—The typical cases of well-developed Cholera present well-defined symptoms. The descriptions in the following paragraphs apply to the ordinary forms of Epidemic Cholera. In the beginning of an attack, the most prominent symptoms are disturbances of the stomach and bowels. Then, in swift succession, though not necessarily in that order, the circulatory, respiratory, muscular, and nervous systems suffer. Then, in severe cases, ensues extreme depression of all the functions of life, which often terminates in death. The symptoms are naturally divided into periods or stages, which may be called those of *Invasion*, *Development*, *Collapse*, and *Reaction*.

Stage of Invasion.—We have not very often to deal practically with more than one or two sets of symptoms in this stage; viz., preliminary disturbances of the bowels. There are doubtless other prelimi-

nary signs which are sometimes felt for a few hours, or even days, by patients, before passing into the well-marked stages. Twining, Annesley, Orton, and others mention among these preliminary symptoms a feeling of malaise, oppression of epigastrium, depression of spirits, pallid, anxious, and sorrowful cast of countenance, sense of exhaustion, vertigo, noise in the ears, headache, tremor, and sense of debility. Annesley gives the following statement of the sensations of a patient, related by himself to his surgeon, Mr. Colledge: "I must knock off work, I feel unable to do more, but do not know what is the matter; I have only a little pain in the stomach and rumbling in the guts." Mr. Colledge says, sighing, peevishness, and uneasiness accompanied these complaints. Annesley relates a case in which, from the countenance of the patient, he suspected the approach of Cholera, and in which he contented himself with merely watching him. Nine hours elapsed after the establishment of his suspicions from premonitory signs, before vomiting, purging, and spasm appeared. Without doubt, after making due allowances for the fancies and terror of people during Cholera epidemics, preliminary disturbances of the nervous system have occurred, and should not be made light of. But it must be acknowledged that in the majority of cases no such forewarnings are perceived. A premonitory symptom for which medical aid is often called, during Cholera epidemics, is diarrhœa. The disease itself often begins suddenly with purging or vomiting, but in numerous cases there is relaxation of the bowels for some days or hours before the real attack begins: the motions, watery or semifluid, sometimes pale, but not always so; three, four, or more in the twenty-four hours, perhaps with griping. There may be some sense of exhaustion with this. There is a strong tendency to diarrhœal complaints during Cholera epidemics, and, though some of the cases may be harmless, many of them do ultimately pass into Cholera. The necessity of checking such discharges should be earnestly impressed upon the medical practitioner.

Stage of Development.—Evacuation Stage.—It is a matter of common observation that the attack commences with purging, very often early in the morning; vomiting seldom comes on till later. The alvine evacuations are copious and fluid. The first stools, generally, consist of the ordinary contents of the intestines mixed with much liquid. The patient often describes them as rushing from him in a full stream. Often so great is the purging, that he sits sometimes on the close stool or privy, until several pints or even quarts of fluid have passed from him. Generally the evacuations are repeated and frequent; they are soon attended with a feeling of exhaustion, so that the patient is glad to get to his bed again. This excessive watery purging characterizes the onset of Cholera. It is frequently painless, but not always so; therefore we should not suppose that a patient has not Cholera because he has griping and pain. So excessive are these evacuations, that in two or three hours, or less, an ordinary-sized stool-pan will be nearly filled. With the exception of those first passed, they are of a light straw or pale drab color. The surgeon should not be thrown off his guard because the evacuations he sees in mass are somewhat colored. There is often fecal matter enough in the first evacuations to color the subsequent stools passed into the same unemptied vessel. The quantity and consistence of the discharge and the effect upon the pulse are of infinitely more importance than the shade of color. The name of rice-water stool has been given to the genuine unmixed Cholera evacuation, and if collected separately, after the first two or three have been passed, the discharges do, indeed, resemble water in which rice has been boiled. It is a thin, pale, slightly opaque or slightly turbid fluid, depositing a sediment on standing, which is like fine, minute, flaky particles of rice broken down by long boiling. Occasionally in the evacuation stage, the fluid is whitish or somewhat milky, or of shades varying between this and the whey-like color before mentioned. With purging, but generally beginning later than it, is combined vomiting. The fluid vomited, if unmixed with ingesta, is clear and watery, often in quantities of a pint or more, and generally ejected with force. The vomiting is less constant in its intensity than the purging, and sometimes is very slight, occurs at irregular intervals, and is readily excited by medicine or drink. When the rice-water evacuations appear, cramps generally set in, not often before this; they are most frequent in the fingers and toes, in the calves of the legs, thighs, and sometimes in the abdomen; they may continue through the next stage. By the time that the vomiting and purging have become fairly established, and even in the earlier stages, the countenance becomes altered. It assumes a somewhat leaden hue, and has a tendency to shrinking; a stony, staring look, with the capillary circulation sluggish. Alteration of countenance often points out the character of the disease, even before any symptoms have been complained of, though they may really have existed for some hours. Whatever may be the state of the countenance, if there have been many stools, or frequent vomiting, symptoms of depression appear; the pulse begins to lose its strength and soundness, and the temperature of the surface falls. Within six or

seven hours from the onset of the purging, or even much earlier, the pulse may dwindle down to the faintest thread, or may entirely disappear from the wrist for many hours; in others, even in well-marked cases, though in milder forms of the disease, never quite ceasing to be felt at the wrist. This period is one of the greatest interest to the surgeon. With the utmost anxiety he watches, hour after hour, the waning, or the stationary, or the returning strength of the pulse, its maintenance or its extinction heralding either a happy issue in the first stages, or a future struggle of the deepest danger through the next stage, that of collapse. In some cases the balance oscillates for hours. If it turn adversely, with the failing circulation come the shrunken face, the lessening of bodily warmth, and the greater exhaustion; and the patient passes into the stage of collapse.

Stage of Collapse.—Algide Stage.—This is very much more dangerous than the last. Evacuations from the stomach and bowels are now less frequent and copious. The alvine discharges generally contain less liquid; sometimes still of many ounces in each, at others merely a little clear fluid with gelatinous mucous-like flakes or masses. The stomach often acts, violently, perhaps, but with less discharge. In the extreme state of the collapse the patient nearly resembles a corpse. There is the utmost depression possible with a capability of recovery. When this stage is fully formed, the patient no longer rises from his bed, though he often tosses about on it with sudden and frantic jerks, throwing off all covering, as if intensely hot, and seeking for cool air. Or he half springs up in bed shrieking from agonizing cramps, or he is still more exhausted with irregular paroxysmal gasping for breath, which subsides to be again renewed, and may end in the respiration being more constantly embarrassed. He passes stools under him, heedlessly or with indifference. Now, the features are shrunk and livid, the eyeballs small and buried in their orbits, the lower lid drooping, and the eye half open. The surface is deadly cold, except, perhaps, sometimes the forehead or præcordia, the tongue icy to the touch, the very breath a cold air stream, the temperature in the mouth 79° to 88° Fahrenheit, in the axilla 90° to 97°. The general surface is pale, bluish, or livid; often bathed in profuse sweats, as may be the forehead, and the shrivelled hands and feet, the hands looking as if they had been long soaked in water; the pulse absent from the wrist, and in very bad cases from the brachial artery; the blood obtained with difficulty by venesection, sometimes not at all, and, when drawn, thickish, or tar-like; the mind apathetic, so that between the times that he is roused by cramps or is involuntarily restless, the patient lies on his side or back, heedless of surrounding objects or persons, but not comatose until, perhaps, the last. Thirst and sense of heat of epigastrium are at times intense and tormenting; water! water! is the urgent and frequent supplication. If given to him, the sufferer spasmodically rises on his elbow, and gulps down the cooling liquid with intense eagerness, perhaps to vomit it up immediately. The voice is feeble, whispering, or suppressed. Muscular strength is almost always greatly reduced, but in some instances it is retained to an extraordinary and disproportionate extent. A man may be sometimes seen to walk across a ward, or to sit up in bed, or to rise to stool, whose pulse is imperceptible at the wrist, In extreme collapse the circulation is at its lowest ebb; the cardiac sounds scarcely, if at all, distinguishable; the respiration is embarrassed, or with alternations of quiet breathing and difficult paroxysms, and, in the later stages of fatal cases, always difficult; the dusky hue of the surface, the coldness and dyspnœa, all show great stagnation in the pulmonary vessels. Absorption, which in the early stages is strong enough to remove collections of fluid from the smaller or even larger shut cavities, is now nearly if not quite arrested. Glandular secretions appear to be stopped also, though at times milk seems to have formed in the breasts of nursing women. The alvine discharges are less, consisting of remnants of fluid exuded before marked collapse came on; the motions contain a little mucus with a small quantity of liquid. There may, nevertheless, be much matter retained in the intestines, the muscular coats of which are too feeble to expel it. The stomach is less frequently excited to vomiting, and there is an absence of saliva in the mouth. The changes between the atmosphere and the blood, and between the blood and the tissues, are as low as is compatible with life; the amount of carbonic acid gas thrown off from the lungs is about 1·5 per cent. instead of about 4. The evacuations are not generally of bad smell; but in some prolonged cases, in which there has been retention in the bowels, they are very offensive in the latter stages; similar to rotten fish.

There is no more distressing state to witness than that of a patient in the state of collapse from Cholera. The contrast between his state of a few hours previously, the sudden affliction of friends and relations, the apparently hopeless condition of the sufferer, all conspire to impress one painfully. But with all this, hope need not be utterly cast aside. The patient may lie for hours without a pulse at the wrist, and sometimes even in the brachial artery, and yet recover. Cases without

pulse in the brachial artery and manifest permanent impeded pulmonary circulation, with dusky and livid countenance, seldom or never recover; but short of this a large number do.

The state of collapse may last for hours, twelve to forty-eight, or in very rare cases even longer, and yet recovery may take place. The patient may die in three or four, he may live twenty-four or more, the pulse never returning, the respiration becoming more and more impeded, the brain more and more torpid, with onset of coma, moaning respiration, and closing in death. Or, after lasting a variable time, five, ten, twenty hours or more, the patient becomes less restless, less thirsty, and jactitation and anxiety give way to calm; he dozes quietly with easy respiration, and this is of most favorable import. The pulse at the wrist flickers, we are in doubt if we do not feel some movement, then we are certain that we do, then this beat is unmistakably established, the superficial veins show themselves filling at the back of the hands, the surface is less cold, the countenance assumes more and more its natural character, and even becomes flushed, respiration is quiet and regular, temperature rises, and reaction may be considered certain.

State of Reaction.—The patient has now passed through the collapse, and is, in many instances, nearly himself again. In some persons recovery is as rapid as decline. The secretions are readily performed, the strength returns, and health is the speedy issue. Twining mentions the rapidity with which many Cholera patients recover, and that it is not uncommon to see a man well on the third day after an attack of the worst symptoms. Mr. Grainger says, "I have seen a man stand at his door on Wednesday, who on Monday was in a perfect collapse." Such cases were common in India in Twining's day. They are not so common in Calcutta now. When the collapse has passed away, our anxiety is not at an end. The perfect establishment of the secretions is our next desire. As said before, during collapse all secretions are stopped, but after reaction has come on they should reappear. The first indication of change is in the color and character of the alvine evacuations. Independently of the influence of drugs, such as lead, mercury, &c., we find that they become sometimes milky or whitish, then grayish, then darker or muddy, and at last brown. We find that the liver is acting, at all events as far as coloring matter is concerned. We also look anxiously for the passing of urine. We may have to wait ten, twelve, or thirty hours, or even more. At first it comes scantily, high colored, acid, an ounce or two with a strong and peculiar animal smell, deficient in urea perhaps, but not in all animal principles, generally albuminous with many transparent casts. The albumen or allied compound when present is not always detected by nitric acid, though often by heat, giving a deposit not dissolved by nitric acid when the nitric acid test alone failed to detect it. The urine often turns pinkish with nitric acid. After the first, the secretion becomes copious, and probably by this time all the functions of the system will have become natural.

The above description applies to the well-marked evacuation and algide stages of severe ordinary cases met with in the present day in India. It is not to be supposed that all these symptoms occur in every instance, or that they are always equally well developed. There is not always the same degree of collapse, the same duration of stages, the same apparent amount of evacuations. The cases sketched above are such as have the characteristic evacuation stage, lasting six or eight hours or more; followed by decided and complete collapse. Modifications and variations will be mentioned hereafter.

As has already been said, the stage of reaction is not always the period of safety. When the algide stage does not last more than eight or ten hours, there is hope of regular convalescence; but when it goes beyond this—say eighteen or twenty-four, or more—we must, in the event of reaction, expect a much larger proportion of secondary risks.

It seems that in the earlier known epidemics in India the proportion of cases with healthy reaction and of rapid recoveries to those of consecutive disease was greater than it is in the present day. In the great epidemic of 1861, about 22 per cent. of the fatal cases died in the reaction stages. In England the opinion is prevalent that consecutive risks are less common in India than in Europe. Dr. Morehead shows that this is a mistake, and says that practitioners in India are as familiar as those of Europe with all the diseases of the reaction stages. I think that most of the surgeons in Lower Bengal will confirm this opinion. Dr. J. Macpherson, in a paper on the Indian Annals, shows that for a period of ten years, one-fifth of the fatal cases of Cholera in the Calcutta General Hospital died after the stage of collapse was over. Whatever may have been the case formerly, I can bear testimony to the large proportion of secondary affections which are now met with in Calcutta, both among Europeans and natives. I regret that writing this paper in England, away from my own hospital records, I cannot now avail myself of them to prove this and many other points by figures. In the Report of the General Board of Health on the Cholera

epidemic of 1853–54, it appears that in 1777 deaths, 249, or about 14 per cent., died of consecutive fever. Dr. Gull states that probably not more than one-tenth died of it in 1848–49. In the Northwest Provinces of India in 1861, as before said, the proportion was 22 per cent., which is larger than it was in England. I have, therefore, little doubt that in India, or at all events in Lower Bengal, the diseases following reaction are quite as frequently seen as in England. The diseases succeeding to collapse may be arranged under the following heads:—

1st.—Imperfect reaction, relapse, and occasional deviations from normal convalescence.

2d.—Complicated reaction ending in uræmia, low fevers, gastro-enteritis, diarrhœas, and various asthenic sequelæ.

Relapse after the circulation has been fairly restored, is not very common, but it is met with now and then. There is a return of vomiting, purging, exhaustion, and death in a few hours or two or three days. In one case known to me it occurred after the administration of a very mild dose of a resinous purgative. Dr. Johnson mentions a case of relapse after some pears had been eaten. In the case of a lady whom I attended the evacuations and algide stages were passed safely through; during the next day great dyspnœa came on, and death, with all the symptoms of general collapse, followed. In this case I thought that the great disturbance of the respiration was due to collapse of the lung tissue.

The milder deviations from ordinary convalescence are slight febrile excitement, obstinate vomiting, hiccup, indigestion, and want of sleep.

Mild Fever.—In some of the cases after collapse the patient appears drowsy and listless, and, though generally easily moved, likes to be quiet. There is often a slight febrile movement with this, the tongue is dryish in the centre, the epithelial coating of the lips and mucous membrane of the cheeks thickened and opaque, and superficial abrasions occur opposite the most prominent teeth. The secretions appear to go on; the appetite is absent. In most instances this state does not last more than four or five days, and perfect recovery follows; or the fever assumes more or less of a remittent or intermittent character, which passes off in a few days; or sometimes it ends in coma or decided typhoid symptoms. Occasionally the fever terminates with an eruption somewhat resembling urticaria; in some cases erythema, which may become prominent all over the body, last two or three days, and then disappear with slight burning desquamation. It is not dangerous. The patient generally recovers as the eruption goes off. In some instances a red exanthematous efflorescence appears for a day or two instead of the urticarial elevations. The fever may be accompanied with irritation of the gastric mucous membrane and slight diarrhœa, but these are not essential.

Vomiting.—Irritability of the stomach may be caused by some degree of congestion of its mucous membrane approaching to subacute gastritis, owing to the frequent straining and vomiting, or to stimulants incautiously given. It is not necessarily accompanied with feverishness, but there is general thirst and burning heat of œsophagus and at epigastrium. The patient cannot retain nourishment at first; the smallest amount being at once rejected. This condition often lasts several days, and requires great care and attention. I do not remember to have seen it fatal. When existing as the only symptom, great debility attends it, and convalescence is often delayed many days by its continuance. Sometimes it passes into a dangerous state of gastro-enteritis. Hiccup annoys the patient greatly, interfering with his rest, and is generally present with loss of appetite. It is not always constant: sometimes it leaves the patient intervals of some hours, then comes and goes again. It is generally accompanied with great eructation of gas, but is sometimes without it. It is not dangerous, though it is very annoying, and distresses the patient so much that he anxiously demands relief.

Want of Sleep.—This is very irksome when prolonged. I have known it to last for two or three days or more, the patient longing for sleep, and restless, and imploring for medicines to soothe him to repose.

Imperfect Reaction.—This is somewhat similar to relapse, but reaction does not proceed in it as in those which I have called relapse cases. In this imperfect reaction, the symptoms of great collapse pass away or diminish; the pulse improves, but does not regain its natural strength; the temperature remains low; the purging, though abated, continues, the evacuations watery, though often slightly tinged with bile; the vomiting may continue; there is anorexia and much prostration; the urine flows freely, and is apparently natural. Though time passes on, the patient does not get stronger; he lives three or four days, and then sinks from exhaustion, with disturbed respiration and coma. It may, ultimately, if the case is sufficiently prolonged, pass into the typhoid state. There is no pyrexia with this important reaction. It is not always fatal, but very frequently is so.

Uræmia.—This is very common. The symptoms of collapse pass off. If the urine is not secreted, we are not long without indications of mischief. Vomit-

ing often returns soon, the ejecta not now colorless, but generally grass-green. Uræmia shortly succeeds. The patient has weight about the loins, listlessness, delirium, drowsiness, coma; the eyes become injected and dark, the tongue dry, the teeth foul. Before coma comes on there often exists a singular state of muscular resistance; the patient opposes all attempts at overcoming the ordinary contraction of the muscles. He strongly resists the opening of the eyelids, the depression of the lower jaw, or the straightening of the fore-arm. There is not spasm, but simple resistance. Dr. Morehead notes that uræmia is preceded by preternatural slowness of pulse. If the renal secretion is not established, the coma becomes deeper and deeper, and death may occur within forty-eight hours after the full reaction; but it is generally delayed longer than this; and even after secretion is apparently restored, the patient may pass into the typhoid state. The length of the collapse has doubtless an influence upon the suspension of the renal secretion; the shorter it is—its duration not exceeding eight or ten hours—the greater probability is there that the kidneys will act easily within twenty-four hours. We must not despair, however, if the secretion is retarded for twenty-four or even thirty-six hours after reaction. In extreme cases even this limit may be passed. Dr. Morehead notices that, when the collapse has lasted eighteen hours or more, there is much greater danger of consecutive disease depending on defective secretion; and the more speedily the full circulation has been restored after long collapse, the greater is the risk from continued suppressed urinary secretion. The comatose cases are very frequently fatal, but not necessarily so. If the kidneys act, bad symptoms will probably diminish; but if they do not, death is inevitable. The secretion of urine, however, does not always relieve the head symptoms, as Dr. Morehead has also pointed out. The bowels are sometimes relaxed, sometimes constipated; the evacuations yellow and feculent. Diarrhœa may carry off some urea, and should not be checked. The whole train of symptoms of uræmia need not be described here. They are the same as in uræmia generally. There is no febrile action, or it is but slight.

Cholera Typhoid.—Writers do not always distinguish between this and uræmia. Uræmia may pass into a typhoid state, but there does exist a consecutive fever independently of uræmia; or at any rate without suppression of urine, or without albuminous urine. Possibly the blood may not be fully purified of urea, and a quantitative investigation might prove a deficiency of excretion, but in some of the cases I have found sufficient urea to crystallize freely on a glass microscope slide. I have never made a quantitative examination for the twenty-four hours. It is likely that other principles, besides urea, indicative of the blood being loaded with effete matter, might be discovered, as in the cases mentioned by Dr. Letheby. In cases of consecutive fever, the patient for two or three days after the collapse may appear to be doing fairly, the excretions and secretions appear to go on sufficiently; then the tongue becomes dryish, and a little quickening of the pulse comes on during some part of the twenty-four hours; perhaps towards evening. Often the febrile movements are pretty regularly paroxysmal. The excitement is not high, and the pulse, though quicker, is generally weak and small. In a short time the patient passes into a typhoid condition; sometimes passing through all the risks of the extreme adynamic state; very often dying; but the disease is not necessarily fatal. The duration of the fever varies. I have known a patient to die twenty days after the collapse had passed away, the urinary and hepatic secretions being apparently natural throughout. Probably the majority die about the eighth to the twelfth day. This fever may possess all the usual symptoms of adynamic fevers—low delirium, coma, dry tongue, parched lips, feeble and quick pulse, bed-sores, liquefaction of the blood, purpura, &c. It would be superfluous to describe more fully all the signs of the typhoid state. The patient may sink under it, or slowly recover, and remain for a long time in an anæmic, debilitated condition. Coma sometimes comes on within the first day or two after reaction. In some cases this may depend upon sudden poisoning by urea, but in many the secretions do not seem to have been in fault. It is generally fatal.

Ulceration of the Cornea.—Disintegration of the cornea is sometimes a sequel of Cholera in India: most frequently among natives, but I have seen it in Europeans also. It comes on a few days after the reaction has set in. An asthenic state of the system is necessary to it. Generally there are some typhoid symptoms, but it does not always appear in the worst state of the low fevers. The lower segment of the cornea is the part to suffer first. It is not caused by inflammation; in the early stages, not a trace of an enlarged vessel may be visible. During the worst part of the collapse the lower eyelid falls away from the upper, the lower segment of the cornea is exposed, and the uncovered part of the epithelial layer at the time often looks dryish and parchment-like. It is probable that this leads to the subsequent disintegration of the part; but whether this be

so or not, the essential cause of the affection is defective nutrition of the cornea, as in the case of Majendie's dogs. We find that in four or five days, or later, after reaction a part of the lower segment of the cornea on one side becomes hazy; the area of cloudiness not very accurately defined. There is so far no discoverable disintegration. In twenty-four hours the epithelial layer over part of the opaque area shows abrasion, or there may be some loss of substance of the deeper layers of the cornea, producing a minute curved groove about one twenty-fourth of an inch from the corneal margin; the groove is less than the hazy part. By this time the opposite eye has generally become hazy in the same manner as the first had been on the previous day. In twenty-four hours more the groove in the first eye may have doubled its size. Examined with a lens, the surface of the groove is of a dirty ash color, and opaque, having indeed all the character of a minute slough. The ulcer may spread, and penetrate the entire thickness of the cornea, and extend at its margins, but in the majority of cases the patient dies before such extensive destruction occurs. The mischief is generally symmetrical; but the eye last attacked is less advanced than the first. If the patient recovers, the eye is generally preserved without any very serious disfigurement. With his general improvement the ulcers also improve. There is first arrest of disintegration, then diminution of haziness and opacity, then cleaning of the surface of the ulcer, which becomes beautifully clear and transparent. Cicatrization follows, perhaps rapidly, and the repair is generally perfect, so that unless the ulcer is very deep, in a few days it is often difficult to discover where the mischief has been. Permanent opacity, hernia of the iris, or staphyloma, are rarer than would be supposed. In the later stages, there may be vascularity of conjunctiva and sclerotic, but they are effects, not causes, of the ulcers. This state of the cornea is frequently met with in India, in cases of chronic diarrhœa and other debilitating diseases, and of course is not peculiar to Cholera.

Gangrene of Various Parts. — Among some of the rarer sequelæ, I have seen, among the natives of Bengal, complete gangrene of the penis and scrotum, and partial gangrene of the scrotum and of the point of the nose; also superficial gangrene of the mucous membrane of the mouth. I have never seen mortification of the intestines. When the mortified parts are small in extent they may be cast off, but in the extensive gangrenes of the penis and scrotum I believe that death always happens.

Parotid Glands.—Swelling and inflammation of these are not unfrequent; they often, though not always, end in suppuration. It is unnecessary to give an account of the symptoms of this affection. It appears during the second or third week, and with low fever. Both sides generally suffer in succession. The patients often sink during its progress. It is always a grave symptom, indicating a very asthenic state of the system, and interfering much with the patient's power of taking food.

Bed-sores, Boils, Ulcers, Low Inflammation of Lungs, Pleura, &c.—The patient often suffers very much from these. They often greatly retard convalescence, but do not require any special notice.

Consideration of the Special Symptoms.— It may be well now to consider more in detail some points not fully spoken of in the foregoing account of the symptoms, in order not to interrupt their description. The intestinal discharges require more notice. The quantity evacuated is generally considerable. It is difficult to ascertain this in hospital patients. They amount frequently to 80, 100, or 150 ounces during the evacuation stages. After the feculent portions of the stools have passed they have not much smell; but in the later stages, as before mentioned, if they have been retained in the intestines, they are often very offensive, but they are not so always. The composition of the Cholera stool has been studied chemically and microscopically. The Cholera stool and the contents of the intestines after death are not exactly alike under microscopic examination, and Dr. Parkes has pointed out the necessity of remembering that Boehm's observations are applicable to the intestinal contents, and not to the discharges collected during life. As before said, the Cholera stool separates on standing into a thin whey-like fluid, and a variable quantity of sediment. The specific gravity of the liquid portion is 1,005 to 1,010. The sedimentary flakes and particles, according to Parkes, Gull, Gairdner, and others, contain organic forms, which are described as follows, lying in a hyaline basis :—

1. Amorphous granular matter and larger granules, often very abundant.

2. Minute bodies having the general character of nuclei, $\frac{1}{2500}$ to $\frac{1}{1800}$ line in diameter.

3. Fine granular cells; some large, some resembling pus cells.

4. A very small quantity of scaly epithelium, generally not easily discernible.

Dr. Parkes states that the actual quantity of sediment is very small; the dried deposit from a pint by measure of a Cholera stool weighed four grains only. The composition of the solid or gelatinous matter of the stools is doubtful: Parkes considers it some modification of fibrine;

Gairdner thinks that it presents the reactions of mucus. It is possibly some modification of mucus.

In contrast with the above, the microscopic appearances of the contents of the intestines found after death, as first described by Boehm, consist of a large quantity of epithelium in various states of aggregation, separate cells, and flakes which are coherent masses of greater or less extent, comprising the coverings of small regions, as apices of villi, or covering the whole villi and their bases in variable quantities. According to the state of aggregation or distinctness of the epithelial particles, and the quantity of fluid in which they are mixed, the appearance of the whole may vary from a rice-water or milky to a creamy, purulent, flocculent, or oatmeal consistence. Dr. Parkes has shown that there are, mixed with these, other organic forms found in the stools. There is very little epithelium found in the contents of the colon. The great difference in the microscopic appearances of the Cholera stools and the contents of the small intestines after death, is in the nearly complete absence of epithelial particles in the stools. The inference from this and other considerations is, that the shedding of the epithelium does not take place during life, but that it is due to post-mortem maceration and detachment.

Chemistry of the Evacuation.—The specific gravity of the liquid part is seldom above 1,012 in the height of the stage of discharge. The reaction is faintly alkaline or neutral. Dr. Parkes, in the February number of the "London Journal of Medicine," for 1849, has given us valuable information as to the composition of the stools, and shown that the liquid portion is not similar to the serum of the blood; that it contains but little albumen, and consists chiefly of the water of the blood with saline matter and a small quantity of animal matter. The following table, copied from his papers, shows at a glance the real character of the discharges:—

Period of the Disease in which the stool was passed.	Specific gravity.	Albumen in 1000 parts.	Extractive in 1000 parts.	Sol. Salts in 1000 parts.	Total of. Solids in 1000 parts
Diarrhœal period	1,012·9	0·466	3·846	9·04	13.9
Diarrhœal period	....	0·29	6·82	5·99	13.1
Early Algide stage	1,009	2·4	1·27	10·98	14.65
Developed and Intense Algide stage	1,009·5	1·18	0·55	9·14	10.87
Developed and Intense Algide stage	....	2·186		7·52	9.706
Developed and Moderate Algide stage	1,008·3	0·27	2·23	8·33	10.83
Developed and Moderate Algide stage	1,005·8	3·2		5·827	8.947
Commencement of reaction	1,014·0	20·84		6·34	27.18
Commencement of reaction	1,008·91	1·48	6·055	9·085	16.62
Relapse	1,017·83	0·855	17·355		18.22
Relapse	....	not weighable.	4·589	3·881	8.47

Nitric acid occasionally gives red reaction in the liquid. This is probably due to a small quantity of bile mixed somehow with the evacuations, but it is not certain what it is.

Blood is rare, but it has occasionally been met with both in the stools and in the contents of the intestines. The pink or reddish discharges often consist only of the coloring matter of the blood, the red corpuscles being generally absent.

Although the albumen and animal matter found in the stools is very small, the quantity of salts contained is considerable. The greatest quantitative loss which the blood suffers is in its watery element. For every 100 ounces passed in the fluid evacuation stage, the loss to the blood in water is 98 to 99 ounces, and of salts nearly or about one ounce. The salts exuded are the chlorides of sodium and potassium, phosphate of soda, carbonate and sulphate of soda, bearing a proportion of seven or eight parts in 1000; a proportion nearly resembling the quantity in the blood within the vessels. The earthy phosphates do not pass through the mucous membrane as in health. In three analyses by Dr. D. Thompson a larger proportion of organic matter was found, but Dr. Parkes's observations probably hold good for the majority of cases. It will be seen that the intestinal surface removes from the blood a large quantity of water, a small quantity of animal matter, and much saline matter, doubtless causing great change in the blood, and in the behavior of the different elements of the blood to each other. The chemistry of the vomited matters has been less studied. In six cases examined by Becquerel, the solid matter in 1000 parts varied from 6·37 to 54·70 per 1000, the albumen from a non-weighable quantity to 31·50 per 1000, the chloride of sodium from 2·35 to 8·24 per 1000. No mention is made of the matters taken into the stomach, nor the period of the disease in which the discharges were collected.

Blood.—The viscidity of the blood in

the algide stage has been mentioned. Chemistry shows that it has undergone considerable changes. The analyses most known are those by Sir W. O'Shaughnessy, Drs. Parkes, Garrod, and Schmidt. They show a diminution of water and a relatively increased proportion of solids. According to Garrod's, taking the maximum of solids in health for males at 240, and for females at 227 per 1,000, he found that the total solids in seven cases were 251, 360, 271, 271, 275, 282, and 284. The blood-globules and albumen were increased; the alteration in quantity of the fibrine was doubtful, but it was less coagulable, and it was probably altered in quality. Dr. Garrod's observations were made on blood obtained, after death, from the cavities of the heart and neighboring great vessels. Dr. Garrod and Dr. O'Shaughnessy differ as to the proportion of salts. The former thought that they bore as high, if not a higher proportion than in health; the latter that they were diminished. Dr. Schmidt's observations were made on blood obtained by venesection. His results show an increased density of the blood and of the morphological elements in proportion to the duration of the exudation process from the surface of the intestinal canal; a relative increase of the solids in the blood, so that after thirty-six hours of the exudation process they reach to nearly half more than their normal proportion. Schmidt thought that the inorganic salts were diminished in the later stages of the exudation process, so that the proportion of organic matter in the serum was doubled. The specific gravity of the fluid is higher than natural; 1076 to 1081 instead of 1062 to 1060. The blood is sometimes found acid. Dr. Garrod thinks that this is due to the impeded excretion of organic acids. Urea in small quantities is sometimes found in the collapse, and generally in the reaction stages. In the main, all the analyses support each other, showing in the collapse stage a greater consistence of the blood, higher proportion of organic solids of all kinds, with impaired coagulability of fibrine, higher specific gravity, occasional acidity, occasional presence of urea, and an undecided proportion of inorganic matter, but nevertheless, probably, a diminution in the absolute quantity of saline elements. In the reaction stages they show often a considerable quantity of urea and softness of the coagula.

Varieties.— As before said, the description of the symptoms given applies to the ordinary cases of Cholera, but we occasionally meet with instances which in some points differ from them. Some of the cases run a much more rapid course; thus in some the evacuation stages are shorter, lasting two or three hours only, and collapse may come on with or without much discharge from the stomach and bowels. Others appear yet more rapidly fatal. Thus at Kurrachee, in 1846, people are said to have died within less than an hour from the time that they were seized. Dr. Milroy states that at Teheran, in 1846, those who were attacked dropped down suddenly in a state of lethargy, and died at the end of two or three hours without convulsions or vomiting, but from a complete stagnation of blood. In these cases it is said that the dose of poison is so strong that death occurs before there has been time for exudation to take place. Mr. Thom says: "Among the first hundred cases which occurred many died in a few hours, and some in less time; one man, I am told, went off in less than an hour. In these vomiting and purging were not always present. Sudden collapse, ending in profuse sweating, were the most prominent symptoms—in fact asphyxia had already taken place. It was often found that the pulse had ceased at the wrist, the eyes turned up, the voice hollow and feeble, before the natural hue had given way to that horrible lividity which is characteristic of the disease, so instantaneously was the power of life arrested." And again, "The next class of cases were those in which the first seizure was equally sudden, and the collapse preceded the vomiting and purging. There were sudden faintness, prostration of strength, restlessness and anxiety, accompanied by vertigo, deafness, loss of vision, alteration or hollowness of voice, weak and slow respiration performed convulsively or in sighs. These were followed by nausea, vomiting, and purging of congel-like stools, sensation of burning heat at the præcordium, intense thirst and desire for something cool," &c.

From such cases as these it has been argued that exudation from the blood is not a necessary part of Cholera. It should be recollected, however, that the amount of discharge voided is no positive measure of the amount of fluid separated from the blood during life, because it often happens that a very large amount is retained within the intestines during life. This will be again referred to.

In some of the cases there is not the same shrivelling of the body as described in the typical cases. The body retains much of its plumpness, and the skin is dry. Collapse, nevertheless, comes on early in these cases.

In a few cases the skin is warm and the perspiration warm, even during the earlier part of the collapse, which comes on early and is complete.

Mr. Twining and others describe cases which appear to have in the first stages more of a *sthenic character* than those usu-

ally met with now, and to which the name of Spasmodic Cholera was, perhaps, more applicable than to those described above. They were cases in which the "actions of the constitution were evidently febrile, and in the febrile stage of the disease were attended with violent and painful spasms, warmth of surface, and free circulation." Dr. Morehead does not find these common in Western India.

Dr. K. Mackinnon relates *another modification* of symptoms which he witnessed in a dreadful visitation of Cholera in the Tirhool jail. He says: "After a check had been put to the vomiting and purging, the voice, breathing, and warmth of skin became natural, the face had none of the peculiar character of the disease, the patients walked about and called for food, saying that they felt well; on feeling the pulse in those cases it was barely perceptible, I think in some cases not to be felt at all. In this extraordinary condition some of the men lived for two days; they all died, and, if my memory serves me, invariably by coma." Dr. Mackinnon does not allude to the state of the secretions. But the feebleness or absence of the pulse with such a degree of muscular strength and general well-doing is remarkable. I have seen some such cases, and they are mentioned by Scot and others.

Dr. John Macpherson mentions two cases with hæmatemesis, occurring in the reaction stage, and in one of them associated with hemorrhage from the bowels.

Choleraic Diarrhœa.—This, though perhaps not a variety of Cholera, approaches that disease so nearly that it may be well to allude to it in this place. It almost always prevails during Cholera epidemics, and sometimes precedes it in a district for weeks or months. It is often difficult to distinguish from Cholera at its onset, and into this, indeed, it often passes. Some of the cases have so much the appearance of mild attacks of Cholera that it is hardly possible to draw a line between them and the real disease. At Paris the name of Cholerine was given to these diarrhœas. It is probable that the diarrhœa is due to a milder dose of the poison than suffices to produce true Cholera. The patient's most prominent symptoms are those of diarrhœa. This begins in the night or early in the morning, and without any assignable cause. The motions are three or four, sometimes six or seven in twenty-four hours—passed without effort or straining or griping. This diarrhœa is generally painless, which circumstance, combined perhaps with an apathetic state of the mind, is the cause of the disease being frequently allowed to run on unheeded. The evacuations are more or less feculent, but very liquid, and generally copious; the color varies from brown to light yellow or pale straw, and when the disease passes into Cholera the motions are increased, and assume the rice-water character. There may be vomiting, and sometimes cramp. There is generally a good deal of sense of exhaustion and weakness. The patient has not much appetite, and is more or less ill. The diarrhœa may last twelve or fifteen days, but may at any time pass into Cholera. The symptoms appear to be amenable to treatment, but the disease may destroy life by exhaustion without passing into Cholera or collapse. It is difficult to estimate the mortality of this affection, because so many of the cases are set down as simple diarrhœa, and when they pass into Cholera they are recorded as Cholera. The mortality is not great, however, of such as remain classified as diarrhœa. The deaths from diarrhœa in the Cholera season of 1854 appeared to vary from 6 to 18 per 1000. The great success in the treatment of these shows how desirable it is to check all cases of Choleraic diarrhœa; by so doing, there is little doubt that many cases of Cholera are arrested.

Cholera Fever.—It has been frequently noticed that at certain periods of the Cholera epidemic, towards its decline, the Choleraic diarrhœas pass into a sort of low fever. In speaking of the Kurrachee epidemic, Mr. Thom says, as Cholera closed its career it gradually changed its type to that of fever; while one out of four or five cases ran into Spasmodic Cholera, the others would terminate in fever, and were registered as such, showing all the characters of a low remittent. Sir Ranald Martin speaks of "Cholera Fever" occurring in Calcutta in 1834, and at other times during the Cholera season, which was remarkable for diarrhœa, and for the tendency which this had to run into Cholera, especially under doses of saline and drastic purgatives, and when these were administered over night. It is not so easy to say what is the connection between these fevers and Cholera. In Calcutta one frequently meets with fevers which begin with diarrhœa. I have elsewhere mentioned this as frequently ushering in the red fever sometimes prevailing there, and it is not unusual for some of the common fevers of children to begin in this way. It is probable that some of these happening to prevail during Cholera times, are influenced by the epidemic constitution of the atmosphere. It is not likely that the fever is a variety of Cholera. We should learn from them the practical lesson of not giving much purgative medicine, even in fever, during Cholera seasons, and especially to avoid salines and hydragogue purgatives, and the administration of any at bedtime likely to operate in the course of the night or morning.

In classifying cases of Cholera, we may,

excluding Choleraic diarrhœas and fevers with Choleraic diarrhœa, arrange them as follows :—

1. Those in which collapse is not perfect.
2. Those which after vomiting and purging pass into well-marked and complete collapse.
3. Those in which collapse comes on with little or no apparent evacuation.
4. The sthenic cases.

The cases most frequently met with are of the second class.

Duration.—This may, including sequelæ, vary from two to three hours in the worst form, to several weeks in those protracted by secondary disease. The time which elapses from the first symptoms of Choleraic evacuations to the period of reaction is more limited. In the Report of the Board of Health on the epidemic of 1853–4, the duration of 1744 fatal cases from the first symptoms is stated to have been an average of not less than 64 hours; the duration of 1856 cases of recovery, 9·06 days. In the general registry of deaths, it is mentioned in the same Report, 9590 fatal cases lasted an average of 2·39 days.

The duration of the stages has been already alluded to; the diarrhœal may scarcely exist (excepting Choleraic diarrhœa), or extend to 24 hours; the collapse stage may terminate fatally in two hours, or it may be prolonged in extreme cases to 70 or 80; the reaction stages may terminate favorably within 48 hours, or may linger on for many weeks, when complicated with fever or other sequelæ.

Mortality of the Disease.—This is very large, and the averages approach each other in different climates and countries. The mortality to strength of a population may vary greatly, but it has been observed for England, that, whether in healthy or unhealthy districts, the deaths to cases are pretty even; the difference in mortality between such places depending more upon insusceptibility of the population in healthy places than on any actual difference of violence of the disease. In India the deaths to cases do seem to vary in different ranks of the army: the officers having a lower mortality than the men. We may say in round numbers that more than 50 per cent. of the attacked recover. In some epidemics the mortality is higher, but in others, lower: in some, 70 to 80 per cent.; in others, 20 to 30 per cent. The mortality varies also with the period of the epidemic. It is generally much higher in the beginning than in the end or towards the end. It varies also in the different stages, being greatest in the collapse stage.

In illustration of the above, we find that in 1832, in England, the deaths to cases were 47 per cent.; in 1848–49, 45 per cent.; and in 1853–54, 46 per cent. In Dr. Ewart's tables for European soldiers in India, for eight years ending 1853–54, it was: for privates, 40·74; officers, 16·66; women, 31·74; and children, 39·163 per cent. For the native soldiery in the three presidencies, for various long periods of years: for Bengal, 30·54; Bombay, 33·06; Madras, 42·91 per cent. In the Report of the Cholera Commission for the epidemic of 1861, we find that the mortality of the European soldiery for that season was very high all over the provinces; the average being 63·8 for all classes in the regiments, but it varied from a minimum of 42·6 per cent. to a maximum of 80 per cent. among bodies of men in cantonments, of strength not below 500. The mortality to cases among the native Indian populations is not ascertainable. For native prisoners in jails for twenty-one years, ending 1853, it was 42·6 per cent. With reference to the excess of mortality in the beginning of epidemics, we find that in 1854, in England, in the fortnight from July 16th to 29th, it varied from a maximum of 59 per cent. to a minimum of 40 per cent.; but in the two last fortnights, ending November 16th, they were only 23·21 per cent. In the severe outburst at Kurrachee, almost all the cases were fatal, and after that they became much more amenable to treatment. Indeed this is the experience furnished by nearly all the epidemics. The greater mortality in the collapse stage may be seen in the results of the Indian epidemic of 1861, in which we find that of 927 deaths, 717, or 77·4 per cent., died in the collapse stage.

An opinion prevails that Cholera is becoming more fatal to European troops in India than it was formerly. Dr. Ewart's tables seem to show, that though the disease is less frequent than it used to be, the ratio of deaths to cases has largely increased. Thus, in Bengal, for eighteen years, from 1818 to 1835, inclusive, the percentage of admissions to strength was 3·25, and the deaths to admissions 26·36, or 263 deaths per 1000 attacks; while for the eighteen years from 1836 to 1853–54 inclusive, the admissions to strength were 2·62, and the deaths to admissions 39·75, or 397 deaths in 1000 attacks; showing an average increase of mortality to attacks of above 13 per cent., or at the rate of 134 per 1000 above the rate of the first series of eighteen years. In Madras, from 1829 to 1838, the deaths were 271 per 1000 cases, and from 1842 to 1851–52, 502 per 1000; or an increase of 231 deaths per 1000 cases. The increase has taken place through all the presidencies, but in Madras it seems to have been the greatest. It is

to be hoped that this increase is not permanent, and that the averages may again descend. In another of Dr. Ewart's tables, the time is divided into quinquennial periods, and it appears that although, in the main, there has been a rise in the mortality within the last and in the later quinquennial period, there were considerable fluctuations in the earlier terms, descending as much as 10 or 12 per cent. in Bengal and Bombay, and then ascending. In Madras the fluctuations have not been felt, but the increase has been maintained during four quinquennial periods. It is possible that a great deal of the apparent difference has arisen from a different method of recording cases. Of late years those cases only in which the presence of the disease was decided, have been entered under the head of Cholera; and cases which would formerly have been classed under this, are now placed in the columns for Diarrhœa. On turning to Dr. Ewart's Table 29, it will be found that there is an increase of cases of diarrhœa in all the presidencies. The returns are not exactly for the same years as those for Cholera, but include them. Thus, in Bengal the returns from 1812 to 1832 give 11·55; and 1833 to 1853–54, 12·15 per cent. admissions to strength; in Bombay, 1803–4 to 1827–28, 6·85 per cent.; and 1828–29 to 1852–53, 12·97 per cent.; and in Madras, from 1829 to 1838, 7·8 per cent.; and 1842 to 1851, 10·3 per cent. It will be seen that in Bombay and Madras, in which the rise in mortality of Cholera is apparently the highest, the increase of percentage of admissions of diarrhœa is also the largest of the three presidencies. It is probable, as supposed by Dr. Ewart, that the diarrhœas have encroached upon the dysenteries, but I think that one may fairly claim a good share of them for cases which would have been called Cholera in the older returns. It would be useful to compare the present with the past mortality of all intestinal fluxes.

Mortality to age, sex, population, &c., has been noticed in the section on Etiology.

Diagnosis.—This is easy in marked cases. Purging, vomiting, anxious countenance, cramps, quick advent of collapse, and profuse sweating, are decisive enough of the presence of Cholera.

There is some difficulty in deciding in the diarrhœal stage. Cholera may be mistaken for bilious diarrhœa, choleraic diarrhœa, purging, produced by irritating drugs or poisons, and the onset of some fevers. We rely for diagnosis chiefly on the absence of any known cause for purging, the absence or slightness of pain or tormina, upon the liquidity and pale color of the stools, and copious rush of fluid from the bowels; the early tendency to exhaustion, and the frequent beginning, in the early morning. If the patient is passing brown feculent matter not very liquid, and if there be griping and colic, and a probable cause in improper diet, we may be pretty easy, and the disease will probably be common diarrhœa. But we should nevertheless watch the patient carefully for a few hours. Choleraic diarrhœa is less violent in its onset than Cholera, and the stools generally contain bile, even after the disease has lasted for days. There is seldom much or any pain in this. It should always be treated as Cholera in the beginning. Vomiting helps in the diagnosis, but it is sometimes absent for the first few hours, and is sometimes present in diarrhœa. The vomiting in Cholera is fluid and colorless generally; that of bilious diarrhœa often consists of undigested food or biliary matter.

The diarrhœa in the beginning of fevers is often very exhausting, and at first it may be difficult to distinguish it from the commencement of Cholera. Generally, however, there is less exhaustion; and though there may be a tendency to collapse, this is not deep, and in a few hours the pyrexial stage comes on.

All surgeons in charge of hospitals must be familiar with indications afforded by the countenances of patients even in the earlier stages. Men are often seen looking somewhat anxious, with the face pointed, and perhaps somewhat shrunk and mottled or leaden-colored. On being questioned, it will perhaps be found that the patient has watery purging on him; a closed stool at his bed-side will, perhaps, be half or two-thirds full of the brownish or pale liquid, and he will be, indeed, in the first stage of Cholera, though ignorant of it, and making no complaint. A glance at a man's face will often save his life, and no man, when in or out of hospital, whose countenance shows this change, should be passed over in a routine way, however trivial his own complaint may be, or although he may be engaged, as sometimes happens, in his usual occupations.

Cases of irritating poisoning often occur either from accident or design, and it is always well to be alive to such facts when called to cases supposed to be Cholera, and not take it for granted that all cases of vomiting and purging are those of Cholera.

It cannot be too strongly impressed upon the young medical practitioner that in every case of diarrhœa which he attends, in seasons of Cholera, he should ask to see the evacuations at the very first visit. In India fortunately there are few privies or cesspools to private houses, so that this inquiry is not so difficult as it might be in England. If the stools have been taken away, the next ones should

be reserved. This is most important for diagnosis, and for the satisfaction of the medical man's own conscience it should never be omitted. Neither rank, nor sex, nor age of the patient should be a cause of the neglect of this.

It should be remembered that many patients brought into hospital with low adynamic fever are sufferers from the sequelæ of Cholera, and that inquiries should be made as to the mode of the beginning of the disease for satisfactory diagnosis, if not for treatment.

PATHOLOGY.—*Morbid Anatomy.*—This must be considered after death in the collapse and in the reaction stage.

(*a*) *In Collapse.*—The surface is generally bluish or mottled, especially in the dependent parts, and there are often subconjunctival ecchymoses. The extremities are shrivelled. The temperature, so low during life, often rises a little after death, and the body cools slowly. According to Dr. Gull, Mr. Barlow and others have noticed a rise of two or three degrees of Fahrenheit above the temperature observed just before death. Rigor mortis comes on quickly, and lasts quite as long as in other diseases. Muscular contractions have been sometimes observed, and have in rare cases been sufficiently strong to alter the position of a limb. They may come on within the first few minutes after death, and last two or three hours. Putrefactive changes are not hastened in Europe. In India they are not more rapid than in other diseases. The abdomen often feels doughy and dull on percussion, except in the upper parts.

Blood— Organs of Circulation and Respiration.—When death occurs in collapse, the disease has not lasted long enough to cause organic changes. Such changes as there are, are chiefly in the distribution of the blood. Ecchymoses are frequent. They are met with under the serous membranes, as the pleura and the pericardium; under the mucous membranes of the small and great intestines, around the dura mater of the spinal cord, and occasionally in other places.

The capillaries of the surface are empty, and the blood is chiefly found in the large vessels of the lungs and right side of the heart, and in the veins and various capillaries of the intestines. Dr. Parkes was the first to point out, in his work on Asiatic Cholera, the real seats of accumulation of blood. Most persons who have had opportunities of making post-mortem examinations of Cholera cases will confirm his statements. It was previously supposed that the lungs and heart were gorged with blood, but he pointed out accurately what was the precise situation of the congestion. He showed that the gorged parts were the vessels of the right side of the heart and the pulmonary artery in the roots of the lungs, from the right side of the heart to the smaller branches; and that the smaller vessels, the pulmonary capillaries, the pulmonary veins, and the left side of the heart, were nearly empty: in fact, that the blood was not arrested in the capillaries of the lungs as in common asphyxia, but in the arteries short of them. On section there was free bleeding from the roots of the lungs, but there was little or none in the peripheral parts; they were generally exsanguine. He says: "On cutting through the roots of the lungs a quantity of blood usually escaped from the divided vessels, and particularly from the *pulmonary artery;* in one case the quantity of this was two pints, in one case one pint, in one twenty-four ounces, in one three ounces, in one six ounces, and in nine between two and five ounces. In fourteen cases the lungs were completely collapsed, appearing in some cases like the lung of a fœtus. In three cases they were considerably, in eight slightly, collapsed; and in the remaining fourteen cases the collapse was in some cases altogether and in some partially prevented by old adhesions." The absence of blood in the tissue or substance of the lung was generally pretty complete, though in some instances there was more redness in the periphery than in others. The small amount of blood in the lungs seems proved by their deficient weight. Thus, he found that the average weight of the right lung in twenty-two Europeans was fourteen ounces, and of the left twelve ounces six drachms. Dr. Clendinning gives the average weight of both lungs at forty-six ounces, and Dr. Reid at forty-three ounces, showing a deficiency in the Cholera cases of fifteen to nineteen ounces. The lung tissue was flabby, with little crepitation when not collapsed, so that it may be considered to have been in an extremely anæmic state, owing to an arrest of blood before it had reached the capillaries and smaller arterial twigs. The color of the lung on section was pale or dark, or an admixture of both. Some writers report a congested state of the posterior parts of the lungs; probably from hypostatic congestion. The right side of the heart was generally full and distended, the blood coagulated, but not firmly. Parkes found the blood incoagulable in rather more than one-fourth of his cases. The left side of the heart was nearly empty, or containing only a little black blood with a few loose coagula. In following out the situations of congestions we find that the larger hepatic veins and the branches of the portal vein, generally those leading from the stomach, duodenum, and small intestines and their small ramifications, are full both on the mucous and peritoneal

surfaces of these organs. The blood is generally of dark color, but tur s brighter on exposure to the air, or to the action of the matter in the small intestines. The chemistry of the blood has already been alluded to. There is some difference of opinion as to the state of the coagulability of the blood. Parkes, Virchow, Briquet, and Mignot have noticed either that it coagulates imperfectly, or that there is a loose state of the coagulum in the great vessels. I think that I have generally observed the same. Dr. Gairdner, however, says that the coagulation in the vessels takes place much as in other diseases.

In the lungs themselves there is little change beyond those due to distribution of the blood and the collapse of the lung tissue. The muscular tissue of the heart is generally found rigid or firm.

Abdomen.—The visceral peritoneum is generally much congested; the venous capillaries darkish, becoming bright after exposure to the air; the surface duller than natural, and sometimes bedewed with a slimy moisture.

Stomach and Intestines.—Mucous membrane sometimes congested. Mucous membrane of small intestines often much injected with fine arborescent vascularity. There is little organic change in the coats; the mucous membrane may seem œdematous, and the folds rendered prominent. Boehm pointed out the prominence of the glandular structure of the mucous membrane of the small intestines, and notably of the lower part of the ileum. Thus, Peyer's patches and the solitary glands are often enlarged, the solitary more decidedly than the agminated glands. Ulceration not observed in simple cases. Occasionally patches of a slight grayish exudation have been noticed. These have been called diphtherial. If the exudation in throat diphtheria is to be taken as the pattern, I should hardly call the name a good one. These patches are not easily removed, as the diphtherial layer is from the throat. The exudation in Cholera seems more like the granular patches which are sometimes seen in the lower three or four inches of the ileum in severe cases of dysentery, and in a more highly developed form in the drab-colored granular patches in the colon in dysentery, and which cannot be removed without some disintegration of the superficial part of the membrane on which they lie. This appearance is rare in Cholera. In many cases of Cholera, however, there is little or no congestion or decided morbid change discoverable on examination of the mucous membrane or glandular structure. The contents of the intestines have already been described. They may fill the intestines completely, or they may be but a small quantity, the consistence varying from that of an ordinary Cholera stool to a creamy or pasty, gritty-looking grayish mass, or fluid mixed with gelatinous or fibrinous lumps or flakes, more or less adhering to the intestines. There is sometimes, but not often, a tinge of yellow, and sometimes, though rarely, a large quantity of grumous blood without any kind of ulceration being traceable.

Colon.—In the majority of cases an absence of injection of the vessels. In a small number there is venous congestion, seldom general, but in patches. The mucous membrane is not so œdematous as that of the smaller intestines, though it is sometimes somewhat swollen. There is not much epithelium mixed up with the contents. In some rare cases large ecchymoses, leading to gangrene of the mucous membrane, have been seen.

Liver.—Great venous trunks generally full of viscid blood. The parenchyma pale and flaccid, and often smaller than natural, with some but not constant wrinkling of the capsule. There is not often general congestion of the organ. Microscopic appearances normal. Gall-bladder generally full of bile.

Spleen generally smaller and more flaccid than natural. Some writers have described both the liver and the spleen as being congested, but this does not seem to me by any means frequently the case.

Pancreas mostly natural. The mesenteric glands are often enlarged or pale.

Kidneys.—These are generally congested, the veins, especially of the papillæ, extending outwards into the cortical substance, which is enlarged. The epithelium is generally natural in shape, very granular, probably from some protein deposit.

Brain and Membranes.—Nothing special or different from the condition in many other diseases in which the brain is not directly involved.

In summing up the post-mortem appearances in collapse, we find an alteration in the consistence of the blood; and that some of the great internal veins and the pulmonary artery are loaded with it; a great deficiency of blood in the capillaries generally except in the majority of cases in those of the intestines and kidneys; generally some prominence of the glandular structures of the intestines, and post-mortem displacement of the epithelium of the small intestines, and in many instances collapse of the lung tissue.

[Besides these appearances, many observers have reported finding the left side of the heart (if examined a few hours after death) firmly contracted; the gall-bladder full of bile; the urinary bladder contracted and empty. Dr. George Johnson has ascribed the asphyxia and collapse to a spasmodic constriction of the pulmonary artery; causing anæmia of the lungs, and deficient aëration of the blood. That this is a part of the pathology of the attack, is

almost certain; but there is good reason to believe it to be only a part of a *general* affection of the involuntary (as well as voluntary) muscles, under the influence of the *materies morbi* upon the nerve-centres of organic life and the spinal cord.[1]—H.]

(*b*) *In Reaction Stages.*—The morbid appearances vary with the nature of the secondary causes of death. It will not be necessary to give a full description of these changes. In the cases of death in imperfect reaction they do not vary much from those found in collapse, except perhaps that there may be more general congestion of the lungs, more decided prominence of the glandular structures of the small intestines, and injection of the intestinal mucous membrane.

In persons dying of some of the consecutive febrile conditions, the intestines and stomach are often found vividly injected, the mucous membrane softened. The colon sometimes participates in this, and the mucous membrane is sometimes œdematous and thrown into folds, and the so-called diphtheritic exudations are met with. In some of the cases not marked with symptoms referable to the alimentary canal there is less congestion and œdema of the small intestines, as has been noticed by Reinhardt and Leubuscher.

In the uræmic cases the kidneys, when death appears early, are full and large, of purplish color, dripping blood on section; in the latter periods pale, perhaps œdematous, infiltrated with granular matter of pale color; are easily torn, and the epithelial tissues more opaque than natural, and filled with granules mixed with oil-globules. The kidneys are somewhat similar to those in cases of scarlatina. In other organs the appearances are like those of ordinary acute uræmia. Secondary inflammations having sometimes existed, we may find evidence of them in the tissues affected. Urea is found in the blood in considerable quantities. Dr. Garrod found as much as 1·14 part per 1000. It is readily found in blood collected from the right side of the heart.

The other changes possess no special characters. There may be signs of low inflammation of lungs, pleura, pericardium, and peritoneum, and of much congestion of the lungs. The brain and membranes sometimes full of blood, as in low fevers, in which the blood is in fault. The blood coagulates loosely. Of course to these must be added gangrenous patches, bedsores, ulcers, ulcerated corneæ, boils, suppurated or swollen parotid glands, gangrene of lungs, ecchymoses, and purpuric or scorbutic patches, which occur occasionally.

Pathology During Life.—From the study of Cholera as shown in the symptoms and post-mortem appearances, we pass to an attempt to account for them. In the section on Etiology the nature of the cause has been discussed. We have now to suppose that a poison has entered the blood in some manner, either by the channel of the lungs or intestinal surface. The poison acts through the blood, possibly in the manner of a ferment; but we do not know whether it acts by a catalytic influence on the blood mass, or whether it multiplies itself in the blood, or not; we are very much in the dark. However, if we cannot trace the minute action of the poison we are able to guess at some of its effects. It appears that at least two great sets of capillaries and small arteries are involved in them: those of the lungs and intestines. They seem to be very differently influenced by it. In the lungs very little of the blood passes freely through them in the algide stage. In the intestines an enormous quantity of certain of the blood elements passes through the capillary walls in the exudation period. In both of these sets of actions, parts of the nervous system appear to be under a morbid influence. In the lungs the muscular fibres of the small arteries seem thrown into a state of contraction. In the intestines a sort of paralysis of the smaller arteries and capillaries seems to exist, much as occurs in the sections of the sympathetic nerve in the neck in Bernard's experiments. That the morbid action in the lungs is of nervous character seems probable from the absence after death of any discoverable mechanical obstacle to the passage of the blood, from the paroxysmal nature of the dyspnœa at first, and from the ease with which the pulmonary circulation is re-established when recovery begins. The nervous character of the actions in the intestines seems probable from its analogy with the results of Bernard's experiments on the sympathetic, in some of which a section of that nerve in the neck caused the

[1 "The bloodvessels of the whole alimentary canal press rigidly upon their contained fluid, and force its serum out into the stomach and bowels; whence it is, by spasmodic ejections, thrown out. The very skin is, by its involuntary muscular fibres, as well as by vascular constriction everywhere, drawn tightly and closely upon the body. The voluntary muscles suffer with cramps. All is cramp, cramp, within and without. The brain is almost in anæsthesia during the collapse—no delirium, but apathy—as from cerebral anæmia. The blood, so compressed, grows thick as tar—it scarcely flows, is not aërated, and cyanosis follows; it is detained in the capillary net-works of the interior organs, in which congestion is found after death. Cholera is, then, a poison-spasm; *a ganglionic tetanus.*" *Essentials of Practical Medicine*, 4th ed., p. 400.—H.]

surface of the skin supplied by the vessels under its influence to become bathed in sweat. It is a matter of dispute whether the disturbances in the lungs and the exudation through the intestinal walls are both effects of the poison, or whether, exudation into the intestinal canals having taken place, the pulmonary symptoms may not be due to the altered condition of the blood, caused by its being inspissated through the exudation of so much of its fluid and saline elements. It must be granted that symptoms similar to collapse may be produced by poisons without any purging. I have seen people under the influence of malarious poison in Calcutta lie for hours as cold and pulseless, and as embarrassed in the breathing, as in Cholera. In gangrene, too, a somewhat similar condition may come on. It appears, then, that symptoms similar to those of the cold stage of Cholera may be produced in certain diseases without any exudation or loss of fluid from the blood. Nevertheless, it is doubtful whether we can say positively that exudation is not necessary to produce the collapse in Cholera. It is true that it is said that some of the most rapidly fatal cases of Cholera are those in which there is no purging, and that therefore we must seek for some other cause of the algide stage. But it does not follow, that because there was no purging there was no exudation into the intestines. The exudation is sometimes poured out, and retained there, and we should never for a moment confound transudation with purging. In numerous instances in which there has been an absence of evacuations, post-mortem examination has shown the intestines to be full of fluid. In others, the symptoms of full collapse have come on before the vomiting and purging. Among these a remarkable case occurred in the Middlesex Hospital, and is reported by the Committee for Scientific Inquiries in the Cholera epidemic of 1845. A child was admitted in a state of collapse, having had only one motion and vomited a little. Soon after admission it was copiously purged. In this, as in the other cases, the exudation had taken place, but had been retained, and but for the evacuation, after the algide stage had fully formed, might have been set down as a case of collapse without transudation. In many cases reported to have died or to have passed into collapse without evacuations, we find the symptoms before collapse to have been similar to those caused by some drain upon the system, as has been mentioned under the head of "Varieties" of the disease. It certainly is not common for the practitioner in India to meet with these cases of absence of evacuations. Every now and then one hears the patient's friends say that such an one threw up a little water from his mouth and sank into collapse, or died at once; but in actual practice they are not often met with face to face, as far as my experience goes. Further careful observations are required to be made upon these cases, in which after death the contents of the intestines should be measured. It may after all turn out that the supposed absence of discharges may be in cases in which there is retention and not absence of exudation. The rapid improvement that follows injections into the veins in collapse strongly supports the view of the dependence of this upon the loss of fluid from the blood. It is hardly possible that this resuscitation of the patient from the depths of exhaustion can be accounted for, as Dr. G. Johnson contends, by the warmth of the injected fluid. Although the subject still demands careful observation, there seems great probability that the collapse has a relation to the transudation, if not to the purging. The cases which at present most oppose the view are those in which the body remains rather plump in the collapse stage; but I think that in these there is generally little sweating, and therefore there exists one drain the less for the superficial capillaries. I am far from denying the adequacy of the Cholera poison to produce the algide stage by its action on the vessels of the lungs, or by a sedative influence upon the centres of circulation and respiration, though I think there is not yet full or complete evidence of it.

It has been argued that the vomiting and purging are salutary, and that they eliminate the poison. It is very questionable whether fluxes produced by organic poisons are necessarily eliminative of the actual poison that was introduced into the system, or of its products. Whether the exudations in Cholera are eliminative or not, there can be but little doubt that they are very destructive methods of cure. If this purging were beneficial, we should expect to see that cases of Cholera with preliminary diarrhœa would be slight; whereas we often see that a man has diarrhœa for a week or more, quite as copious as could be produced by a few doses of castor oil, and yet these very cases often pass into profuse purging, collapse, and death. Most men who have had much experience in Cholera will say that when they succeed in checking the discharge, before collapse comes on, their patients are saved. They justly fear the result of active purgative medicines given in seasons of Cholera. It is hardly possible that experience can have gone quite wrong in these matters. We do not know how the poison may be eliminated. For aught we know, it may be decomposed in some way, and not eliminated at all in its entire state.

In collapse the patient's state is a re-

markable one. Circulation is nearly stopped, and indeed all vital functions reduced to a minimum, as shown in the description of the symptoms. Little or no natural chemical action goes on in the system; but it does not follow that some chemical changes do not go on in the blood itself, as they might do out of the vessels at a similar temperature, and in prolonged collapse this may furnish some impurities to the many mingling with it. The altered density of the blood consequent upon the loss of so much water must have brought about many changes, involving both the intercellular fluid and the blood-globules, and materially affecting the relations between them. Schmidt supposes that after about thirty-six hours the morphological elements of the blood are nearly one-half more than the normal proportion. The proportion of inorganic salts is not increased, except perhaps for the first four hours, during which the water passes out more rapidly than the salts; after eighteen hours they sink much below the natural standard, and still more in thirty-six or forty-eight hours. The result is that the proportion of albuminates in the blood-globules is increased by one-half, and in the intercellular fluid to double of what would be normal. He considers that the solids are retained in the vessels with more force than the water, the organic solids with more force than the inorganic, the phosphates with more than the chlorides, and the salts of potash with more force than the salts of soda. In the exosmosis from the globules into the intercellular fluid, he supposes that the same laws of the succession of diffusion of their contents prevail; the chlorides of the alkalies being the first salts to transude both from the intestinal capillaries and from the blood cells into the liquor sanguinis. It is probable that the blood, thus altered in the relations of its component parts one to another, does not recover its natural state at once, even after fluid has been absorbed into it during the reaction stage, and that the abnormal state thus produced may help to cause some of the consecutive disturbances.

[Amongst others, Binaghi, Loder, Orton, Delpech, Lizars, Coste, Favell, Greenhow, G. Johnson, and C. W. Bell have called attention to the indications of disturbed ganglionic innervation in Cholera. Dr. C. W. Bell's expression is, that it is not an adynamic, but a sthenic (spasmodic) collapse.—H.]

In healthy reaction, full purification of the blood by the liver, kidneys, and intestinal surface occurs, and this is promoted by free absorption of water; and the normal composition of the blood is further brought about by absorption of the saline matter still lying in the intestines or administered purposely. Dr. Morehead has observed that the more rapid the reaction without return of the secretions, the greater the probability of secondary fevers, &c. Doubtless, the more rapid the reaction the more rapid the tissue changes, and the more rapid the accumulation of effete matter in the blood; and hence the greater urgency that there should be free secretion and excretion. Some writers attribute the consecutive morbid actions, or at any rate a great part of them, to the stimulants wrongly given in the collapse stage. Injudicious stimulation and drugging may certainly still further poison the blood by themselves, or, by inducing over-speedy reaction, they may assist in bringing on fevers; but I have seen a very large number of people suffer from secondary evils, who have undergone no treatment whatever, and am convinced that the natural progress of the disease is, of itself, sufficient to bring them on. Judged by the coloring matter in the intestines, the functions of the liver appear to be more readily restored than those of the kidneys. Disturbances of the liver are rarer than those of the kidneys. We can scarcely expect the secreting functions of any of the organs to be restored immediately under any circumstances, and some hours must be allowed for the return of normal action. Prolonged cessation of urinary secretion, of course, ends in coma and death. In some of these cases the worst symptoms may be delayed by the urea being excreted through the surface of the intestines and stomach. It is probable that in some of the consecutive fevers, without there being a suspension of the separation of the urea, there may be some diminution of the amount passed in the twenty-four hours, and there may be at the same time a loading of the blood with creatine, creatinine, and other effete matters. To some of the forms of blood-poisoning are due the parotid swellings, purpuric spots, boils, and aplastic inflammations of various parts. To the deficiency of available elements in the blood is due the defective nutrition of the cornea, and perhaps the gangrenes. I have not seen any reason to suppose that any of the last are caused by embolism. The causes of the dyspnœa may be two—spasm, or constriction of the smaller branches of the pulmonary artery and collapse of the lung tissue. It is probable that constriction of the arteries may frequently be the cause, especially in the instances in which the embarrassment is temporary and paroxysmal, one set of arteries being contracted at one time, and one at another, in areas of varying sizes. It seems likely that the permanent dyspnœa may be caused by collapse of the lung tissue, especially in the later stages of the disease. Collapse of lung tissue is not necessarily permanent, but the solidified

part would probably continue unexpanded in the feeble inspiration of the stage of exhaustion, if any considerable area of the lung were affected.

The purpuric conditions, besides the causes already mentioned, may be produced by large doses of calomel. When this was used in scruple doses to check vomiting, it was not uncommon to give two or three such doses. One can imagine that forty or fifty, or even fewer, grains of calomel, accumulated in the stomach or intestines, might be absorbed in reaction, and be ready to add its liquefactive action to the other blood-dissolving agents already at work. Cases of this sort, which I have seen, have made me cautious about the use of large doses of mercury in Cholera.

The anæmia and debility which succeed Cholera are readily accounted for by the profound disturbance to which all the blood elements have been subjected, and which may destroy blood-globules, or interfere with their renewal.

Chronic diarrhœas and dysenteries may depend upon the irritation which has been set up in the small and large intestines respectively; producing in some instances ulceration and softening of the mucous membrane. The softening of ecchymosed blood may also produce symptoms of diarrhœa or dysentery, by giving rise to patches of inflammation or disintegration of the mucous membrane.

I once saw a case of chronic albuminuria which appeared to have originated in an attack of Cholera a year previously.

PROGNOSIS.—*Favorable signs.*—In the *Diarrhœal Stage:* The not passing rapidly into collapse, the pulse keeping some strength, the countenance being tranquil and natural. In some cases the prolonged diarrhœal stage may be fatal without collapse. These are probably choleraic diarrhœa.—In the *Collapse Stage:* The collapse not becoming deep, the pulse remaining in the brachial, respiration tranquil or not much embarrassed, without deep lividity, the algide period not lasting beyond six or eight hours, the cessation of jactitations, and the tendency to quiet dozing or to snatches of easy sleep, gradual return of circulation, progressive improvement in strength even if gradual, milkiness or grayness of stools. The continuance of scanty stools is not of much importance; if the belly is at all tumid, they are advantageous, because they free the intestines of matter already exuded into them. The more mucous and gelatinous they are, the better it is that they should pass away. Cessation of vomiting and purging without reaction after a little time is not important.—In the *Reaction Stage:* Early establishment of the secretions, within twenty-four or thirty-six hours, refreshing sleep, and tolerance of food and drink.

Unfavorable signs. — Quick disappearance of pulse from the radial or brachial arteries, early embarrassment of respiration, and lividity of surface, profuse sweating. In the *Reaction Stage:* Suppression of urine, advent of typhoid symptoms, bloodshot eyes, secondary inflammations. Coma is always dangerous. Want of progress in the symptoms of improvement, and continuance of diarrhœa or vomiting, are unfavorable. Pink or bloody stools or hemorrhage from the stomach are almost always fatal signs. Disproportionate muscular strength without corresponding improvement in other functions is not necessarily favorable. Allusion has already been made to this under the head of "Varieties."

TREATMENT.—This is most successful when commenced early; before collapse. In full collapse it is pretty certain, as remedies cannot be absorbed, that they cannot be of much use. Still, it must not be supposed that all treatment is useless in Cholera, or even that it is useless in all the states of the stage of collapse. It cannot do much good in perfect and complete collapse, but in all the other stages of the disease it is as beneficial as in any other severe and dangerous illness. Excluding collapse, judicious management, free from routine, and adapting the treatment to the individual case, is of great importance. Cholera is not merely a disease of vomiting and purging; and this will have been apparent from the foregoing pages.

The treatment should be considered in relation to the evacuation, collapse, and reaction stages.

Evacuation Stage. — The discharges should be checked, if possible. I believe that the great object of treatment is to restrain the passage of exudation from the blood into the intestines. The remedies used for this generally check the vomiting and purging, so that in the condition of these, taken with the state of the pulse, we have a sort of indication of the degree in which the transudation is interfered with; a sort of indication, because there is not always any close relation between the time of the transudation and the discharges from the bowels. The first medicine given should be a full dose of opium—to an adult 2 grains; in India, 1 to 5 grains of calomel are generally combined with it. I do not know that the calomel does good; it does no harm. If this dose is retained, probably little more medicine will be required. We must not expect the purging to cease immediately that the medicine is swallowed. Perhaps one or two motions will pass away after the dose has been given. Soon after the opiate, if it has been retained, say in half

an hour, in my own practice I give an astringent, generally the following mixture:—℞. Plumbi acetatis, gr. xxx; acid. acet. ℥x; aquæ distillatæ, ℥vj—1 oz. or ½ oz. every half-hour or hour. At the end of an hour from the administration of the first dose of opium, if the purging persists, I give one grain of opium and continue the astringent. The sugar of lead mixture may be given every half-hour or hour, if the purging continues smartly; but if it seems inclined to cease, and the pulse keeps good, the mixture may be left off, or given at wider intervals. It is necessary that the medicines should be presented to the stomach in the simplest and least irritating form. In the form of pill, opium is probably more easily retained than laudanum, which often nauseates; but when a pill is not at hand, forty minims of tincture of opium should be given in a little cold or iced water, or in a little brandy. The acetate of lead mixture is nearly tasteless, or may be made so by the addition of a little iced water. Dr. Graves's acetate of lead pills may be given instead of the mixture, but are probably less active than the liquid medicine. These pills are made as follows:—Acetate of lead, 20 grains, opium, 1 grain, divided into twelve pills, of which one may be given every hour. It would be better to increase the lead to 2 or 2½ grains in each pill. Tannin or gallic acid may be given instead of the lead, but they are more likely to cause sickness. If the above medicines check the discharges, all danger will probably be over in a few hours. Consecutive disease is not likely to follow such a simple case. When there is vomiting there is more difficulty. The pill should be given as before. If rejected in the course of a few minutes, we shall probably find it in the discharges. It will be well to wait ten to fifteen minutes before giving more opium, and (while waiting) to apply a large sinapism to the epigastrium. If we are satisfied that the first pill has been rejected, we should repeat the dose of two grains in about a quarter of an hour after the vomiting. If we are uncertain of this, we should in half an hour give one grain only. After removal of the sinapism, if the stomach is quiet, the acetate of lead should be given in mixture or in pills. Perhaps half-doses of the mixture may be retained if the full quantity is not. When the stomach is very irritable, a solution of half a grain of muriate of morphia may be injected hypodermically. When simple opium cannot be tolerated by the stomach, hydrocyanic acid and other anti-emetic drugs are rarely more efficacious. Stimulants are not wanted in this stage unless the pulse begins to flag; then a little brandy and water, or, better still, iced water, should be given, a teaspoonful or two at a time. Champagne is sometimes tolerated, when other wines are not retained. In the majority of cases a little brandy diluted with ice dissolved in it, or with plain cold water, is the best stimulant. Cramps are generally relieved by friction with chloroform, or with the hand; if severe, by the inhalation of small quantities of chloroform. The doses of opium recommended may of course be modified. If the symptoms are mild, one grain may be given instead of two. Some prefer to give smaller doses every half-hour or hour, but it seems better to give a decided dose at the onset, and not repeat it often, and not to give more than three grains in all, within the first three hours, unless the medicine has been vomited; then the doses should be managed so as to make good the quantity supposed to have been rejected. As a general rule we should limit the quantity of opium to three grains and the acetate of lead to ten or fifteen grains in the first three hours. If the disease should show signs of yielding, we can diminish the doses, or stop them altogether. In some cases, if collapse does not come fast, the evacuations still proceeding, the opium may be continued in half-grain or grain doses about once in three or four hours for two doses, beginning three hours after the third grain has been given. The lead mixture may be continued in half-ounce doses every two hours for five or six doses if necessary. When the collapse is progressive, no opium should be given after the third grain, and, indeed, if it seems to be approaching fast even the third grain should be withheld.

[A method of treatment which met with encouraging success in Philadelphia in 1849, and in succeeding epidemics, as well as in Columbia, Pa., in 1854, was what may be best called *antispasmodic* medication. It was first suggested by the late Prof. W. E. Horner, of the University of Pennsylvania. It consisted in the administration *every five minutes*, in incipient collapse, of small doses of chloroform, laudanum, camphor, and ammonia; each dose being accompanied by a small piece of ice to promote its retention by the stomach. At the same time sinapisms were applied to the epigastrium and back, and the limbs were rubbed with whisky and red pepper, to produce reaction. A number of cases were, by this treatment, restored from a well-developed state of collapse. It is difficult for me, after practical acquaintance with this mode of treatment and its results, to believe that any other method of practice can be more promising in Cholera.[1]—H.]

[1 Dr. Hodder, of Toronto, in 1850, injected several ounces of strained milk into the veins of three moribund cholera patients. Two of them recovered.—H.]

Collapse.—When this is complete, opium should be entirely avoided, and sugar of lead would be useless. The exudation is probably quite stopped, and the evacuations now passed are merely residues of matter previously poured into the canal, and add nothing to the danger or exhaustion of the patient. In the worse stages of collapse, stimulants are of little avail. Æther and ammonia properly diluted may do no harm. If they can be retained, small doses may be given every half hour. A little weak brandy and water is sometimes retained better than any other stimulant. But the administration of stimulants in the stage of collapse requires great care and discrimination. If they are of use, they make themselves felt in the pulse. If it revives under them, ever so little, and falls back or disappears in their absence, they may be continued cautiously; otherwise they, or at all events the alcoholic stimuli, should not be persevered with. The dependence of the pulse upon the stimulants is certainly sometimes seen. Unfortunately, if stimulants, such as brandy and wine, do not act beneficially, they are likely to be hurtful in the after stages. A large quantity of them may be collected in the stomach or intestines, and when absorption begins they may be taken up into the circulation, causing undue rapidity of reaction before secretions can be formed, and so adding to the mischief already at work in the blood. So that we may be pretty sure that if they do not work for good they will be hurtful. Nothing is more pernicious than the system of pouring large quantities of brandy into a pulseless patient in Cholera. Sometimes when the irritability of the stomach has gone off he will, in the course of two or three hours, swallow a large quantity of brandy, water, æther, ammonia, sago, wine, &c., and the friends perhaps congratulate themselves upon the quietness of the stomach; the pulse remaining absent nevertheless. In the course of time the patient grows very uneasy, and presently vomits the whole accumulation, perhaps two or three pints. Such cases as these show the uselessness of overloading the stomach in the torpid condition in which the patient is in collapse, and what a reservoir of mischievous elements may be provided against the return of the circulation. We often see cases in which stimulants, mixtures, &c., are given and as constantly vomited. These are often instances of too much medication. The irritable stomach is made more so by the drugs, and on leaving these off the vomiting ceases. The dependence of the vomiting upon the time of giving the doses should always be inquired into. We ought not to take it for granted that the vomiting is always the effect of the disease; it may, and I have so seen it scores of times, be due to the administration of remedies. A few teaspoonfuls of brandy and water and a few doses of æther and ammonia will do no harm in the pulseless state, but it is vain to expect to do good by constantly pouring them down the patient's throat. The best thing for him in the stage of full collapse is to gratify pretty freely his instinctive desire for cold water. This cannot do harm: a little may run off by the bowels, or be vomited, but if any remains to be absorbed in the reaction stages it will have the best possible effect. It is of no use to keep up the patient's vomiting by giving him as much as his morbid thirst impels him to swallow; but a considerable quantity may be given in the course of two or three hours by tablespoonfuls or wineglassfuls at a time; the colder the better. If ice be available, it will be better to give iced water, or to place pieces of clear ice in the patient's mouth frequently. This quenches his thirst and seldom causes vomiting. There need be no restriction placed upon the quantity of water given beyond that it is advisable not to provoke too much vomiting by it. Although I have urged the uselessness, nay mischief, of pouring down drugs and stimulants into a patient in the state of perfect collapse, there are certain conditions of collapse in which when given cautiously they may be of great use. The cases are those in which collapse is not perfect, in which the circulation still goes on, though feebly, throughout the algide stage. In these the volatile stimuli, æther and ammonia, in mixtures, and weak brandy and water, are certainly beneficial. They maintain the strength, as they do in any other diseases with exhaustion. Ten to twenty drops of liquor ammonia, or thirty of sulphuric æther properly diluted with iced water or cold water, given every half hour or hour, with an occasional tablespoonful of brandy and water, may be continued while the pulse seems to be maintained by them. In these cases the Cholera mixtures and Cholera pills so often used in India are of service. They are made up in various ways, but contain generally such stimulants as æther, ammonia, tincture of camphor, and essential oils, as cajeput, aniseed, cinnamon, peppermint, &c., or some of them. Most of them also contain laudanum, but it would be better to omit this, and add it to the dose when considered necessary. Cholera pills can be made of camphor, two grains; asafœtida and long pepper, each one grain, with a little mucilage. A pill should be given every one and a half or two hours. They are useful in the same circumstances that the Cholera mixtures are. When acetate of lead has been used as the astringent, the liquor ammonia should be used in preference to

the carbonates of ammonia, or their preparations. It will be observed that the principles advocated in the use of stimulants are that we should be guided in the administration by the pulse. While that remains, absorption and other vital actions go on, although perhaps in a diminished degree; and while these continue, stimulants may assist the patient through the crisis, and may prevent his passing into the pulseless state. The volatile and diffusible stimuli are less pernicious than alcohol, but a small quantity of the latter is useful, and should be occasionally given. There are few persons to whom three ounces of brandy may not be safely given during eight or ten hours if their circulation responds to it,—in some cases even more, but probably not often. As soon as it is found that the pulse maintains itself without them the stimulants should be withdrawn; the alcoholic first. Administered in this way they will not do harm; they are taken into the system by degrees, and their action is spread over a sufficient space of time, and they thus act very differently than they might do if allowed to accumulate in large quantities in the stomach, to be absorbed all at once and added in mass to the blood when the circulation is renewed. Many of the extreme collapse cases will come round by themselves as well as or better without stimulants than with them, and, therefore, we must not suppose that we are accelerating death by leaving the patient to cold water in such cases. For the sake of trying whether the pulse can be roused we may give a little stimulus now and then, taking care that the total quantities given shall be too small to do harm. I have made these observations because I think that there is a tendency to consider stimulants as inadmissible in the treatment of Cholera. Much mischief may have been done by them sometimes, but with due attention to the principles that I have mentioned for both opium and stimulants I think that good, not evil, may result. The discriminating use is very different from the abuse of remedies.

The profuse perspiration should be rubbed off the surface with a dry soft towel. In the Indian hospitals dry ginger powder is often used with friction for absorbing the moisture. If used, care should be taken that the powder is impalpable, and free from grit or dirt; otherwise troublesome excoriations will ensue.

Cramps are, as in the earlier periods, soothed by friction with turpentine and chloroform. Even in great debility, moderate chloroform inhalations may be used without increasing the weakness of the pulse. Food will not be borne, except, perhaps, in the prolonged cases of semi-collapse, but when signs of reaction appear, a little beef-tea or chicken broth may be given. Starchy matters are apt to generate gas, but sometimes a little sago or gruel may be given with advantage, as they lubricate the coats of the stomach. When the pulse is very weak, in cases in which stimulants are given, a little port wine may be added to the sago or arrowroot.

Warm baths, or vapor baths, or medicated wet sheets, are of no use, and the patient is so restless, and throws himself about so much, that it is difficult to apply them.

Reaction Stages.—If the patient is regaining strength, and his secretions return, there is little to be done. He is best left to nature, a little liquid food, and cold water. To compensate for the loss of saline matter from the blood, a little common salt should be added to his food, and he may occasionally take a draught of water to a pint of which a drachm of carbonate of soda is added. This last also is grateful to the patient when he has heartburn or heat of stomach. It is not necessary or desirable to give a dose of aperient medicine within the first few hours of reaction in order to promote the secretions. The officious use of purgatives and mercurials, too soon, often brings on a relapse. If the bowels are confined, and there is tension and uneasiness, a warm water enema—say a quart of water with a little asafœtida, or sal volatile, or æther—will be the safest remedy.

Vomiting is often continued in the reaction stage, and may have been caused by the irritation left by frequent previous retching, or by excess of stimulating remedies. In these cases a single dose of opium or morphia—say a grain of opium—will often act admirably. It may be repeated every four or six hours if the vomiting continues. The food requires careful management. Sometimes a tablespoonful of milk with one of lime-water may be given every half-hour or hour for the first day or two, or a small quantity of ice-water only. It is desirable to give the stomach as much rest as possible. In some cases the irritation is due to subacute gastritis, and requires a few leeches to the epigastrium—a blister is useful in all forms of the vomiting in the earlier stages of reaction, and sometimes one-half or two-thirds of a grain of morphia may be applied to the raw surface with excellent effect. Food should not be pressed upon the patient. For days he may not be able to take more than teaspoonfuls or dessertspoonfuls of milk and lime-water, arrow-root, barley-water, &c. In some instances it will be necessary to trust entirely to nutritive enemata. Effervescing draughts with excess of soda are sometimes useful, and hydrocyanic acid may be tried.

Diarrhœa, if slight, should not be inter-

fered with at first, especially if the evacuations are at all offensive or bilious. Where decided enough to be weakening, gallic acid, or tannin, or mild opiates, or chalk mixture with carbonate of soda, should be given. If there be tenesmus, a warm water enema with a drachm of carbonate of soda should be used, and followed after operation by one of laudanum and a little water. Turpentine stupes should be applied to the abdomen in all the states of intestinal irritation.

Hiccup is frequently accompanied with much eructation of gas, and is difficult to stop; it is not dangerous. It may be treated by sinapisms to the spine, in the cervical and dorsal regions, and by ten minims of chloroform in mucilage every half-hour, or by chloroform inhalation, by morphia, or small doses of belladonna, and, if there be acidity, by alkalies. The common anti-spasmodics seem to have little effect.

Want of sleep, without pyrexia or heat of head, may be relieved by a dose of calomel and opium, and perfect quiet, hot pediluvia, and, if very obstinate, chloroform inhalation. Sometimes a mild dose of alterative aperient medicine is beneficial.

Imperfect reaction is very troublesome. Unless the discharges are arrested, the patient will sink in three or four days. A small dose of calomel and opium may be given. The following astringent mixture sometimes arrests the vomiting and purging: Gallic acid, 10 grains; tincture of opium, 10 minims; dilute sulphuric acid, 10 minims; water, 1 ounce. This should be mixed with a little cold or iced water, and given every four hours. Turpentine fomentations to the abdomen, or sinapisms; non-irritating food should be prescribed. Champagne is sometimes useful. It is better not to give too much liquid, but thirst may be moderately gratified.

Uræmic Stage.—Purging should not be checked in this. It is exceedingly difficult to restore the renal secretion. The kidneys are gorged with blood abounding in urea, their natural stimulus, and yet they will not act. When it is seen that suppuration exists, the patient should be encouraged to drink as much water as he can. It is the best diuretic; and sinapisms, dry cupping, or cupping, should be applied to the loins. He should take a drachm of liquor ammoniæ acetatis, half a drachm of spiritus ætheris nitrici, and 5 to 10 minims of tincture of digitalis in water, every three hours. The special symptoms of uræmia should be treated as uræmia generally, and need not be detailed here. When coma comes on or is approaching, it would be advisable, if the pulse is firm and good, to try the effect of a moderate venesection. This might relieve the renal congestion. There is no more unfavorable state to treat than that of approaching head symptoms, with vomiting of grass-green matter, and diarrhœa. The discharges are exhausting, and if stopped, coma is hurried on all the faster, because the urea is to a certain extent relieved by the evacuations.

Fevers.—The mild cases require little interference. They need only a small quantity of nourishing food, not forced upon the patient, perhaps a little alterative and mild aperient medicine, effervescing drinks with excess of alkali, and quiet. The more severe fevers need all the attention and prolonged care of severe adynamic fevers, and, according to the state, stimulants, wine, tonics, and nourishment; but there is nothing special in the treatment. The same may be said of boils, gangrenes, inflamed parotids, sloughing corneæ, purpura, &c.

Much pallor and debility is often left after protracted illness in Cholera. These demand quinine, iron, strychnine, and other tonics. Change of air is often the most efficacious remedy, and such a total change as a voyage from India to Europe, and residence there, is often demanded.

Throughout the collapse and subsequent changes of debility, the patient should be kept in the recumbent posture, and during the uræmia and fevers the most scrupulous cleanliness of the bed should be preserved, and no wet or soiled sheets allowed to remain unchanged. The sacrum and hips rapidly excoriate, and very troublesome bed-sores are quickly formed. The surgeon should examine these points himself frequently. The hypogastric region should be examined twice daily in order that retention should not be mistaken for suppression of urine, a mistake which may now and then happen in the early reaction stages, and sometimes in the typhoid and comatose periods.

From the first to the last there is no disease which requires more careful attention during treatment than Cholera, or more watchfulness on the part of the surgeon. In the early stages it will be impossible to leave patients with mere routine directions for treatment if we wish to be successful. The when to do, and the when not to do, in a disease with such rapid changes as Cholera, require much more discrimination and knowledge than is usually possessed by nurses or friends. The case requires throughout the constant guidance and vigilance of the surgeon, and he must specially assume this action during the critical periods of the disease. Good nursing is always of the utmost value, but patients in Cholera need that this should be frequently superintended. In military and civil hospitals, especially in India, in which the nursing must be confined to native servants or to the com-

rades of the sick man, the surgeon is obliged to be doubly watchful.

All patients with Cholera should be treated in well-ventilated rooms or wards. On account of the sad spectacle presented to the patients with other diseases in hospitals, when a large number of patients are brought in for treatment, and to avoid all possible chance of diffusion of the disease, small though that chance may be, it is desirable that all Cholera cases should be treated in separate wards, and with ample space and ventilation about them. Should there not be such wards or rooms, tents should be used. I prefer rooms to tents, if they are well ventilated, and have non-absorbing floors and walls that can be washed with water and chemical solutions. Tents, unless they are thoroughly cleansed and purified, are as likely to spread disease as hospitals are. They are colder than rooms, and the attendants and patients themselves are likely to keep them shut up, and from their size the air within them is sooner rendered impure than in an airy ward. Those surgeons who remember the severity of hospital gangrene at Ferozepore, in the Sutlej campaign of 1845-46, know that tents present no immunity from hospital evils. The only real advantage of a tent is the power of changing its floor by shifting the ground, but against this are to be placed the coldness at night, and the discomfort and consequent diminished efficiency of attendants. Of course there are times when the tent must be accepted, but I should in preference select to treat my Cholera patients in airy wards, in which I could regulate the temperature, in which I could wash and scrub the floors and walls, which permit of economical use of nurses and attendants, and in which they have that comfort and warmth which promotes their efficiency. In speaking of tents, of course I refer to their use in warm climates; in cold climates, except in summer, they could hardly be used. In India, in the warm seasons, too, we are subject to heavy storms, which render tents most uncomfortable, and I have seen several tents, with wounded men, blown down in one night. Those only who have spent weeks under canvas in the Northwest Provinces in India know how exhausting is a sojourn in them in the daytime, and how much cooler a well-covered house is. In the rains, too, they are most uncomfortable; either chilly in the heavy showers, or close and stifling if shut up. I have made these observations because I believe that there is a disposition to adopt tents on all occasions for treatment of Cholera. I think that this is an error. There are times when tents must be resorted to, and when to use them at all times and all seasons is the least of two evils; but I should use them in times of necessity only.

The duty of the medical practitioner is not limited to the treatment of cases presented to him. It is most important that all cases of diarrhœa, choleraic diarrhœa, and incipient Cholera should be treated early, and the physician should take the initiative in impressing this upon all within his reach during Cholera epidemics. Patients should be provided with instructions on the symptoms, and, when living in distant places, in addition, with remedies that they can use until efficient assistance can be had. Surgeons of regiments should organize daily inspections of the men in barracks, and all cases of relaxed bowels should be treated on the spot, and the patients not sent to hospital unless for real Cholera. The fear of being sent to hospital constantly prevents men from reporting these ailments, which they would readily do if it were not for the dread of being made in-patients. These cases can be readily treated out of hospital, and with the advantage of lessening the crowding of the wards.

Treatment of Choleraic Diarrhœa and Diarrhœa.—The discharges should be restrained. A grain of calomel with a grain of opium morning and evening, and with a dose or two of chalk mixture and soda, or of tannin or gallic acid, with sulphuric acid in the middle of the day, will perhaps suffice, if continued for two or three days. With these should be combined turpentine fomentations to the abdomen, light digestible food, and if there be exhaustion wine with arrowroot or sago, or warm brandy and water occasionally. The patient should give up work, and keep to his bed in cold climates, or to his couch or sofa in warm ones. If there be rice-water stools the patient should be treated as for the first stage of Cholera. If the diarrhœa continues for several days in spite of treatment, the patient should, if possible, be moved altogether out of the locality in which Cholera prevails. Choleraic diarrhœa, like Cholera, is much more intractable and dangerous in the commencement of epidemics; following indeed the rule of Cholera itself. In these periods many cases run into Cholera in spite of early and assiduous treatment. The disturbances which sometimes precede both choleraic diarrhœa and cholera, may be treated with mixtures containing sal volatile, æther, and aromatics, tonics, and regulation of diet. Many of these sensations will be due to the imagination, to fear, &c.

[Rest in bed, or, at all events, in the recumbent posture, is important in Choleraic diarrhœa, or "Cholerine." Dr. Jules Worms, of Paris, has especially advocated sulphuric acid, in dilute solution, for this affection. Some confirmation of its utility was obtained in Philadelphia in 1866.

Drs. Cox, Buxton, and Fuller, in England, made use of the same remedy many years ago. Dr. Worms's method is to give to patients, having prodromic diarrhœa, a "lemonade" composed of about half a drachm of concentrated sulphuric acid to a pint or more of sweetened decoction of salep. Arrowroot will answer equally well as a vehicle.—H.]

Cholera in children must be treated upon the same principles as in the adult. Laudanum must be given in preference to solid opium. It is necessary to be very cautious about the repetition of doses of the opiates. Indeed, unless the first dose be vomited directly, it had better not be repeated for the young child under three-quarters of an hour, or an hour, and not at all if there be any signs of drowsiness. The best stimulants are sal volatile and tincture of camphor given in a little iced water or thin arrowroot; a few drops of port wine or brandy in arrowroot may be given occasionally. In reaction it will be better not to give it the breast for twenty-four hours, but to feed it instead upon milk diluted with water and a little lime-water. The mother's milk if given should be pressed from the breast and diluted with a little water; or, if available, a little donkey's milk may be given. If the child has been in the habit of taking broths or farinaceous foods they can be given in small quantities, and not strong.

It would be vain to attempt to give an account of all the plans of treatment and remedies proposed for Cholera. They are of opposite character and very numerous; two or three deserve notice.

Venesection.—This was formerly a good deal used in India, but is not often resorted to now. In the spasmodic febrile forms Mr. Twining bled, and he thought with advantage, in the early stages. In collapse little blood flows, and on the whole probably when it does the abstraction does more harm than good.[1]

Saline Injections into the Veins.—This, when first introduced, seemed of brilliant promise. From the extreme of collapse the pulseless man rapidly recovered strength and sat up in bed, and talked, and looked himself, but the amendment did not often last; purging returned, and with it the collapse. No means have been found of keeping the injected fluid within the vessels; alcohol, quinine, laudanum, albumen, and other things, have been tried, but have failed, and from this the injection plan has just missed being a great and glorious discovery. The following formulæ for the solution have been recommended:—By Dr. Latta, carbonate of soda, ʒj; muriate of soda, ʒiij; water, 6 pints. By Schmidt, chloride of sodium, 60 parts; chloride of potassium, 6 parts; phosphate of soda, 3 parts; carbonate of soda, 20 parts—all by weight: 140 grains of this mixture to be dissolved in 40 oz. of distilled water and filtered. The temperature of the injection should be about 108° to 110°, and the specific gravity 1005 or 1004. An endeavor is made to make a solution of similar composition to the fluid lost by the blood. It should be injected slowly, not above 40 or 60 ounces at a time, and not faster than at the rate of 2 oz. per minute. If purging and collapse return the injection can be repeated. In some of the cases it was repeated several times. Dr. Owen Rees thought that a fluid of higher specific gravity than that usually injected should be tried, and recommends the following:—Chloride of sodium, ℥ij; phosphate of soda, ℥j; carbonate of soda, ℥jss; sulphate of soda, ℥ss. To be dissolved in distilled water at 98° F., making the fluid of specific gravity of 1030. This solution would seem to be too dense.

Most of the cases on which the experiments were tried were in a state of extreme collapse, but the recoveries, as shown by Dr. Wright, Dr. Mackintosh, Mr. Twining, and others, do not show that more escaped than might have been expected if they had been left to themselves.

Inhalation of Oxygen.—The patient cannot use any apparatus so as to inhale during collapse; therefore any gas used must be brought into contact with the air cells by some form of artificial respiration. It is doubtful, however, whether oxygen would act upon the blood in its altered state. Some experiments have been made of impregnating the atmosphere with oxygen gas by decomposing chlorate of potash at the bedside, but no real benefit seems to have resulted.

Saline Plan.—Dr. Stevens recommended draughts of fluids containing chloride of sodium, carbonate of soda, and chloride of potassium in order to supply the loss in the blood; but it has been of no avail, the mixture would not be absorbed, or if absorbed, not retained in the blood.

Dr. Ayre's Method.—This consisted of administering 1 or 2 grains of calomel and 1 or 2 drops of laudanum every five or ten minutes for several hours, the object being to bring on a secretion of bile. This plan has not proved successful in the main, though, at the time of its introduction in England, some practitioners thought it beneficial.

Prophylaxis.—Whatever may be our views as to the contagion of Cholera, it is well to act upon the principle that it may under certain circumstances be spread in a locality, and thus be upon the safe side.

[1 Bleeding from the arm was practised by me in one case of collapse in Philadelphia in 1849. The patient, a boy 12 years of age, reacted and recovered.—H.]

We ought not to neglect the evidence brought forward by Dr. W. Budd, and at all events act as if the fecal discharges may become injurious to the healthy. They should never be thrown into the common privy, but be treated with chloride of zinc, and buried in trenches out of harm's way, at some distance from habitations, and where no drainage from them may find entrance into wells or cisterns. The bed-pans and other vessels used should also be washed with chloride of zinc, and thoroughly cleansed. The patient's bed should be covered with waterproof sheeting to prevent the soaking of the mattresses, and the sheeting should be well cleaned with disinfecting materials. Better still, in hospitals the beds should be made up of soft straw, and, when once used by a patient, the straw should be taken out and burned. The covering, the bed clothes, and all linen should be boiled in alkaline lye, and well washed. I believe that there is not the slightest necessity for burning the linen used. I believe that Cholera patients may be safely nursed by their relatives or friends, and that they run no unnatural risk in attending to them. Nursing mothers should not resume suckling their children, even if their milk returns, until several days after convalescence; the milk having been previously drawn off by some apparatus for the intervening days.

Removal out of an Infected Locality.—When practicable it would be well for people, whose occupations permit them, to move out of diseased places, but that can never be done on any considerable scale, except in the cases of troops. Under present rules issued by the Government of India, all troops are to move out of stations if epidemic Cholera prevails among them. This is therefore no longer a question for the consideration of the medical officer; he has only to do with the time of moving. Hitherto the experience of removal has been favorable as far as the diminution of cases and cessation of the disease are concerned; the deaths to cases have not diminished. Without doubt there are certain risks to be encountered in moving out, but they are smaller than the dangers left behind. The risks will vary with the weather and the seasons; but these moves have sometimes been made even in hot weather and rains, without increase of other sickness. These are the cases in which the discomforts and risks of tents must be accepted as preferable to remaining in the midst of the poison. It would be better to have lived under a tree or an umbrella than in some of the hospitals during the late epidemic at Meean Meer. It is not necessary to move far; a few miles are sufficient, and the march should be at right angles to the wind. Of course, in removal into camp, there should be realized absence of fatigue, good food, pure water, and thorough camp conservancy, or else there is no reason why troops should not suffer just as much from the disease as the Madras regiments seem to do, and as the regiments marching down from the hills to the siege of Delhi did in 1857.

Good food and pure water should be especially attended to in Cholera epidemics. Even without these being more than predisposing causes, there is sufficient necessity for attending to them. All indigestible food and all tainted articles should be specially avoided. It is well to bear in mind the teachings of Dr. Snow and Dr. W. Budd; and whether we believe that the exciting cause can be conveyed into the system by drinking water, or not, take care that all water consumed be entirely free from any fecal pollution. On the same grounds that all food or water likely to produce irritation of the intestines, and thus determine choleraic action, are to be avoided, we should be extremely cautious about the administration of purgative medicines in the times of Cholera epidemics. Of course, occasionally they will be required, but they should not be given without decided necessity, and then never of the saline or hydrogogue character, and never at bedtime.

It has not been intended to give in this paper a full account of the hygienic measures required in the Cholera epidemics, and for lessening their severity. Fortunately these are fully treated of in Dr. Parkes's recent work on Practical Hygiene, Dr. N. Chevers's Review of the Means of Preserving the Health of European Troops in India, and the Report of the Commission appointed to Inquire into the Cholera Epidemic of 1861 in Northern India, which last may, I believe, be obtained in India. These from their nature are mostly applicable to troops, but their teachings may be made equally useful for all classes and for all communities.

CONSTITUTIONAL SYPHILIS.

By Jonathan Hutchinson, F.R.C.S.

The specific fever known as Syphilis differs chiefly from its congeners in the much more prolonged duration of its several stages. Like smallpox, measles, scarlet fever, and the others in this group, it is communicable from the diseased to the healthy, and can be produced by no other means; like them it has its several stages of incubation, efflorescence, relapses, decline, and sequelæ; as in them, so in Syphilis, the most prominent symptom is an exanthem, or cutaneous rash. The various stages of Syphilis tend to pass away of themselves in the course of time, just as certainly as do those of smallpox; and a well-developed attack affords for a time immunity from a second. As is the case in the other zymotic diseases, the poison of Syphilis is one which possesses the power of breeding in the patient's body, and the smallest possible quantity of virus suffices in due time to inoculate all the solids and fluids of the system. The time required, however, is much longer, and the stages are much more protracted. Instead of counting the duration of the stages by days, we have to count by weeks, or by months. From this circumstance there follow in the most natural manner certain apparent differences between Syphilis and the other fevers. Thus, because the evolution of the exanthem is slow and gradual, the pyrexial disturbance attending it rarely rises to any great height; because each stage is so much longer, correspondingly wider margins of occasional variation in length must be allowed. It further follows that because the disease extends over years, its subject is often not incapacitated by it for social life; many while still infected become parents, and transmit their own taints to their offspring, a circumstance which can but very rarely happen in the more short-lived and acute fevers.[1] These apparent differences are by no means real one. Nor is it probably by any means correct to allege that Syphilis is the only fever which has a tertiary stage. What are called the tertiary symptoms of Syphilis find their analogies in many cases of smallpox or scarlet fever, in what are known as the sequelæ of those diseases. It is true that these occur only in a small proportion of cases, but the same holds good of the tertiary syphilitic phenomena. If we observed more carefully, it is probable that the sequelæ of the exanthems might be recognized much more often than they now are, and that many of the diseases classed as "strumous" inflammations of the eye, the ear, or the skin, with also diseases of bones and joints, are in reality the tertiary consequences of some specific fever. So, too, if it be alleged that the stages of Syphilis may be shortened and otherwise modified by treatment, while those of the other exanthems cannot, we may reply that those of the latter are too short and transitory to give time for a fair trial of remedies, and also that the power of remedies over syphilitic phenomena have probably been much exaggerated. Syphilis does fortunately differ from most of the other specific fevers, in that its virus is incapable of diffusion in the atmosphere, and that consequently it is contagious only, and not infectious also, but it is by no means solitary in this feature.

Having thus insisted upon the rank which true Syphilis ought to occupy in our nosological classifications—a point of the utmost importance to our correct appreciation of its nature—we may next ask the question, whether under this name we have to deal with one disease or with several. Every surgeon is aware that there are many venereal sores which are not really syphilitic, and very different opinions have been entertained as to the relation which these hold to the genuine disease. Some have assumed, with Carmichael, that there are several distinct poisons, and others, especially a modern and now very extensive school, hold that there are certainly two. Without entering at length into the controversy, I will simply remark that the evidence in favor either of plurality or duality,[1] is to my

[1] Hereditary transmission happens in all specific fevers if it chance that offspring are produced while the parent is suffering.

[1] The doctrine of so-called "duality of Syphilis" seems to me to rest on the most unsubstantial foundation. Surely it is absurd to speak of the "duality" of things which have scarcely any features in common. The production of constitutional phenomena is the essential feature of Syphilis, and there are not two forms of Syphilis as thus denoted. We may freely admit that, in impure sexual

mind quite inconclusive. Let us accept clearly the doctrine so essential to the explanation of numerous pathological phenomena, that all living pus is contagious, and is capable of producing an inflammation similar to that in which it originated, and we shall not have much difficulty in explaining the different forms of venereal sores. The majority of the latter are probably *abortive inoculations*. In the performance of vaccination, the utmost care is taken to secure a pure virus, yet every now and then an abortive sore is produced. If it were the practice to inoculate again from these abortive sores when suppurating, we might soon produce an analogous state of things to what we now have in respect to the soft and hard chancre. The inoculation of Syphilis is of course a matter of mere chance, and the virus with which it is effected can be but very seldom in a state of purity. How rarely in the female do we meet with a healthy type of the indurated sore? Then, too, we must remember that this stage of the primary sore is transitory, and those who have once had it but seldom have it again. A large proportion of the women by whom contagion is communicated have had Syphilis long before, and are now no longer capable of originating the true virus. The sores which they possess are analogous to those caused by vaccination in protected persons. Attention to these considerations would, I feel confident, enable us to put aside the unnecessary hypothesis of duality.

In a medical essay on Syphilis any detailed consideration of the primary symptoms would be out of place, since these by common consent come under the care of the surgeon. We will content ourselves by saying that a successful syphilitic inoculation is denoted by the formation of a base of induration beneath the abrasion first noticed, that this induration usually occurs within a week of the contagion, but may be delayed for several weeks; that it is usually attended by very little either of ulceration or suppuration, and that it causes an enlargement of the proximal lymphatic glands. The bubo shows the same tendencies as does the chancre. There is hardness with but little swelling, and rarely much tendency to the formation of pus. These characters will be the same, or nearly the same, in both sexes, at all ages, and on all parts of the body.[1]

Stages of Syphilis.—A successful inoculation having been effected, a period of incubation now ensues, which may last from one to three months, and usually averages about six weeks. During the later part of this period the patient is often sensible of slight malaise and discomfort. At length an exanthem makes its appearance, affecting both the skin and the mucous membranes. The skin shows a scattered eruption, which may vary very considerably in some of its characters, but usually conforms to rule, in being of a peculiar coppery hue, and in preferring the flexures of the limbs to their dorsal aspects. This rash may be merely congestive, resembling that of measles; it may be scaly, papular eczematous, pustular, or bullous. In some few cases it is attended by ulceration, but as a rule it involves only the superficial layers of the skin, differing in this respect from the tertiary manifestations. There is not the slightest reason for believing that these differences in the character of the exanthem imply difference in the nature of the virus. Chancres exactly corresponding in their characters may be followed by most diverse kinds of eruption. Simultaneously with the eruption on the skin, we usually observe evidences of similar implication of the mucous surfaces. In the tonsils symmetrical ulcers form; these ulcers are of kidney shape, have a tawny gray base and abrupt edges. They are attended by but little pain, and do not spread either laterally or in depth. After lasting for a few weeks they usually heal. These tonsilar ulcers are rarely absent in the exanthematous stage. Very often, but not nearly so constantly, we observe also certain superficial patches of inflamed mucous membrane in the mouth, on the pharynx, palate, tongue, or cheeks. On the tongue, and at the corners of the mouth, and sometimes in other positions also, these patches become very considerably raised, and assume the condition known as condylomata. If condylomata be observed in the pharynx, they will almost always be observed at the anus also, and *vice versâ*. They are to be regarded as patches of cutaneous eruption modified by their position, and by the moisture of the parts. Whether or not in these cases any eruption extends through the alimentary canal is a point upon which we have no evidence.

The exanthem usually takes from a fortnight to a month before it is fully out, and about two months are usually occupied in its gradual decline. In some cases it is very transitory, and in some it is

intercourse, pus of very various qualities and endowments may be inoculated, and thus very different kinds of local ulcers may result, but there is only one which can produce Syphilis.

[1] Some writers hold that certain parts never exhibit the phenomenon of induration when inoculated. My own experience is, however, very decidedly in favor of an opposite view. I have seen chancres with well characterized induration on the most various positions, *e. g.*, the glans penis, the nipple, the lips, the eyelids, the hands, &c. &c.

greatly prolonged. When it is at its height, or just when it begins to decline, it is not unfrequent for inflammation of the irides to occur. The iritis, when it happens, is usually symmetrical; it is attended by the free effusion of lymph, often in elevated nodules of a salmon or rust tint, the characteristic zone of ciliary congestion is usually well marked, and there is often a dotted deposit in the posterior lamina of the cornea. In other cases, instead of iritis, or coincident with it, inflammation of the retina occurs. The retinitis is, I think, usually a little later than the iritis, and we rarely see it until the eruption on the skin is well disappearing. This form of retinitis is not uncommon, and is a very insidious and most important malady. The patient notices nothing, excepting that his sight is very dim; he has no pain, no congestion of the front of the eye, no intolerance of light. The ophthalmoscope shows us the retina hazy, and as if stained with port wine; the optic disk swollen and its margins indistinct; whilst not unfrequently numerous small extravasations of blood are seen.

Whilst the local phenomena just mentioned are occurring, there are usually present others of a less definite character. The patient loses flesh, he is restless and slightly feverish, the appetite is deficient, the bones and joints ache, and the hair becomes dry and thin. Now and then swellings occur on certain bones, more especially on those of the skull; but in this stage periostitis is always slight and transitory, and never leads to suppuration. Such are the various symptoms which make up what are called the secondary symptoms of Syphilis. In many cases only a few of them occur, the rash on the skin and the ulcers in the tonsils being those which are most constant. In many individuals all the secondary symptoms have disappeared within six months of the original contagion; but in a larger number, a year elapses before such is the case. It is rare for any symptoms belonging to the secondary group to linger after eighteen months have passed, although their effects are often seen much later. That these symptoms disappear in a most satisfactory manner, quite irrespective of any treatment which may be adopted, is a fact confirmed by every day's experience.

After the group of secondary symptoms has passed away, there usually follows a period of apparent health, during which the patient believes himself wholly cured.[1] After this interval, which may vary in length from a few months to many years, symptoms of a different kind ensue. Inflammatory indurations of a chronic character, and tending slowly to suppuration and ulceration, occur in the deep-seated tissues, or in the deeper parts of the superficial ones. In this way the skin, the subcutaneous tissue, the periosteum, the muscles, the internal viscera, the organs of special sense, and even the cerebro-spinal system itself may be attacked. A remarkable difference is to be observed between secondary and tertiary symptoms, as regards the symmetry of the inflammations produced. In the secondary stage, a tendency to symmetry, often very exact, is observed, proving that the producing cause is free in the blood, and is supplied impartially to both halves of the body. In the tertiary stage, the lesions are of tensingle, or, if multiple, they display but little accuracy as to symmetrical arrangement. This fact implies that they result from impaired organization of the solids, rather than from any poison still

[1] The interval which elapses between the well characterized secondary symptoms and the well characterized tertiary ones, is one of different degrees of immunity in different cases. In many, I think the majority, the poison is wholly latent, and the patient experiences nothing whatever to remind him of his taint. In many others, however, recurrences of symptoms, which it is difficult to assign to either group, continue to show themselves. Superficial sores on the tongue or the mucous membrane of the mouth, isolated patches of scaly or desquamating eruption on the skin, especially psoriasis palmaris, are the more frequent of these. Sometimes they are symmetrical; at others not so. Such symptoms may continue to recur for many years (even to twenty) after the contagion. They probably depend rather on permanent tissue contamination than on still existing blood disease. At any rate I may safely assert that we never witness any true recurrence of the secondary epoch. The eruption is rarely very copious, and rarely of such a character as to deceive an experienced eye: nor is it attended by the ulcers in the tonsils and iritis, which are so common in the secondary stage. In rare instances, at an interval of perhaps a year or eighteen months after infection, a relapse of a general symmetrical rash may occur. This rash may assume the form of rupia and be attended by severe constitutional symptoms.

An idea is prevalent that syphilitic iritis may occur at almost any period in the course of the disease. I speak after close attention to this subject, when I assert that all our well-marked examples of this disease present themselves amongst the secondary phenomena. From three to six months after the chancre is the usual date. When once iritis has occurred, and adhesions have been left, relapses are liable to happen, and it is this fact which has led to the error which I am endeavoring to confute. In these relapsing cases, however, the symptoms are very different from those of the first attack. The effusion of lymph is much less free, no nodules are seen, and rarely are both eyes simultaneously affected.

existing in the circulating fluids. The occurrence of the tertiary symptoms is to be explained by the fact, that during the exanthematic stage, when the whole blood was loaded with the virus, the various solids have received from that poisoned blood the elements necessary for their growth, and have been built up, so to speak, with syphilized plasma. Hence an impairment of organization in such tissues, and a liability under slight exciting causes, or even in the ordinary course of nutritional change, to the occurrence of specific forms of inflammation. It is easy to see, that in Syphilis, with its very prolonged period of blood-poisoning, the risk of permanent tissue modification must be much greater than in the other fevers, in which it is so short. During the exanthematic stage of smallpox, probably but little in the way of nutrition is effected; the changes are chiefly those of waste and disintegration. From this, it follows that the more prolonged the secondary stage in Syphilis, the greater the risk of tertiary symptoms, and that severe febrile disturbance during this stage, by interfering with nutritional development, tends also in the same direction. Do we get from these considerations any glimpse into the principles of treatment? My impression is decided that tertiary symptoms, as a rule, follow earlier in the young than in the old, and if so, the fact may be explained by reference to the greater rapidity of elemental change in the former.

The following is a statement, in tabular form, of the various symptoms of the different stages of Syphilis:—

1st Stage.—*Inoculation* and *Incubation.* Average duration, six weeks to two months.	An ulcer with indurated base and but very sparing secretion. Induration of the nearest lymphatic glands, with but little adjacent inflammation or tendency to suppurate. Slight febrile disturbance. Slight enlargement of lymphatic glands in all parts.
2d Stage.—*Exanthem*, or Secondary Symptoms. Average duration, two to six months; may extend over a year.	Symmetrical ulcers in the tonsils, not spreading either in width or depth. A symmetrical eruption on the skin. Condylomatous patches on the mucous surfaces, and on the skin adjacent to the mucous orifices, usually symmetrical. Iritis or retinitis; mostly symmetrical. Pains in bones and joints. Febrile disturbance. Loss of hair.
3d Stage.—Interval of Latency or of Relapses. May vary from a few months to many years.	In some cases the patient is wholly free from symptoms, but in a certain number reminders occur from time to time in the form of scattered scaly patches, psoriasis palmaris, sores on the tongue, lips, &c.
4th Stage.—Tertiary Symptoms or Sequelæ. Of uncertain duration, and characterized by a remarkable tendency to relapse.	Unsymmetrical ulcerations in the mouth and throat, tending to spread widely and deeply. Unsymmetrical ulcerations on the skin. Nodes of periosteum, cellular tissue, muscle, tendon, fascia, or nerve; not usually symmetrical, chronic in progress, tending to ulcerate or even to slough. Diseases of viscera.

Having thus sketched the normal course of syphilitic fever in its several stages and its sequelæ, we must next consider the conditions under which its orderly evolution may be interfered with. These conditions are precisely the same as those which disturb the course of any other exanthem. We have, 1st, idiosyncrasy; 2d, the coexistence of some diathesis or of some other specific disease; 3d, immunity, partial or complete, obtained by a previous attack of the same disease; 4th, inherited immunity, partial or complete; 5th, imperfect inoculation; 6th, the influence of treatment.

1st. To take first *idiosyncrasy*, I may simply remark that it is a matter of general experience that certain constitutions resist the specific animal poisons in a most remarkable manner. We are unable to offer any explanation of the fact; on the other hand, we meet with those who succumb easily and suffer severely. The influence of these inexplicable peculiarities in individuals is frequently observed in reference to Syphilis.

2d. The existence of some *special diathesis* or of some *other specific fever* at the time of syphilitic inoculation may modify the course of the latter. Possibly we overrate rather than otherwise the effects of these influences. Although it is not infrequent to find a delicate scrofulous subject suffering with unusual severity from syphilitic poisoning, yet the converse is almost equally common, in which either the delicate escape easily or the robust suffer very severely. There can be little doubt, however, that the tendency to suppuration and ulceration is much greater in those of fair skin and sanguine temperament than it is in others.

The influence of diathesis (scrofula) is also often felt in preventing recourse to specific treatment. There is probably no reason to believe that the existence either of a diathesis or of another specific fever will modify the duration of the several stages of Syphilis.

3d. *A previous attack of the same disease* is well known to exert a most important influence upon the course of a second in the same individual. It is generally understood that Syphilis, once had, is protective in the majority of cases against any second attack, and we have but little trustworthy clinical evidence as to the nature of second attacks when they do occur. There is reason to believe that second inoculations are common, and that they usually end in the production only of the soft chancre (abortive sore).

I have myself witnessed in one instance two attacks of Syphilis in the same patient, and in it the disease was slight on the second occasion. It appeared to have undergone quite as much modification as we usually observe in smallpox after successful and recent vaccination. A young surgeon consulted me for an indurated sore, in 1860. He was treated by mercury, but suffered very severely from all the usual train of secondary symptoms. It was two years before he had wholly got rid of the latter, and from that time to the present he has enjoyed excellent health. In April, 1865, he contracted another sore, which indurated, and was followed in a month by a copious roseolous rash. Excepting slight redness in the tonsils, there was no sore throat. He was scarcely ill, and both chancre and rash disappeared in a comparatively short time. I have seen other cases in which it seemed probable that the patient had had true Syphilis twice, but this is the only one in which both attacks have occurred under my own observation.

M. Diday holds that second attacks are not so rare as they have been thought, and records twenty-seven cases which have been under his own observation. Of these, however, in sixteen a chancre only occurred, indurated, it is true, but without secondary consequences. (Some of these may have been examples of the relapsing chancre, and not true instances of secondary contagion.) In nine others the secondary symptoms were mild, and in two only were they severe. In these last two the average interval between the first and second attack was nineteen years and a half. (*See* New Syd. Soc. Year-book for 1862, p. 233.)

It is clear from these facts that second attacks of true Syphilis are very infrequent, and that when they occur within a moderate period of distance from the first the character of the disease is much modified. In these respects Syphilis conforms closely to all that is ascertained regarding the other exanthemata. An interesting fact in connection with my own case of second attack of Syphilis (given above) is that the same patient had also had two well-marked but mild attacks of smallpox, the interval between the two having been four years.

4th. Of yet wider importance is the question as to *the influence of disease in the parent in affording protection, partial or complete, to the offspring.* If we grant, as we must, the two postulates—first, that Syphilis is transmissible to offspring, and second, that it is protective for a certain time against second contagion—then we are obliged to admit that just as the disease itself may be transmitted, so may the immunity which it affords. Here again we have as yet very little clinical evidence on which to build, but what we do possess certainly favors the view that those who have suffered severely in infancy from inherited disease are to some extent protected. In the history of Congenital Syphilis, however, nothing is more common than to meet with cases in which the eldest child of a family suffered severely in infancy, the second slightly, the third still more slightly, and the others not at all. I have at present several families under observation in which this has been the case, and in which all the children have lived, and the intervals between them are but short. The younger members of such families often appear to be in robust health. Now, if in such cases the oldest enjoys immunity, probably the second also does so in some degree, and so on through the whole, the degree of protection diminishing in ratio to the distance from the original taint. Do we not here touch upon a law of the utmost importance, not only in respect to Syphilis, but to its congeners also? Is it not probable that a very considerable portion of the community, being the descendants of those who have suffered, enjoy in a certain degree, infinitely slight in many but powerful in others, immunity from further attacks? The manner in which a slight degree of inherited immunity would become manifested would probably not be in entire escape from contagion, but in the production of a much milder form of the disease. This is what occurs in cases of smallpox after vaccination, or after a previous attack of the true disease, and indeed in second attacks of any of the specific fevers. It is surely impossible to believe that the constitution of a person who has passed through the stages of any of these diseases ever again returns into precisely the same condition in relation to the virus in question that it occupied before, and it is equally inconceivable but that some share of this peculiarity shall be transmitted to offspring. A child born

of parents neither of whom are liable to smallpox or to Syphilis, as the case may be, must be in a different position, as regards those diseases, from the child of parents both of whom are liable. In like manner a half result ought to be expected where one parent is exempt and the other liable. Now it is a matter of well-proven observation that any specific disease will be especially severe when imported into a community previously free from it. The ravages of smallpox in a virgin race are something far beyond what is ever known in a community long accustomed to the disease. There are also good reasons for believing that Syphilis has become during the last two centuries a milder disease than it was when it first invaded Europe. This amelioration we may most satisfactorily explain by recourse to the hypothesis above suggested.[1]

5th. The next mortifying influence which we have to examine is *imperfect contagion*. A large majority of the accidental inoculations by which Syphilis is conveyed are probably impure in a double sense. The essential virus is mixed with other fluids, is diluted, drowned, it may be, in common pus and other secretions. Then again the fluid merely comes in contact with a thin mucous surface; no precautions are taken to secure its gaining entrance into the tissue. Hence, as already explained, a majority of such contagions prove abortive. It is probable however that through all these difficulties the essential virus retains its characters unmodified, and that if it once produce its own first effects all the rest will follow. However small the quantity producing it, if once the sore have become indurated, the usual results may be expected. We have no reason for believing that there is any second variety of constitutional Syphilis, other than that which follows an indurated sore. The questions as to imperfect contagion therefore concern the surgeon rather than the physician. Under its influence a great variety of venereal primary sores are produced, but we have no real variations in the specific fever and its results.[1]

Lastly, we have to ask the all-important question, *whether the ordinary evolution of Syphilis can be altered in any way by measures of treatment.* It will probably be admitted that physicians have abandoned the idea that it is practicable by medication to regulate in any way the course of the other exanthemata. They are generally acknowledged to be diseases which always run their course. With the exception perhaps of cinchonism as a remedy for malarial fever, no single specific in the present day enjoys any repute as to cutting short the course of these diseases. But we must not too hastily assume *à priori* that the same will hold true as to Syphilis. It is possible that the stages of the other exanthemata are too short to permit of the beneficial influence of antidotes. Few questions as to therapeutics have been more hotly debated than the efficiency of certain drugs in reference to Syphilis. By some their specific power

[1] This subject will be found very ably treated in Mr. Lee's Lectures on Syphilis (Lecture xi. page 209). Mr. Lee quotes the important observations of Dr. Ferguson (1812) as to the mildness of Syphilis amongst the Portuguese being explained by the acquisition of hereditary immunity, and adds, "That which Dr. Ferguson observed in his day may be seen at present. A person who has had hereditary Syphilis in his youth, will either not contract the infecting form of Syphilis in after life, or will have it in a modified form."

Four years ago I published in the British Medical Journal some cases in which patients who had suffered from inherited Syphilis subsequently contracted venereal sores. These cases were, I believe, the first facts relating to the subject which had been recorded. Others had arrived at the same conclusions, but it was by *à priori* reasoning rather than by deduction from facts.

Subsequently I published a case in which a patient who was the subject of inherited taint, not only contracted a venereal sore, but experienced an outbreak of constitutional symptoms. This young man is still under my care, and suffers from inherited Syphilis and acquired Syphilis at the same time. I have recorded a number of facts bearing on this subject in the second volume of the London Hospital Reports.

[1] If we reflect on the mode in which syphilitic inoculation is usually effected, the wonder will be not that apparent varieties as to both primary and secondary symptoms occur, but that the disease preserves so close a connection with its type as it undoubtedly does. Here, if anywhere, are the conditions under which we might expect a new species to originate. In the first place the virus is constantly mixed with other secretions, and very frequently with those of inflammatory origin. In the first place the virus is constantly mixed with other secretions, and very frequently with those of inflammatory origin. In a great many instances the person from whom the contagion is received is one whose own body has been previously rendered proof against the disease. Most prostitutes probably suffer from Syphilis early in life, and during the greater part of the period during which they continue their vocation are incapable of being themselves again affected by true Syphilis, although still liable to contract and to transmit primary sores of a modified character. Then not only must we make allowance for differences in the kind of secretion with which the inoculation is effected, but also for differences in the recipient's state as regards it. Hence the differences in the type of cutaneous rash which follows; from a roseola to psoriasis and to rupia.

has been positively asserted, and by others as strenuously denied. As far as the purposes of our present argument are concerned, we may, I think, admit that there is no proof that the exanthematic stage of Syphilis can be prevented. If the sore have presented well-marked induration, a rash more or less copious is almost certain to follow in due time. A few exceptions occur, but they are as frequent when no treatment has been used as under opposite conditions. The statistics which have been collected on this point are for the most part valueless, because the kinds of primary sore have not been carefully distinguished. Any conclusions of trustworthy character must be based on the observation of indurated chancres only. For myself, I may state that I have treated some hundreds of these by the mercurial plan, and that in a considerable number of others I have carefully abstained from all medication, and that I am not in a position to record any single instance in which after mercurial treatment no exanthem followed.[1] That mercury can procure the healing of syphilitic sores and the absorption of syphilitic lymph, no one who has had opportunities of observation, and who dare credit the evidences of his senses, can doubt; but that it can prevent the occurrence of one of the stages of the disease, is a very different assertion. I shall discuss the question of treatment in its practical aspects as a further part of this essay; for the present, and in reference merely to the natural history of the disease, it may suffice to observe that there is no more proof that it can prevent the evolution of the exanthem of Syphilis, than that it can do the same in variola. Whether the tertiary symptoms can be prevented or made milder by treatment is again another question, since they, strictly speaking, do not constitute a true stage, but are rather the sequel, more or less accidental, of the secondary one. Any remedy which, although important to prevent, is yet able to modify and shorten the secondary stage, may very possibly influence the occurrence or otherwise of the tertiary inflammations; and whether mercury does so or not, must be determined solely on clinical evidence.

Modes of Communication.—Whilst the other exanthemata are for the most part communicable only by direct contagion or infection to the individual concerned, Syphilis, in consequence of its very protracted duration, may be conveyed in any one of three different modes. First, contagion direct to the individual; second, contagion indirect through the fœtus (possible only in women); and third, by hereditary transmission.

The period during which direct contagion is possible extends from the first appearance of the indurated chancre to the decline of the exanthem. The primary sore is more actively contagious than are any in the secondary stage, but there can be no doubt that under favorable conditions the germs of the disease may be conveyed by the latter. When Syphilis is communicated to a mother by contamination from the fluids of a fœtus with which she is pregnant, the course of the disease is materially different from what it is when received by other means. The absorbed materies seems to be scarcely capable of breeding in the blood of its recipient; it merely contaminates it, the degree of the contamination being in exact proportion to the amount received. The evidence of contamination is greatest during the pregnancy, and increases with each successive one. The symptoms produced are of the tertiary class only; for the most part the secondary stage is wholly omitted. A taint thus obtained rarely attains any high degree of severity.

When Syphilis is transmitted from parent to offspring, various important peculiarities are observed in its manifestations. In the first place the phenomena of the secondary and tertiary stages not very unfrequently occur together, or at any rate we have a superficial rash on the skin resembling a secondary one, coincident with nodes and with deposit in the viscera. These cases are, however, exceptional, and as a rule the stages occur as in the adult, the secondary rash disappearing after a few months, and there being a prolonged period of health before the tertiary symptoms show themselves. A few symptoms are peculiar to the inherited disease, and do not occur in adults who have acquired it. Amongst these I may mention, of the secondary stage, diffuse stomatitis without ulcers, diffuse inflammation of the mucous membrane, of the nares resulting in the well-known symptom of snuffles, and of the tertiary ones a form of phagedænic lupus and interstitial inflammation of the cornea. The latter, which is a common and very well marked condition in inherited Syphilis, has no parallel condition whatever in the acquired disease. Deafness and amaurosis from nerve

[1] By this I mean that I have never seen a case in which after a well indurated sore I kept the patient continuously under observation, and assured myself that he never had any constitutional symptoms. I have seen many in which the constitutional symptoms were so slight that they might easily have been overlooked, but these are sufficiently frequent without treatment to make us very cautious in assuming, when such a result follows mercurial treatment, that is a *propter hoc*. Many patients whom I have treated by mercury for the chancre have been lost sight of as soon as the latter was healed, and it is of course possible that in some of these no secondary stage occurred.

or cerebral disease are both of them far more common in the inherited form of the disease than they are in that which is acquired. The effect of the syphilitic poison upon the ovum is in many instances to destroy its vitality at an early period, and consequently to induce abortion. Unfortunately this is far from being its constant effect. In the great majority of such conceptions the tainted fœtus is carried to its full period. In exceptional instances it is then brought into the world with manifestations of its disease apparent in the form of skin disease; but in most this is not so, and the infants who when a few weeks old will suffer most severely, appear at first to be perfectly healthy. In these a period of from a fortnight to two months usually elapses, and then simultaneously a rash appears, and the nostrils become stopped by swelling. At this stage the mouth is usually hot, its mucous membrane red and tumid, and the gums swollen. The child wastes, and assumes a shrivelled senile aspect. Sometimes acute, well-characterized iritis occurs. Condylomata are frequently seen. The cutaneous exanthem may vary in character, much as we find it does in the adult. Many children die during this evolution of secondary symptoms. If they survive they usually in the course of a year get rid of all traces of disease, excepting perhaps an unusual pallor of skin, and certain scars which may have been left in the face by the eruption, and an expanded nasal bridge caused by the long-continued swelling of the parts within.

I have said above that the tertiary and secondary stages are sometimes strangely mixed in the early symptoms presented by syphilitic infants. Amongst those which we occasionally meet with under these circumstances are nodes of the long bones, nodes of cellular tissue, of tendon, or of muscle, and disease of the liver, kidneys, thymus gland, &c. Such children are certainly more liable than others to serous inflammations. Serous arachnitis to a slight extent is very common, and pleurisy is not an infrequent cause of death.

A condition of extreme anæmia usually results during the outbreak of early symptoms in a syphilitic infant, and from this death often results. In many cases, however, the child does not emaciate, but retains an appearance of good health which is remarkable, considering the nature of the disease. I have occasionally seen an infant who was well grown, stout, and strong in an unusual degree, who yet presented well-characterized indications of inherited taint.

In the child as in the adult the secondary symptoms pass away in due time, and a period of health or latency ensues, of variable duration, after which the tertiary phenomena show themselves. These are of precisely the same character as in the adult, with, however, the addition of several others which are not met with in connection with the acquired disease. There are few more remarkable facts in the history of this most interesting malady than that the disease known as Interstitial, or, according to Mr. Dixon, as Syphilitic Keratitis, should never occur as a consequence of acquired disease, but only in the inherited form. I must also here note a remarkable exception to what I have stated to be the characteristic of tertiary symptoms in the adult, that they are exceptionally symmetrical. This form of keratitis, although it often occurs many years after the secondary stage, is as a rule symmetrical. So also are the nerve affections, which result in the forms of deafness and amaurosis which we now and then encounter in these patients. As a rule, I believe all syphilitic symptoms in the inherited disease, without regard to stage, are symmetrical. In the cases in which tertiary and secondary symptoms in infants appear to occur together, the latter are rarely well characterized. Thus I do not know of any instance in which a copious scaly or papular rash with acute iritis were coincident with any symptom of a tertiary kind. It has been asserted that a parent transmits to his child the precise form of Syphilis from which he at the time suffers. But to any rule of this kind exceptions are far more frequent than are confirmatory instances. It is very common for a man who does not himself display a single symptom of any kind, and who appears to be in perfect health, to beget a syphilitic child, the symptoms displayed by the child being usually those of the secondary class. There is no doubt that the nearer to the occurrence of the primary symptoms in the parent is the birth of the offspring, the more certain is the latter to show symptoms of a severe character, and typically secondary in stage. Instances, however, are met with in which infants, born ten years after the original disease in the parent, still displayed first a secondary rash, with the characteristic snuffles, &c. In several instances I have known a whole family of children, born during a period of from five to ten years, display each one the characteristic and transitory rash soon after birth.[1]

The following appear to me to be well-established conclusions as to the transmission of inherited taint.

1st.—In all stages of constitutional Syphilis—whether during the secondary or tertiary symptoms, or even during a pro-

[1] For facts on this subject I may refer the reader to my paper in the London Hospital Reports, vol. ii. p. 184, *et seq.*

tracted period of latency—an individual may become the parent of a tainted child. The degree of severity of the inherited taint will be in proportion to the shortness of the period which has elapsed.

2d.—A child may inherit Syphilis in a severe form from but one parent—from its father alone, or from its mother alone.

3d.—When both parents are the subjects of Syphilis a child is more certain to suffer, and also more likely to suffer severely than when only one is so.

4th.—We have as yet no data on which to ground an opinion as to whether a child is more likely to suffer severely when its father is the source of contamination than when it derives the disease from its mother, or the reverse.

5th.—In a large proportion of the cases met with in practice, the taint is derived from the father only.

In connection with the hereditary transmission of Syphilis, an exceedingly important question arises as to whether any degree of taint is transmissible to the third generation. There is no doubt that persons of marriageable age often present heredito-syphilitic lesions in an active stage, such as keratitis and nodes. I have repeatedly seen patients of various ages, from twenty to eight-and-twenty, become the subjects of syphilitic keratitis for the first time. We might conjecture that such persons would be likely to transmit to their offspring some degree of taint, seeing that the taint is still in full activity in their own bodies. I am not aware that any facts have as yet been published on this question. Conjectures abound, and several surgeons have expressed their belief, that the influence of Syphilis once acquired is felt through several subsequent generations. About eight cases have come under my own observation in which persons, undoubtedly the subjects of inherited disease, have become parents. With one exception, I have never been able to discover any evidence of disease in the offspring. In several instances the offspring appeared to be in excellent health. I have always made a point of seeing the children for myself, never relying upon the parents' statement—a precaution which is essential, as I have here occasion to illustrate. The exceptional case just alluded to is strongly in favor of the belief that the third generation may suffer. As no parallel one is on record, I think its details worthy of brief mention. A respectable young woman came to me about six months ago on account of an inflamed eye. She had interstitial keratitis in a typical form, her teeth were notched, and her physiognomy characteristic. She told me that she was suckling her first child, an infant of two months. I inquired if it were healthy. She said it was a fine baby and ailed nothing whatever. I asked her to bring it with her at her next visit. She did so, and on having it stripped I found it covered with coppery blotches, with condylomata at the anus, and snuffles in the nose. Under subsequent treatment by mercury all these symptoms disappeared. There remains of course the source of fallacy that this child's parents, one or other of them, may have had acquired Syphilis. As to its father, I may state that he has been long under my treatment for sycosis, and that I have made the most detailed inquiry of him as to any venereal disease. I believe strongly that he has never had any. A fact, which is perhaps of more value than his own statement, is, that his sycosis has not been in the least benefited by iodide of potassium. Of course I have not ventured to insult him by inquiring as to his wife's antecedents, but there is no reason to entertain suspicion in that quarter, whilst the fact that she is the subject of inherited disease makes it probable that she would not be liable to the acquired disease. Having therefore carefully balanced the evidence, I incline to believe that we have in this instance an example of the transmission of Syphilis to the third generation.

TERTIARY SYMPTOMS OR SEQUELÆ.—I have endeavored to draw a strong line of distinction between secondary and tertiary symptoms. The secondary phenomena constitute a stage; they come on at a certain known period; they are in their nature transitory, and undergo spontaneous cure; they affect the two halves of the body at the same time, proving that they depend upon blood-poisoning; when once passed they rarely return. The tertiary symptoms are not so properly a stage, but must count rather as the sequelæ, more or less accidental, of the preceding stages. They are as a rule not symmetrical, making it seem improbable that they depend upon blood-taint; they have no tendency to spontaneous cure—quite the reverse. They relapse over and over again after remedial treatment. The period which intervenes before their outbreak is of very different length in different cases, and in many they never occur at all. From these facts we infer that they are due rather to the ill constitution of the affected structure than to any free virus still circulating in the blood. Let us briefly enumerate the principal tertiary symptoms which occur in acquired Syphilis.

We may conveniently take them in their relation to special organs or structures. *First, the skin and mucous membranes.* Tertiary affections of these tissues differ in a most marked manner from those which occur in the secondary stage. With the exception perhaps of palmar psoriasis, they all involve ulceration of

greater or less depth, and consequently leave cicatrices. Very frequently the patch assumes a crescentic form, spreading at its edges and healing in its centre the well known horse-shoe or serpiginous ulcer. If the disease commence in the middle line it may spread equally on the two lines, and may thus appear to be symmetrical; but it is decidedly unusual for symmetrically-placed patches to appear on the opposite limbs or on corresponding parts of the trunk. In many cases the skin is involved secondarily to the sub-cutaneous cellular tissue, the disease having begun as a gummous tumor or node of the cellular tissue. A form of lupus attended by rapidly-spreading phagedænic ulceration, occasionally occurs in tertiary syphilis, but there is good reason for believing that the common forms of lupus, whether exedens or non-exedens, have no connection whatever with syphilitic taint. The appendages of the skin, the nails and hair, are frequently affected during the secondary stage, and but very rarely at later periods.

The most frequent affection of the mucous membranes which we encounter in connection with tertiary syphilis, is a rapidly-spreading ulceration of the palate and pharynx. This again is totally different from the throat affections which occur in the earlier stages. Instead of being superficial and marked chiefly by swelling and inflammatory deposit, it is characterized by deep ulceration and loss of tissue. Instead of showing itself symmetrically on the two sides, it commences at one, two, or more points, and spreads quite irregularly. The cicatrices left by these deep ulcerations not infrequently narrow the pharynx and occasion difficulty in deglutition. In a few cases the ulceration may extend down the œsophagus, and in many the larynx is involved. Every now and then we see cases of tertiary syphilitic ulceration of the mucous membrane of the rectum, and again we must note that it is ulceration, and that it is not attended by the development of condylomata or mucous patches, as usually seen in secondary Syphilis. Stricture of the rectum is much to be feared when these ulcerations heal. Several authors have described cases resembling dysentery in all their symptoms, but occurring in syphilitic patients, and cured by anti-syphilitic remedies. Mr. Paget has recently recorded a case of this kind, and have myself seen some very well-marked ones. It is probable that in such cases ulceration of the mucous membrane at a considerable distance above the anus is present. I have seen several cases in which syphilitic ulceration extended higher than the finger could reach.

The cellular tissue is frequently involved in common with muscle, with periosteum, or with fascia. In not a few cases, however, we meet with what are called cellular nodes, in which the disease begins, and is, up to a certain period, confined to this tissue.

These may occur in any part of the body, but are much more usually met with in the lower extremities than in any other part. They are very common close to the knee, and especially so in the female sex. It is a very interesting fact in respect to these cellular nodes that they are comparatively very infrequent in men. Whether this is to be explained by the greater abundance of cellular tissue in women, or by the fact that many women obtain Syphilis in a manner wholly peculiar to them, that is, by fetal contagion, may be open to some question; probably both influences have their share in the result.

In the early stage of a cellular node we find a small lump of induration, often exceedingly tender. At first it is firm, but as it extends it becomes doughy and softer. When of considerable size there is frequently a very deceptive sense of fluctuation in it. The overlying skin becomes adherent and of a dusky red color. At length ulceration takes place, and a large core is exposed, consisting of sodden and infiltrated tissues, much resembling in appearance soaked wash-leather. Unless specific remedies are used, this core is very slow in separating, and the ulceration of the skin over it may spread widely.

Cellular nodes are not infrequently multiple, but more usually single. The patient frequently has scars of former ones on the opposite limb, but it is exceptional to find them simultaneously present on corresponding parts.

A period varying from four to ten or fifteen years has usually elapsed between the occurrence of primary contagion and the development of cellular nodes. In close connection with syphilitic inflammation of the cellular tissue, we must mention that of *the subcutaneous bursa*. It is not at all uncommon for a bursa to suffer in connection with the disease of the tissue around it, and sometimes there appears to be clear evidence that the disease began in the bursa itself. The bursa in front of the patella is the one most frequently involved.

When ulceration takes place the inflamed bursa is usually involved in the core, and has to be entirely removed before healing can ensue.

Inflammations of the periosteum and bones have for long occupied the most prominent place amongst the tertiary symptoms of Syphilis, and they are still some of the most common. In enumerating the symptoms which characterize the secondary stage, we have mentioned pains in the bones, attended occasionally by slight and temporary swelling. This kind of perios-

titis, however, never lasts long, and, as far as my own observation goes, never leads to suppuration. True nodes seldom occur until at least two years have passed since the first contagion, and generally the period is much longer. They may affect almost any parts of the osseous system, but the bones which are superficial, and therefore most exposed to external influences, are those most frequently attacked; *e. g.*, the calvaria, tibiæ, and the clavicles.

The bones of the palate, the alveolar processes of the maxillæ, the vomer, and other bones in the nasal passages, are very frequently affected, and when such is the case, exfoliation of portions usually occurs.

Syphilitic periostitis may vary considerably in its degree of severity and in its tendencies.

In some cases there is but little of acute inflammation, and the result is a great thickening of the bone affected, without the occurrence of suppuration. This frequently occurs in the bones of the skull—the whole calvaria acquiring greatly increased thickness and density. It is also not uncommon on the surface of the tibia and other long bones, constituting what is known as the osseous node. In other cases suppuration occurs, and in these very frequently large portions of cellular tissue become involved, and we have a swelling consisting in part of periosteal abscess and in part of a cellular node. When the bone is exposed by ulceration, exfoliation of portions often results.

When the bones of the skull are attacked by syphilitic periostitis it is very possible that inflammation may occur internally as well as superficially, and that we may have symptoms referable either to irritation of the cerebral coverings or to compression consequent upon intra-cranial abscess. In association with nodes on the skull, various symptoms of mental disturbance show themselves; extreme irritability of temper, liability to fits of uncontrollable passion, melancholia, and sometimes acute mania occur. These symptoms of mental disturbance may or may not be associated with those of local paralysis. They not infrequently result in attempts at suicide. The proof that they really are dependent on syphilitic lesions is afforded by the ease and rapidity with which they are relieved by the iodide of potassium. Some remarkable instances of this kind have recently been under my care.

Periosteal nodes are not very frequently met with on the short bones; we must, however, be prepared to recognize them occasionally on these also. The patella and the os calcis are not very infrequently affected, and now and then the other bones of the tarsus or carpus suffer.

Diseases of the muscular system occur chiefly amongst the most remote sequelæ of Syphilis, and they are by no means frequent. They usually take the form of nodes or gummata, developed in the substance of some single muscle. The induration is usually very considerable, and in many parts abruptly limited. The diagnosis from cancer is often very difficult, and many a mistake leading to an unnecessary operation and to a supposed permanent cure of cancer has occurred.

The muscular substance of the tongue is that most frequently attacked, but they have been met with in almost all the muscles of the body. We may mention especially the sterno-mastoid, the masseter, the supra- and infra-spinati, the gastrocnemius, and the rectus femoris.

I have recently had under care an extremely interesting case in which a tumor, which we at first suspected to be cancer, was developed in the left masseter of a lady who had twenty years before suffered from Syphilis. She presented at the time the tumor appeared no other syphilitic symptoms, and the correct history was only obtained with much difficulty. The tumor has wholly disappeared under the use of the iodide of potassium.

Some forms of syphilitic indurations of the tongue are exceedingly difficult to distinguish from cancer. They are very hard, have well-defined edges, are painful, and when they ulcerate present an unhealthy surface. Iodide of potassium in full doses will usually in the course of a week or ten days clear up the diagnosis. The heart itself is sometimes the seat of syphilitic nodes. Of this, M. Ricord[1] was, I believe, the first to record an example; but several others have been subsequently mentioned by other observers.

The Glandular System.—The chronic enlargements of the lymphatic glands, sometimes resulting in suppuration, are every now and then met with as the sequelæ of Syphilis, but it does not appear to me that they occupy any very important position. It is a remarkable fact in reference to tertiary syphilitic lesions generally that they do not cause any secondary enlargement of the adjacent lymphatic glands. This is true of syphilitic ulcerations of the skin and mucous membranes, of all the various forms of node, and of syphilitic tumors in muscles, and it often constitutes a very useful means of differential diagnosis between cancer and Syphilis.

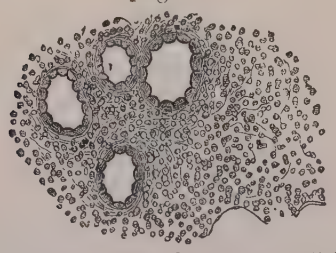
The Internal Viscera.—Of late years the investigations of pathologists have fully

[1] See Traité complete des Maladies Vénériennes, Planche xxix. In this instance the patient was a man aged 41, who had suffered from a chancre followed by constitutional symptoms eleven years prior to his death.

confirmed the conjectures of the older writers on Syphilis, as to the frequency with which the viscera of the trunk, and more especially the liver, suffer in constitutional Syphilis. In connection with this subject we must especially mention the very valuable contributions of Dr. Wilks. As to the exact period in the course of the disease at which the viscera are attacked, it is difficult to obtain any positive evidence. What we discover in the post-mortem examination is usually the result of long past disease, and it is comparatively infrequent to find it in a recent stage. What evidence we have, however, favors the belief that it is not until the later periods that the viscera suffer.

The liver appears to be far more frequently affected than any other organ.

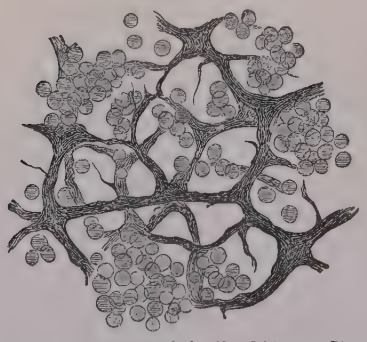
[Fig. 15.



The peripheral portion of a gummy growth in the kidney. Showing the small-celled granulation growth in the intertubular tissue. × 200. (From Green.)]

Indeed, in the examination of the bodies of those who have suffered from tertiary Syphilis, it is decidedly exceptional not to

[Fig. 16.



From a gummy growth in the kidney. Showing the reticulated structure occasionally met with in the intermediate zone of these formations. × 200. (From Green.)]

find proof of hepatic mischief. The most common condition consists in large white patches of fibroid thickening on the surface of the organ. These patches are evidently cicatricial. The liver is knotted and puckered by them, and bands of cicatrix dip from the surface into the substance of the organ. Sometimes, when the destruction has been great, the whole bulk of the organ is diminished. In recent disease the affected parts of the organ are enlarged, and on section exude a material not unlike bees-wax, or glutinous and gummy. I am not aware that abscesses have as yet been met with in the liver in supposed connection with Syphilis. Virchow recognizes two forms of disease—a capsular hepatitis and an interstitial hepatitis. Of these the capsular inflammation is the more common and the less serious. It is probable that the two are generally associated to a greater or less extent. Ascites occurs every now and then in connection with Syphilitic disease of the liver. An instance of it in a woman, the subject of inherited Syphilis with a contracted liver, has recently been under my care. The disease was of several years' standing, and paracentesis had been repeatedly performed. By a long course of iodide of potassium, with ammonia, the fluid was entirely removed and her health much benefited. Ascites from liver disease is not very infrequent in the subjects of inherited taint.

Testes.—Syphilitic sarcocele or syphilitic orchitis has usually been classed by authors as a secondary symptom. I feel sure, however, that this is not quite correct. It is amongst the earlier of the sequelæ, but seldom if ever occurs during the secondary stage. It is commonly met with in conjunction with nodes, and with deep ulceration of the skin rather than with the superficial rash of the secondary epoch. It consists of the free effusion of lymph (fibro-plastic material) into the substance of the testis, or, more rarely, into the epididymis.

The swelling often attains a very considerable size, and when it does so it presents the peculiar feature of feeling very light in the hand. Syphilitic sarcocele is much more frequently symmetrical than any other form of tertiary Syphilis. This circumstance we might expect from the fact that it occurs much nearer to the secondary stage than do most of the others.[1] Still, however, it is only exceptionally symmetrical.

Nervous System.—We come lastly to syphilitic affections of the nervous system itself.

I have previously adverted to the occasional occurrence of cerebral symptoms in

[1] On this point Mr. Curling writes, "Sir A. Cooper thinks that in the majority of cases, the disease attacks both testicles. The eight examples recorded in his work do not, however, bear out this remark, for in only two of them does it appear that both organs were attacked. According to my observation, the disease is more commonly confined to a single gland, though it occasionally affects both; and this also appears to be the opinion of Ricord."

connection with syphilitic inflammation of the bones of the skull, and to the formation of intra-cranial nodes; but, quite apart from the disease of its osseous case, the brain itself may suffer directly from the formation of tertiary syphilitic deposits in its structure. We may also have deposits of like nature into the substance of nerve trunks, producing special forms of local paralysis. To these isolated deposits the term syphilitic neuromata has been given, and several well-authenticated cases are on record in which the diagnosis has been confirmed by an autopsy. In a far greater number of cases the diagnosis has received an almost equally valuable confirmation in the cure of the disease by iodide of potassium. So frequently indeed is tertiary Syphilis the cause of paralysis, that investigations in this direction ought never to be omitted in cases in which the nature of the disease is in the least doubtful. It is, indeed, safe to go further than this and to say that in all cases of paralysis, without evident cause, and in which syphilitic antecedents are even possible, it is advisable to try the effect of iodide of potassium. I allude chiefly to cases of paralysis of the cranial nerves, for it would appear that neuromata of these are more frequent than of the spinal ones. Of the cases of paralysis of the fifth nerve, of the third, fourth, and sixth, which have come under my notice at the Ophthalmic Hospital, a large proportion of these have been of syphilitic origin, and most of these have been cured by the administration of iodide of potassium.

Syphilitic affections of the nervous system are usually among the late tertiary phenomena. I have rarely seen them at an earlier period than about five years after the primary disease, and in most instances the interval is much longer. In many cases the patients have had time to regain the appearance of good health, and almost to forget the malady from which they had formerly suffered. Under such circumstances the diagnosis is often surrounded with difficulty.

Syphilitic affections of the nerves of special sense do not appear to be common in connection with acquired disease, but they are not infrequent as results of inherited taint. In the subjects of the latter a form of cerebral deafness is often met with, and also one of complete blindness in association with white atrophy of the optic nerves.

TREATMENT OF SYPHILIS. — In approaching the question of the treatment of Syphilis we must always keep clearly in mind the facts which have been established as to its nature. Not indeed that *à priori* reasoning is to supersede empirical experience in such a matter, but rather that we shall do well to guide the one by the other. Viewing Syphilis as a disease of the zymotic class, caused by a specific virus which accomplishes its development within the body of the infected person, and passes through distinct phases or stages, nothing can be more probable than that in order to influence its cause we shall require very different measures in the different stages. The treatment which we should adopt in the onset of variola is not that which we should resort to after the exanthem has disappeared. We have then in respect to Syphilis to ask: First, what treatment should be adopted in the exanthematic or secondary stage? Second, what should be used against the very various sequelæ classed as tertiary symptoms? Third, whether there is any reason for believing that the development of stages, more especially of the exanthem, can be influenced by internal treatment adopted immediately after inoculation? Fourth, whether the sequelæ are rendered less or more severe by interference with the development of the early stages of the complaint?

As the present essay is on the *medical* aspects of Syphilis, we may suitably leave out of debate a matter of the utmost interest to the surgeon, that, namely, as to the prevention of constitutional infection by local treatment of the inoculated part (so-called "abortive treatment").

To cite an array of facts on this extensive subject would be wearisome, and without adequate result, nor should we more easily accomplish a summary of the very diverse opinions which have been published by medical authorities. [A general agreement exists among practitioners as to the propriety of endeavoring to arrest the specific morbid process of chancre by local applications. Early cauterization with solid nitrate of silver will sometimes eradicate the disease. If that fail, the alterative action of powder of calomel may be tried. Iodoform is preferred by some. Astringent washes, as lime-water, solution of sulphate of copper, &c., usually do good. When a bubo forms, in the inflammatory stage it may be treated with lotions of lead-water and poultices. If suppuration occurs, it should be freely opened with a bistoury. Should it then be slow to heal, the treatment will be essentially the same as for an indolent ulcer.—H.] The question as to the treatment of Syphilis resolves itself chiefly into one as to the efficiency or otherwise of mercury. We will submit for consideration answers to the following questions: Does mercury in any way influence the course of syphilitic symptoms? On this point I think almost all are unanimous. When given during the stage of induration of a chancre, mercury causes the absorption of the induration, and the healing of the sore; when given during the outbreak of

the secondary rash, it causes the rash quickly to disappear; it also causes the ulcers in the tonsils to heal. In cases of iritis and retinitis we have the most conclusive proof of the rapid absorption of syphilitic lymph under mercurial influence, inasmuch as in each of these conditions the inflamed structure is directly under our inspection. If we inquire as to the value of mercury against the tertiary symptoms, we obtain a much less positive answer, but we shall still meet with evidence in proof that over many forms it possesses a most decided power.

Having seen that mercury does, beyond all doubt, possess the power of shortening the duration of the primary sore, or if not used until the secondary manifestations have appeared, of causing these latter to disappear; we next have to ask whether the mercurial cure of any single stage, whether primary or secondary, influences beneficially the subsequent progress of the disease. The difference between an antidote for the syphilitic virus and a remedy for extant syphilitic inflammations, must be clearly recognized. It is one which has been acknowledged from the time of Hunter to the present day. It appears to me that the balance of evidence is in favor of the belief that mercury is a most potent remedy against syphilitic inflammations, but that it does not act as an antidote to the virus. I fear we have but little proof that mercury tends, on the whole, to abridge the duration or mitigate the severity of the syphilitic fever and its sequelæ.

During about two years in my practice at the Metropolitan Free Hospital, I systematically abstained from adopting any treatment in my cases of indurated chancre and its consequences. The chancre and the rash were allowed to develop themselves and to disappear spontaneously, and they did so in a fairly satisfactory manner. The duration of each was considerably longer than when mercury is given; otherwise I could observe no difference. The rash did not appear earlier, nor was it more copious than in the cases in which the remedy had been used. On the whole I had no reason to think that the patients suffered from the experiment beyond the fact of a more prolonged illness. As to what may be the relative frequency of tertiary symptoms in these cases it is as yet too early to speak. I have also, in private practice, not infrequently treated indurated chancres and secondary rashes without giving mercury.[1]

[1] I have preferred to speak from my own experience rather than to refer to the large amount of published evidence which exists. My own trial of the non-mercurial plan was made purely as an experiment, and without the slightest sentiment of partisanship.

As already stated, I think there is no proof whatever that by giving mercury for the primary sore, we diminish the probability that secondary symptoms will occur. These latter are for the most part inevitable, whatever may be the treatment employed. They are sometimes very slight indeed, and in some cases, perhaps, wholly omitted, but their non-occurrence is quite as frequent when mercury has not been given as under the opposite conditions. It is, therefore, unsafe to assume because in any one case in which mercury was given early, and no secondary symptoms ensued, that, therefore, the treatment prevented them. Such sequences are probably mere coincidences. Thousands of cases may be quoted in proof that mercurial absorption of the chancre does not prevent the secondary stage; and, further, that the mercurial treatment of both primary and secondary stages does not prevent the occurrence of tertiary sequelæ. We might also quote another class of facts in proof that mercury is not in any strict sense an antidote, those, namely, in which relapses occur either during or immediately after its use. These cases must be familiar to all. A patient, whilst actually salivated on account of iritis in one eye, becomes affected by the same inflammation in an acute form in the other, or just after the mercurial cure of iritis, retinitis occurs. Of the latter occurrence I have seen several marked examples. I admit that these quick relapses are exceptional, but they are still sufficiently common to become of great value in reference to the question under debate. The belief that mercury given in the early stages in any way complicates the case or adds to its subsequent severity is, I think, to a very large extent an error; at any rate it is quite certain that the worst forms of syphilitic symptoms, whether secondary or tertiary, not infrequently occur in cases in which no mercury has been used; especially is this a fact as regards tertiary symptoms, such, for instance, as extensive disease of the bones. In former days, when mercury was given so freely, it was not so easy to find cases of tertiary Syphilis without the history of previous mercurial treatment; in the present day, however, it is not at all infrequent.

If I might be allowed to express my own impression, founded as it is on a considerable number of facts, but for obvious reasons not easily susceptible of categorical proof, it would be to the effect that the course of Syphilis is on the whole rendered somewhat milder by early mercurial treatment.

Quite apart from the questions as to the general influence of mercury upon the course of Syphilis, we must estimate its value in the speedy removal of local in-

flammations. We have ventured to consider that it is a proven and admitted fact that this remedy does produce the rapid absorption of syphilitic lymph. When the lymph is effused into the skin, or at the base of the original chancre, it may be a matter of little or no consequence whether it is allowed to remain two weeks or two months. There is no material danger as to the integrity of the organ concerned. In the case of the eye and certain other organs, however, the facts are very different. If the iritis be allowed to proceed unchecked, it will in all probability end in obliteration of the pupil, either partial or complete. It will effect but little to use atropine, unless we use mercury also, for in many cases during the acute stage of the inflammation the pupil can be scarcely made to dilate until the lymph effused into the iris is in part absorbed. The longer the lymph is allowed to remain, the longer the inflammatory process is allowed to continue unchecked, the greater will be the risk of disorganization of the structure implicated.

These remarks apply with yet more force to syphilitic retinitis than to iritis. That it is the bounden duty of the surgeon to administer specific remedies in these diseases, no one who has considered the facts can, I think, doubt. It is not uncommon to see the retina in a case of severe retinitis become almost clear after a fortnight's mercurial treatment, with corresponding benefit to the patient's vision. It is on the other hand very common to see this disease remain unchanged for several months, if mercurial treatment be not adopted.

Those anti-mercurialists who carry their doctrines so far as to refuse to employ specific remedies when the eye is attacked, incur a responsibility probably far greater than they suppose. A case has recently come under my own observation so much to the point that I must mention it.

A gentleman engaged in the city consulted me concerning some symptoms which I easily recognized as the sequelæ of Syphilis. Amongst others he had muscæ and evidences of a past attack of retinitis. He gave me the history that he had been treated for the primary disease by a surgeon well known as an opponent to mercury. This gentleman explained to him in very strong terms the evils which he supposed to result from that drug, and so far secured his confidence that he continued under expectant treatment for several months. The eyes were attacked, and still specific remedies were abjured. "At length," said my patient, "when I was all but blind, and when for several weeks no improvement had occurred, I determined to take other advice, and consulted Mr. Critchett. Mr. Critchett assured me that the fear of mercury was all nonsense, and that the only chance for my sight was at once to go home, keep myself in a warm room, and take mercury till the mouth was sore. This I determined to do, and the result was that in the course of a week, I could see very much better, and that subsequently I regained almost perfect sight."

In this case, not only did the mercurial treatment rapidly cure the retinitis, but it removed the syphilitic rash and restored the patient's general health in a way which, to him, was marvellous.

When Syphilis attacks the larynx it becomes also of great consequence to adopt energetic treatment, on account of the danger to life which may accompany the local disease.

Although in the case of the skin we have to deal with an organ not essential to life, and the functions of which may be long interrupted with comparative impunity, yet it is still probable that a very extensive cutaneous inflammation, such as occurs in the exanthem of Syphilis, is not wholly without its injurious influence on the general health; thus there may be some reason alleged for preferring to get rid of a secondary rash in a few weeks by mercury, rather than to allow it to disappear spontaneously after several months' duration.

We come, lastly, to the question as to the treatment of the tertiary symptoms, or the sequelæ which occur several years after the contagion.

The marvellous power of the iodide of potassium in the cure of these affections is universally admitted, and has led to the almost entire disuse of mercury in their treatment. The only drawback to its employment is, that cures thus effected are rarely permanent, and that relapses are very frequent in a short time after its suspension. Some surgeons of large experience hold that mercury, even in regard to the tertiary symptoms, is more efficient than the iodide in bringing about a permanent cure. This is, however, open to much doubt. Practically, in all cachectic subjects suffering from tertiary symptoms, we usually administer the iodide alone, and if the cachexia be less severe we combine it with a mercurial. Another general rule on this point is, that the earlier in the rôle of tertiary symptoms the greater the desirability of mercury, and the more remote from the original taint the more likely is the iodide alone to be found efficient. Thus, cases of node, of gummous tumor, of tumors in muscles, and of affections of the nervous system, all of which are among the later of the tertiary class, are usually treated most satisfactorily by iodide of potassium.

What little experience I have had of the treatment of tertiary Syphilis, either by the mineral acids or by sarsaparilla,

has not been favorable to these remedies. We must not overlook in the management of cases of syphilitic sequelæ, the importance of change of air, of nutritious diet, and general attention to the patient's health. Such sequelæ, are especially apt to occur and to become severe in patients whose general health has been broken down. Any debilitating influence brought to bear upon a patient the subject of a latent taint may permit such taint to become active.

Thus, patients who have for many years enjoyed excellent health, and have believed themselves long ago and completely cured, may become, at the climacteric period, or when by any chance underfed and enfeebled, the subjects of tertiary Syphilis. Yet, whilst fully admitting the importance of sustaining the general vigor as a prophylactic measure against tertiary Syphilis, we must keep in mind that these measures will by no means prove always efficient. Many of the worst cases of tertiary Syphilis, whether consequent on inherited or acquired taint, occur in those whose circumstances of life enable them to enjoy every hygienic advantage.

With regard to the details of mercurial treatment, differences of opinion still prevail among surgeons. The majority still think the stomach the most fitting organ by which to introduce the remedy into the blood. There is no doubt that absorption by the skin, whether by inunction or the moist vapor bath, is very efficient, but there is no proof that it is superior to the other. Under the able advocacy of Mr. Langston Parker, Mr. Lee, and others, the calomel vapor bath has of late years obtained much repute. I have often used it, and have still more frequently seen cases in which it has been used by others, and have not been able to persuade myself that it possesses any real advantages. The fact is, that mercury in any form is so prompt in its results against syphilitic inflammations, that whoever is in the habit of prescribing it in one special form is very apt to come to the conclusion that his peculiar mode of use must be superior to others.

When there is a rash or ulceration on the cutaneous surface, it is always well to apply mercury directly to the inflamed part in addition to its internal use. This may be done conveniently by prescribing the mercurial ointment when there is no ulceration, and the black wash when the latter exists. Of the various preparations of mercury, I much prefer calomel or the bichloride, and rarely find that these, when used with suitable adjuvants, in moderate dose, disagree to any material amount. For the early forms of secondary rash I usually employ mercury alone, and for the latter ones mercury in combination with iodide of potassium. A mixture containing the solution of the bichloride with the iodide in excess is extremely useful. It probably amounts to bin-iodide of mercury with excess of iodide of potassium. The addition of ammonia to this mixture appears to increase its efficacy, and the same is the fact as regards its use with the iodide of potassium under all circumstances.

In prescribing mercury it is the surgeon's duty to carefully watch its effects upon the disease, and upon the patient's health. The fact that a given patient is cachectic and feeble is not in itself any reason for precluding resort to specific treatment. On the contrary, it is under such circumstances not infrequently that the value of the remedy is best shown. Nor can we lay down any rule as to the avoidance of mercury in treating certain forms of rash—ecthyma, rupia, &c. It may be stated in general terms that the more nearly the rash keeps to the scaly type, the more certain is it that mercury will agree; whilst the greater the tendency to ulceration and suppuration, the more is the risk that it may disagree. Should it disagree in any case, the observant surgeon will soon discover the fact. If the ulcers spread instead of healing, and if the quantity of secretion is increased, then mercury should be at once laid aside and substituted by iodide of potassium with tonics. The cases in which this substitution becomes necessary are, I believe, often those in which the patient has inherited partial immunity, and consequently suffers from a modified form of the acquired disease. Although mercury is very efficacious in the infantile stage of Congenital Syphilis, it often disagrees most markedly with the subjects of this taint when they have attained adult age. As a rule, in the management of secondary Syphilis, it is well to push mercury to a mild degree of ptyalism. Not infrequently symptoms resist its action until the gums are sore, and then yield at once. I have often observed this in inflammation of the eye, especially in retinitis.

With regard to the iodide of potassium against tertiary symptoms, a few simple rules may be given. It is well always to combine it with ammonia. It is well to begin with a small dose, *e. g.*, five grains three times a day, and to gradually increase, not going beyond ten or twelve unless necessary. If the disease does not yield to the latter, the diagnosis being yet undoubted, then much larger doses should be given; say, half a drachm three times daily. In a few rare cases, nothing short of these large doses will produce any benefit.

THE DIAGNOSIS OF CONSTITUTIONAL SYPHILIS.—The power of recognizing syphilitic diseases when brought under

notice is one of the most valuable gifts which the physician can possess. These diseases meet us at every turn in practice, and present a most bewildering variety of external aspect. At one time we have to distinguish a syphilitic rash from a simple one, at another to diagnose between Syphilis and Cancer, or Syphilis and Rheumatism, or the problem presented may be to form a correct opinion as to the nature of a paralysis, an ascites, or an attack of mania. The first requisite to success is a mind constantly awake to suspicion, and fully impressed with the all-important fact that diseases of the most diverse character may have their origin in this taint, and that if so they will prove to be curable only by treatment directed against it. This suspicion must be present, whatever may be the position in life or the reputation of our patient. A gentleman, who now at middle age bears the most irreproachable character, may chance to have been less circumspect during college life, and it is not fair that his subsequent purity of conduct should be the means of preventing his relief from the consequences of youthful error. We meet every day with cases in which women, whose characters are spotless, have become the subjects of syphilitic taint without their having the slightest suspicion as to the nature of their malady. Under many circumstances it is out of the question to make any direct inquiries, and the physician must depend upon his own acumen for the opinion which shall guide his treatment.

The recognition of syphilitic symptoms in the *secondary stage* is not usually difficult. The copiousness of the rash; its symmetry; the copper-tint; the frequent coincidence of several of different types of skin eruption in the same case; the presence of febrile disturbance; the absence of cutaneous irritation and the coexistence of sores on the tonsils, and frequently on the mucous membrane of the cheeks also—are all features which help to make the diagnosis easy and certain. To these we may add that the syphilitic exanthem usually appears first on the abdomen, chest, and fronts of the arms, that it very commonly affects the face, and that it avoids the backs of the elbows and the fronts of the knees, localities which are almost always attacked in cases of common psoriasis. Although syphilitic rashes vary very much in outward characters, yet they have always in the features just mentioned a basis of close similarity. When mistakes occur they are usually those of insufficient attention. The patient is allowed to show only a small part of his surface, instead of being made to strip, or at any rate to expose the whole of his bust. If the latter course be adopted, the symmetry of the rash and its other peculiar features will almost always arrest the attention of the observer. Amongst minor points which occasionally assist may be mentioned the gyrate or ringed form of the patches, and in some cases of syphilitic psoriasis the comparative absence of desquamation.

The cases which cause most difficulty are those in which Syphilis occurs in a patient who is already the subject of some other skin disease. In hospital practice it is very common to see scabies and a syphilitic rash coexisting, and in some such it is most difficult to pronounce with certainty as to the nature of the eruption. Examination of the mouth and throat and of the genitals will often remove doubt; but if not, a few sulphur baths as a measure of diagnosis will usually prove successful.

Next to that of exanthem itself comes the diagnosis of the *relapses of eruption*, which often occur between the secondary and the tertiary epoch. In these there is rarely any copious outbreak, usually only a few isolated patches. These are most commonly met with in the palms of the hands or soles of the feet, or on the front aspects of the forearms or legs. They are almost dry and attended with peeling of the epidermis. Very frequently there are small sores in the mouth or on the tongue; at the same time a form of acne, chiefly affecting the forehead, and leaving little pits or scars, is very often seen in this stage. If iritic adhesions are present, or if there are pits in the skin of the face and trunk, left by a former rash, the suspicion is much strengthened. If the diagnosis remains doubtful, we may cautiously try the effect of a short treatment with the bichloride or biniodide, and observe the result.

Lastly, we must consider the question of the recognition of the various diseases which come into the category of *tertiary symptoms*. When these occur, it is often many years since the patient has suffered from any other; and it is quite possible that he may appear to be in excellent health. A few of the tertiary symptoms have been so long recognized in relation to their true cause, and are so rarely met with in connection with any other, that in themselves they almost constitute their own diagnosis, and often also help us to that of more obscure lesions. Periosteal swellings or nodes are the chief of these. To speak generally respecting other forms, we may say that the diagnosis must be founded in part upon the patient's previous history, in part upon any still existing remnants of former disease—such, for instance, as iritic adhesions—and in part upon the peculiarities of the disease itself. As regards the patient's antecedents, I may just remark, by way of caution, that we must not hastily assume

that he is syphilitic because he tells us that he has had the venereal disease. A soft chancre with its suppurating bubo, or even an attack of gonorrhœa, although both of them quite innocent as regards constitutional infection, often leave more vivid impressions on the patient's mind than does an indurated sore and its exanthem. Those who are most ready to suspect a venereal cause, are often those who have never had true Syphilis at all. If, however, there is a clear history of a chancre followed by secondary rash, sore throat, &c., then we have obtained a fact which, whatever may be the present ailment, may be safely permitted to modify our treatment. The majority of tertiary lesions are by conventional usage regarded rather as surgical than medical, and it would be out of place to speak in detail of the diagnosis of ulcers, gummous tumors, &c. I may briefly remark that the serpiginous form of ulceration, healing in the centre and spreading at the margin, is a feature always to be regarded with suspicion; that tumors in muscle, which will only melt away under the influence of the iodide, are sometimes as hard and as defined as any variety of chancre, and have often led to needless operations.

In cases of disease of the nervous system in which Syphilis is suspected, an examination of the patient's eyes, throat, tongue, and tibiæ should never be omitted. The existence of iritic adhesions, of cicatrices of the soft palate or of periosteal nodes, will often decide the question. The occurrence of nocturnal exacerbations of pain is also always suspicious. If the disease implicate only one nerve-trunk, especially if only one cranial nerve be involved, the suspicion of Syphilis becomes very strong. Probably a full half of the cases of paralysis of the third, fourth, fifth, and sixth nerves, when such paralysis affects only one nerve, are due to Syphilis and are curable by specific treatment. In these cases the disease is hardly ever symmetrical, and the paralysis is usually complete. The seventh nerve is occasionally attacked, but not so frequently as the others. The nerves of special sense are not so frequently affected in acquired Syphilis as they are in the inherited form. Nevertheless, cases do occasionally occur in which amaurosis or complete deafness are met with in the subjects of syphilitic taint, and without other assignable cause. In these the loss of function is usually symmetrical, and probably depends upon disease of the cerebral centre rather than neuromata developed in the nerve-trunks. I am not aware of any cases in which paralysis of the branches of the eighth pair have been traced to Syphilis, but no doubt such occur and might be recognized by due search. Paralysis of single nerve-trunks of any of the spinal plexuses—more especially those of the brachil plexus—are now and then encountered.

In cases of tertiary Syphilis the bones of the calvaria not infrequently increase greatly in thickness and weight without any formation of external node, and under such circumstances there is very often a roughened state of the surface in contact with the dura mater. Various forms of disturbance of the sensorial functions are usually observed in these cases. In addition to violent headache there is irritability of manner, loss of memory, and sometimes actual mania.[1] The diagnosis must depend upon the facts to which I have already adverted.

The Diagnosis of Constitutional Syphilis when consequent upon Inherited Taint.

The diagnosis of inherited Syphilis rests on somewhat different data to that of the acquired disease. Indeed, the whole course of the disease, as thus transmitted, presents some remarkable features of difference which I have endeavored to bring into clear contrast in the appended tabular parallel. (See p. 443.) Some local lesions, not infrequent in those who have inherited the taint, never occur at all in those who have acquired it, as for instance, interstitial keratis. Others present important modifications of character; thus, when periosteal nodes occur in children, they are much more extensive than is usually the case in adults. Speaking generally, the tertiary symptoms of inherited Syphilis, however long may have been the interval of latency, are for the most part symmetrical. We have seen that those of acquired Syphilis are but rarely so.

The stages observed in the course of inherited disease are very similar to those of the acquired form, but they much more frequently run into each other.

We must consider the question of diagnosis in reference to the three different stages: first the infantile period; second, the stage of latency; and lastly, that of tertiary symptoms (usually about the age of puberty).

In the infantile period we recognize Syphilis by the peculiarity of certain single symptoms, or, more frequently, and

[1] A young man was recently admitted under my care into the London Hospital, having attempted suicide (by cutting his throat) in a state of mania. He had disease of the alveolus and nasal bones, which led me to diagnose Syphilis. We gave him full doses of iodide of potassium, and he rapidly recovered. When his mental faculties had returned, he gave us a history which fully confirmed our suspicions as to specific taint.

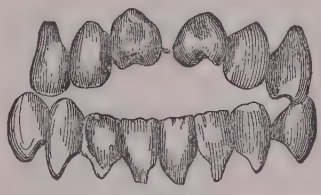
with greater certainty, by the peculiar grouping of several different symptoms. First in importance is the rash on the skin. The rash, as in acquired Syphilis, may vary much in its character, but the commonest are those of erythematous or papular character. If it is erythema, the redness will show itself in abruptly margined patches, and will be characterized further by its peculiar red or coppery tint, compared by some authors to that of the lean of ham. Sometimes we see instances of dry, scaly rashes in infants, but these are rare. Pustular, vesicular and bullous rashes are also not infrequently witnessed. Condylomata at the anal orifice are common, though less frequent during the first few months than at later periods. At the same time as the rash the little patient almost always displays the characteristic symptom known as "snuffles," and there is usually inflammation of the mucous membrane of the mouth, and sores at its angles. (See p. 429.) Iritis occurs in a few cases and has similar tendencies to those witnessed in the acquired form, and is equally under the influence of specific treatment. It occurs also at the same stage, always amongst the secondary symptoms. Inflammations of the deep-seated structures of the eye—of the vitreous, retina, choroid—are as frequent as they are in the adult, and present the same characters.

During the stage of outbreak of the exanthem, which lasts on the average from the fourth week to the sixth month, the child becomes fretful, pale, and emaciated; growth is for a time arrested, and his shrivelled face resembles that of an old man. Emaciation is certainly the rule, but it has many marked exceptions, and I have often seen syphilitic infants who were fat and plump and looked remarkably well.

At or about the age of one year, if the child have survived, it is usual for the secondary symptoms to wholly disappear. The period of latency now ensues, during which the child enjoys often very good health. Sometimes relapses occur, and especially are such subjects liable to be affected by condylomata. These relapses scarcely ever involve a return of cutaneous rash. I think that all observers will bear me out in the statement that the characteristic rashes so often seen in syphilitic infants are never witnessed at later periods of life. The tertiary epoch may begin at any period after the fifth year, but it is commonly delayed till at or near the period of puberty.

The recognition of the subject of inherited Syphilis, at or after the age of puberty, may be sometimes made with great certainty, and is at others surrounded by difficulties. Our most valuable aids are the evidences of past disease, more especially of the inflammations which may have occurred in infancy. A sunken bridge of nose, caused by the long-continued swelling of the nasal mucous membrane when the bones were soft, a skin marked by little pits and linear scars, especially near the angles of the mouth, the relics of an ulcerating eruption, and a protuberant forehead, consequent upon infantile arachnitis, are amongst the points which go to make up what we recognize as an heredito-syphilitic physiognomy. Added to them we have very valuable aid furnished by the shape of the incisor teeth. In these patients it is very common to find all the incisor teeth dwarfed and malformed. Sometimes the canines are affected also. These teeth are narrow and rounded and peg-like; their edges are jagged and notched. Owing to their smallness their sides do not touch, and interspaces are left. It is, however, the upper central incisors which are the most reliable for the purposes of diagnosis. When the other teeth are affected these very rarely escape, and very often they are malformed when all the others are of fairly good shape. The characteristic malformation of the upper central incisors consists in a dwarfing of the tooth, which is usually both narrow and short, and in the atrophy of its middle lobe. This atrophy leaves a single broad notch (vertical) in the edge of the tooth, and sometimes from this notch a shallow furrow passes upwards on both anterior and posterior surface nearly to the gum. This notching is usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge, and at others they slant towards each other. The appended woodcut (Fig. 17) illustrates a good

Fig. 17.



Syphilitic teeth.

example of the deformity. In any case in which the malformation was as marked as

Fig. 18.

Syphilitic teeth.

in this sketch, I should feel no hesitation in pronouncing the possessor of the teeth

to be the subject of inherited Syphilis, even in the absence of other testimony. I have never yet seen such teeth, excepting in patients of this class. In the majority of cases, however, the condition of the teeth is sufficient only to excite suspicion and not to decide the question. In a few rare cases only one of the upper central incisors is malformed, the other being of natural shape and size. A good instance of this state of things is shown in Fig. 18.

In a considerable number of cases of heredito-syphilis the teeth show no deviation whatever from the normal standard, and in such the diagnosis must be guided by other conditions. In addition to the peculiar malformations above described and illustrated, there are others which, although less characteristic, are yet very valuable to a trained observer. They do not, however, admit of description without great risk of misleading the reader. Before leaving the subject of dental malformations, I may again ask attention to the fact, that it is only in the permanent set that any peculiarities are observed. The first set are liable to premature decay, but are not malformed.

In addition to the peculiarities of physiognomy and the malformations of the teeth, the diagnosis may be much helped by observing the state of the eyes and of the bones. If there be evidences of past iritis, or if there be clouds in the substance of the corneæ, the results of past keratitis, or especially if the corneæ be now attacked by this peculiar inflammation in its acute stage, very valuable evidence will have been obtained. The phenomena of syphilitic keratitis in its acute stage are peculiar and easily recognized. Both eyes are usually affected at the same time. The corneal tissue becomes very extensively opaque by the effusion of lymph into its substance. Its tint may vary from that of ground glass to a red salmon color. There are no ulcers on its surface. A zone of ciliary congestion is usually well marked. The patient is often for several months, whilst the disease is at its height, practically blind. The intolerance of light is usually considerable. After the inflammation is passed away the cornea usually clears in a most remarkable manner, but it rarely regains such perfect transparency that the experienced observer cannot detect traces of what has taken place. These traces consist in a somewhat dusky and thin sclerotic in the ciliary region, and in the presence of slight clouds here and there in the corneal substance, there being no scars in its surface. The difference between these interstitial clouds and ordinary leucomata is easily observed.

In a few cases the existence of nodes on various long bones may help us to a diagnosis, and in others we may obtain aid from finding that the patient has become deaf without otorrhœa, or that he is partially amaurotic from choroiditis.

With regard to the general arrest of development in heredito-syphilis, I may remark that it is a very untrustworthy indication. In a few cases this taint dwarfs the whole body in a most remarkable manner, but in most cases no retardation of general growth is observable. A pale complexion is most always met with. It is exceedingly rare to meet with a florid good complexion in a young adult who is the subject of this taint. We do, however, every now and then see a physiognomy which neither in shape of features nor in color of cheeks and lips furnishes the slightest clue. I have met in one or two instances with arrest of sexual development. In one of these, a young woman under the care of Dr. Hughlings Jackson in the London Hospital, there was such an entire absence of all sexual characteristics that I could not but suspect that the ovaries had been destroyed by syphilitic inflammation in early life.

Contrasted Parallel between the Course of Symptoms in Acquired and Inherited Syphilis.

I have endeavored in the following tabular statement to place as clearly as I can the resemblances and differences which we observe in the course of symptoms when arising from acquired or from inherited taint. To some of these I have already incidentally alluded, and respecting the others the statements in the table will, I trust, explain themselves:

	Acquired Disease.
Primary Stage. — Local or stage of inoculation. The sore appears after an incubation period of from ten to twenty-eight days, and if not treated may remain from a fortnight to six months. Liable to relapse.	An ulcer (chancre) usually with indurated base. Indurated lymphatic glands. Induration is to be regarded as the earliest proof of successful inoculation, but the latter is sometimes effected without any hardness of the original sore having shown itself.
Secondary Stage.—Constitutional or exanthematic. Usually commences within six weeks or two months of the inoculation, and if not treated, may last from three to six months or to a year. Essentially transitory, and will disappear without treatment.	Febrile disturbance, malaise and muscular pains. Slight engorgement of lymphatic glands in many parts. A symmetrical, and usually copious eruption on the skin, and often on exposed mucous surfaces. Symmetrical ulcers in tonsils. Iritis, retinitis, &c., usually symmetrical. Loss of hair, loss of flesh and of strength. This stage may be either exceedingly slight or very severe. Its severity appears to bear proportion to the degree of induration of the preceding chancre. It is often noticed that the rash comes out in successive crops. The rash may also vary very widely as to its character, roseolous, scaly, papular, pustular, ecthymatous, &c., being modified probably by peculiarity—first, in the source of contagion; secondly in the state of health of the recipient.
Intermediate Stage.—Stage of latency and of relapses. This stage may be said to commence at from a year to a year and a half after the contagion, and to extend over a period which may vary from three to five, ten, or even twenty years.	The patient may be either wholly free from symptoms and in good health, or he may remain pale and rather feeble, and liable from time to time to slight returns of eruption on the skin, sores on the mucous membranes, condylomata, &c. He is protected as regards fresh contagion, and should he beget children they are almost certain to suffer. The relapses during this stage are usually easy to be distinguished from true secondary symptoms. There is little or no febrile disturbance, the rash is not copious, and often not symmetrical. Acute iritis, retinitis, &c., never occur; that is, they do not occur for the first time; they may occur in the form of relapses.
Tertiary Stage or stage of sequelæ. This stage commences at from four to ten or to twenty years after the contagion, and extends indefinitely, very often to the end of life.	All the symptoms in this stage occur, as a rule, without symmetry; sometimes multiple, but not infrequently single. They consist of chronic inflammations of deep tissues, or of the deeper layers of superficial ones, *e. g.*: —Inflammations of periosteum and bone, resulting in nodes; of cellular tissue, tendon, or muscle, resulting in gummy tumors; ulcerative destruction of the palate and pharynx; serpiginous ulcerations of the skin; inflammation of nerves, or even of cerebro-spinal centres, inducing various forms of paralysis; deposits in liver, lungs, &c. Probably but little liability to transmit the disease to offspring. Protection against a new contagion incomplete. All the inflammations in this stage are remarkably under the influence of treatment by iodide of potassium, but tend to relapse. Unless so treated, all of them tend to progression and permanent disorganization of the part attacked, none of them to spontaneous recovery.

INHERITED DISEASE.

Primary Stage.
The infants usually remain without symptoms for from one week to three months.

This stage has been passed through by one or both of the sufferer's parents within from a few months to twenty years of the infant's birth. The infant is usually free from all symptoms at the time of birth.

Secondary Stage.—Constitutional or exanthematic.
From the age of two to four weeks to the end of the first year.
This stage is essentially transitory, and will disappear without treatment, if the child lives.

Inflammation of nasal mucous membrane causing "snuffles."

A symmetrical and usually copious eruption on the skin. Wasting; fretfulness; a peculiar odor; a withered, senile aspect; inflammation of the mouth, and condylomata at anus; iritis, usually symmetrical; arachnitis and slight effusion; disease of liver (rare); nodes (very rare). The eruptions which occur differ from those of acquired disease, chiefly in being more moist, and in preferring the thighs and genitals. These differences may in part be due to peculiarities in the skin of young infants, and to the constant irritation from urine to which the nates are liable. Dry scaly rashes are rare. Iritis is much less frequent than in the adult, but just as well characterized when it does occur.

In infants this stage often proves fatal.

Intermediate Stage.—Stage of latency.
This stage extends from the end of the first year or eighteen months to the second dentition, the time of puberty, or even very much later.

The patient will probably be wholly free from active symptoms, but will show various indications of his diathesis in pallor of skin, sunken nose, protuberant forehead and premature loss of the upper incisor teeth. Sometimes there will be a remarkable retardation of growth and general development. If second dentition have occurred, the central upper incisors will be malformed. Unlike what happens during this stage in acquired Syphilis, we scarcely ever observe any tendency to recurrence of the secondary symptoms. Now and then we see condylomata at the anus returning during the first five years, but the rash of infantile Syphilis having once disappeared, I think scarcely ever relapses. A certain degree of nasal obstruction sometimes persists, but not often.

Tertiary Stage or stage of sequelæ.
This stage may commence with the second dentition, at the time of puberty, or not till much later. Its duration is quite indefinite.

Most of its symptoms are symmetrical:—

Keratitis (interstitial); kerato-iritis; periosteal nodes; cerebral deafness (not infrequent); cerebral blindness (rare); disease of liver and kidneys; phagedænic or serpiginous ulcerations of skin; cellular nodes (rare). Probably not liable to transmit the disease to offspring. Protection against a new contagion incomplete. The symmetry of the symptoms is in marked contrast with what occurs in this stage of acquired disease. The paralyses of single cranial or spinal nerves, so common from acquired Syphilis, are, I believe, never met with in the inherited form.

Most of the inflammations tend, unless arrested by treatment, to permanent disorganization, but one (interstitial keratitis) tends to recovery even without treatment. They are much less easily influenced by treatment than those of the acquired disease.

GENERAL DISEASES, OR AFFECTIONS OF THE WHOLE SYSTEM.—*Continued.*

§ II.—*Those determined by conditions existing within the body:—*

Scorbutus.	Rickets.	Rheumatoid Arthritis.
Purpura.	[Scrofula.]	Rheumatism.
[Chlorosis.]	Gout.	Gonorrhœal Rheumatism.

SCORBUTUS.

By Thomas Buzzard, M.D.

Scurvy, or Scorbutus, as it is technically called, is a peculiar state of malnutrition, supervening gradually upon the continued use of a dietary deficient in fresh vegetable material, and tending to death, after a longer or shorter interval, if the circumstances under which it arose remain unaltered. The condition is essentially marked by a dull leaden pallor of complexion; excessive bodily debility and mental lethargy; dyspnœa upon slight exertion, unaccounted for by the auscultatory signs; spontaneous effusions of blood-colored fluid into the various tissues of the body, causing petechiæ and bruise-like patches to appear on its surface; together with (commonly) a livid, swollen, and spongy state of the gums, and a disposition for them to bleed upon the slightest irritation.

Synonyms.—*French*, Scorbut; *German*, Scharbock; *Italian*, Scorbuto; *Spanish*, Escorbuto. These terms, as well as the English Scurvy, take their origin from the Danish Skörbeck, "Disease of the mouth," of which the word "Scorbutus" is a barbarous Latinized version.

Etiology.—Very much has been written upon the subject of Scurvy. Long before the disease was styled by the name which it now bears, and when, indeed, it was often called the "unknown disorder," historians noted its ravages in armies located, under circumstances of difficulty, in foreign lands, or in garrisons shut off from obtaining supplies of requisite nutriment. In the long sea-voyages which the intrepid navigators of the fifteenth and sixteenth centuries commenced, the crews suffered terribly from a disorder which destroyed the lives of large numbers, and affected the working power of the survivors in a manner equalled by no other disease. It is probable, indeed, from the records which have come down to us, that Scurvy, either alone or as influencing the severity of accompanying maladies, has proved more destructive to mankind than any other disorder. There is no more interesting fact in the history of medicine than that this condition, which has been looked upon at various times as plague, as a mysterious infliction of Divine justice against which men could only strive in vain, or as a disease inseparable from long voyages, should have been proved, by evidence of the most satisfactory character, to arise from causes in the power of man to prevent, and to be curable by means which every habitable country affords.

Scurvy only occurs when fresh vegetable nutriment has been for some time partially or completely withheld. A variety of forms of impaired nutrition will follow the want of other descriptions of food, but this particular condition is only seen as a sequel of that special privation. Scurvy

does not occur when the supply of wholesome and fresh vegetables is abundant, even though the food generally may not be adequate to perfect nutrition. There is a degree of positiveness about these two assertions which can rarely be ventured upon in the etiology of disease. In this case, so abundant and conclusive are the proofs, that to assert less strongly would be to imply a doubt which cannot be allowed to exist. The grounds for these statements are to be found in the voluminous records of the circumstances under which the disease has occurred, as related by observers, either unbiased by any theory of their own, or widely differing in their opinions as to the cause of the disorder. They may be said to form the negative side of the argument. The affirmative is based upon the fact, universally allowed by those largely experienced in the disease, of the power possessed by fresh vegetable material, and by that alone, in removing the disease.

It is not our intention to quote at length the records of Scurvy outbreaks, in proof of these assertions. We shall but glance at some of the more prominent instances in recent times, referring the reader for more elaborate details to the vast bibliography published upon the subject by writers of almost every nation. In the exhaustive treatise upon this disease, by Dr. Budd, in the Library of Medicine, will be found a history of Scurvy, which has served more than any other publication to place the disease in its true light, and the views expressed in it have been remarkably confirmed by the experience of the several outbreaks which have occurred since it was written.

In 1846 the potato crop failed in the United Kingdom. In the autumn, winter, and following spring, numerous cases of Scurvy occurred amongst all classes of society. Dr. Christison has described[1] an outbreak of the disease amongst the laborers employed upon the Scotch railways. Their food consisted of bread, salt pork, butter, cheese, coffee, tea, and sugar. "Potatoes were, of course, out of the question. Fresh vegetables were never thought of, and were, indeed, in most places inaccessible." The quantity of food seems to have been sufficient. In the Royal Infirmary of Glasgow, 83 cases were admitted. Dr. Ritchie[2] writes: "The general fact in regard of the food of all was that it failed in variety, and in the quantity of its animal constituents, and that in all but a fraction of the cases in which they were very deficient, the patients had been exposed for months to a total deprivation of fresh succulent vegetables." "In Carlisle and its vicinity," Dr. Lonsdale informs us,[1] "the persons chiefly afflicted were weavers and their wives and daughters working in the factories, shoemakers, and comparatively few of any other kind of artisans. Bread, oatmeal, treacle in very small quantities, tea and coffee, with an occasional herring, formed their entire food. None had tasted potatoes after the harvest of 1846, or for a period of seven or more months." Dr. Lonsdale states also that in a great number of the huts occupied by the railway excavators, amongst whom there were numerous cases of the disease, some of which proved fatal, he saw the men breakfasting off beef-steaks or mutton-chops and bread. The dinner comprised bread, boiled beef or bacon, pea soup or broth, and suet puddings containing currants. The animal food was taken in large quantities; there were no potatoes or fresh vegetables. At Workington (a seaport town of seven thousand inhabitants) the disease did not show itself. Dr. Dickinson, a resident, assigned as a reason, "that vegetable food was more abundant there than in many situations, particularly turnips, of which large quantities were used."

The same kind of evidence is adduced by Dr. Curran[2] in his description of the occurrence of the disease in Ireland. "In four-fifths of the cases reported to me, bread and tea or coffee was what the patients had been living on when attacked; the others had been using grains of various kinds, or grains and flesh or fish; but *in no single instance* could I discover that green vegetables or potatoes had formed a part of their regular dietary." Dr. Shapter[3] remarks, in reference to the cases of Scorbutus observed by him in Exeter, that the only difference from the usual diet of the sufferers consisted in the absence of the potato.

There is no doubt that the failure in the potato crop, besides depriving the population of this vegetable, incidentally also rendered their nutrition imperfect, by increasing the price of provisions in general. This circumstance might naturally, therefore, be believed to bear its part in the causation of this particular morbid condition, were it not that the disease was not confined to the poorer classes of society. Dr. Shapter relates that many of his patients were persons who had experienced no difficulty in procuring an abudance of the necessaries of life, with the exception, however, of potatoes or fresh vegetables. Dr. Christison notes the occurrence of Scurvy amongst railway laborers "earning ample wages, and whose extravagance in good living was a

[1] Edin. Monthly Journal, July, 1847.
[2] Ibid.

[1] Edin. Monthly Journal, August, 1847.
[2] Dublin Quarterly Journal, 1847.
[3] Lond. Med. Gazette, vol. iv.

frequent subject of remark in their neighborhood."

The allied armies of England, France, Turkey, and Sardinia suffered severely from Scurvy in the Crimea and Asia Minor during the war with Russia, 1854–56. The disease first began to show itself amongst the British troops in Bulgaria, when they had been living for some months upon an inferior diet, with but a very scanty supply of vegetables. On their arrival in the Crimea, the men found an abundance of grapes, cabbages, &c., which were eagerly consumed; and although the rations in other respects were most imperfect, no cases of the disease were recorded in September. As the winter advanced, however, and the vegetable food ceased, the affection reappeared. It was no part of the ordinary duty of the Commissariat to supply vegetables, which did not constitute a part of the soldier's rations. In consequence of representations, a supply of lime-juice was ordered, and arrived by the *Esk* on the 10th December. By one of those accidents, of which so many lamentable instances occurred during the early part of the war, no portion of the juice was issued until the first week in February, 1855, and then the supply was very insufficient. In March nearly all the sick arriving at Scutari from the Crimea were suffering from Scurvy. The total admissions from Scurvy during the war amounted to 2096, but "the returns convey but a faint conception of the disastrous part which it acted among the troops, for although it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was wide-spread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fever and affections of the bowels."[1] As the supply of fresh vegetables and lime-juice became more constant, the disease gradually disappeared, and comparatively few cases occurred during the second winter.

The sufferings of the French from this cause were proportionally much greater than those of our troops. This must be ascribed to the fact that the distribution of lime-juice formed no part of their practice. In fact, even at the present time, the French authorities do not appreciate at its full the value of this addition to the diet of either soldiers or sailors. No less than 23,000 cases of Scurvy are recorded as occurring amongst the French troops.[2] From the month of February, 1855, fresh meat was supplied to them at first twice and then five times a week. It was of good quality, but lean. The supply of bread was irregular, and fresh vegetables formed no part of their rations. Rice was allowed, and occasionally dried vegetables, principally peas, beans, and lentils, figured in the diet, but in small proportions.[1] There was this which was remarkable in the outbreak: The disease first showed itself in the winter of 1854–55, and committed terrible ravages. As the season opened and the earth began to bring forth vegetation, Scurvy diminished. The troops were encouraged to collect herbs, and especially dandelion, which was very plentiful, and of which the effects are highly anti-scorbutic. The improvement which took place under these circumstances was very marked, and there is no reason to believe that it would not have been permanent had the supplies continued. But as July approached, the rays of the sun dried up the surface of the ground on which the troops were encamped, no more dandelion was to be obtained, and in the course of three months, the finest and warmest in the year, no less than 5000 cases of Scurvy occurred. It may be remarked here, incidentally, that this outbreak furnishes a striking contradiction to the theory which ascribes the scorbutic condition to the influence of cold and damp.

The Sardinian army, which arrived in the Crimea in the early summer of 1855, was very generally affected by Scurvy. Its ravages were checked by vegetables supplied to the troops as the season advanced.

It is most probable that the Turks experienced even more severe losses from Scurvy than our other allies; but, from the imperfect organization of their medical department, exact statistics are wanting. There is no doubt, however, that the original force which formed part of the expedition from Bulgaria to the Crimea was almost entirely swept off by disease, of which Scurvy formed an important element. Of the fresh troops, under the command of Omer Pasha, which reinforced these men, and which were posted at Eupatoria during the winter of 1854–5, as many as 1000 were sent down monthly to Varna, all of whom were suffering severely from this cause, and a very large number of whom died upon the passage. During the summer the remainder, amounting to about 20,000, were encamped in the neighborhood of Balaklava. We had ourselves the opportunity of observing them narrowly. Their food was very imperfect; but they showed ingenuity in availing themselves of such

[1] Med. and Surg. Hist. of the British Army, 1858. By Authority.

[2] Rélation Médico-Chirurgicale de la Campagne d'Orient, par le Dr. G. Scrive. Paris, 1857.

[1] L'Union Médicale, 1857, p. 419: M. Perrin.

fruits and vegetables as were obtainable, and they were supplied with onions. They consumed, especially, large quantities of watermelons, which were procured from the Tartars inhabiting the country, or from sutlers. No cases of Scurvy fell under our observation at that time, nor could we detect any scorbutic taint in patients suffering from other diseases. In November they quitted the Crimea, and campaigned in Mingrelia. During the winter all supplies of vegetables ceased, and, with the early spring, shiploads of sick were brought to Trebizond, all of whom were severely affected with this disease. Large numbers died upon the sea-passage, in their transit from the shore to the hospital, and soon after their admission. The sick included numerous examples of Scurvy, developed to an extent which recalled the terrible descriptions of the disorder contained in the narratives of our early voyagers. Such cases as these were rare amongst the other allied troops. In explanation of this fact, it must be noticed that throughout the winter fresh vegetable food had formed no part of the rations distributed to the Turks, and, superadded to this, was absolute starvation, from the absence of food in sufficient quantity. Their diet, indeed, had consisted entirely of biscuit, a little rice, haricot beans, and "yagh," a coarse butter made from mutton fat.

Dr. Hammond informs us that, during the recent war in America, no confirmed Scurvy appeared among the Federal forces, but a scorbutic taint often manifested itself. He attributed its occurrence to occasional deficiency in the supply of vegetable food, exposure to cold and damp, and mental depression. He did not find that salt meat had any influence in its production. If the men had vegetables they could eat salt meat with impunity. Raw potatoes preserved in molasses were commonly issued to the troops, and were found of signal service in preventing the disorder.

So also on the Confederate side, Dr. Darby, late medical director, in a communication which he has been kind enough to send to us, says, "The type of the disease characterized by petechiæ and spongy gums was rarely known in the Confederate army. Diseases and injuries incidental to army life assumed at times a scorbutic taint, at such seasons and under such conditions as give rise to this malady. The abundance of proper supplies in the early stages of the war prevented scorbutic tendencies. In proportion as a decrease of supplies took place, there was an increase of the scorbutic type in all disease."

Since the year 1795, Scurvy, unless under very exceptional circumstances, has been all but abolished from the British fleet, and for this the name of Dr. James Lind, "the father of nautical medicine," deserves to be held in lasting honor. The combined observation of exploring navigators had tended to show that the disease could be cured by supplies of fresh vegetables, and its occurrence prevented by a similar diet. It was reserved for Dr. Lind, in his celebrated work on Scurvy, published in 1753, to give overwhelming proofs of the efficacy of lime-juice as a prophylactic in this disease, and forty-two years afterwards (!) the Admiralty took the hint. To Captain Cook, especially, science is indebted for a practical exposition of the influence of vegetable food. By providing his crew with abundance of sauer-kraut, and encouraging them to seek wild vegetables wherever he landed, he preserved their health completely during a four years' voyage of his ship *Discovery*. The same principle, though in the more convenient form of a daily ration of lime-juice, suffices to prevent the disease in the royal navy. The merchant service still, however, continues to furnish cases of this preventable disorder. Although the Legislature insists, under a penalty, that lime-juice shall be issued to the crews, the provisions of the Act are but too frequently evaded, and the Dreadnought Hospital still continues to receive annually an average of ninety cases of the disease—about one in twenty-five of all patients admitted.[1] Practically, in many cases, no lime-juice is furnished; or a cheap imitation of the juice, consisting of tartaric acid, sugar, and water, flavored with essence of lemon, is substituted. On the other hand, emigrant and convict vessels sailing to Australia, being under more complete Government supervision, convey their passengers without loss from the disease.

Now, it may be argued against such facts as we have recorded, that inasmuch as these outbreaks of Scurvy have always occurred amongst persons in an unnatural state of existence, in periods of famine, landed with insufficient provision in an enemy's country, or cooped up on board ship in long voyages, there may be some other special privation to which the occurrence of Scurvy is quite as likely to be

[1] There is an excellent article upon the subject by Dr. Barnes in the Sixth Report of the Medical Officer of the Privy Council, 1863. See also an able report by Mr. Harry Leach, resident medical officer to the *Dreadnought* in a House of Commons' return, "Scurvy in Merchant Ships," June, 1865. Mr. Leach ascertained by inquiry that of eighty-three Scurvy patients admitted in 1864, forty-two had received bad lime-juice; eighteen had had none at all; fourteen could give no exact account; and in the case of nine only was good lime-juice declared to have been taken.

due. This argument, in fact, has been repeatedly employed, and even at the present time it is still occasionally urged. In opposition to the view which we advocate, several causes of Scurvy have been insisted upon by various writers. Some have attributed the disease to the action of some one noxious agent, whilst others, and this is more common, urge that a combination of circumstances is necessary for its production. The point upon which there still exists the most important difference of opinion is regarding the influence of *salt meat*. There is no doubt that, in the great majority of Scurvy outbreaks, salt meat has formed an important part of the food taken by the sufferers. But, in order that this circumstance should be possessed of any weight, it would be necessary to show that the disease was never known to occur unless this description of food had formed at least a part of the dietary. There is evidence in plenty to the contrary.

During the campaign of Louis IX. of France, in Egypt, 1249, the army was frightfully ravaged by Scurvy, of which a most graphic description is afforded us by the historian.[1] During Lent, which was very strictly observed, the troops ate no meat, but subsisted "on eel-pouts, which is a gluttonous fish."

During the war in Hungary, in 1720, between the Austrians and Turks, many thousands of the former were cut off by Scurvy. Kramer informs us that they ate no salt beef or pork; but, on the contrary, had plenty of fresh meat at a very low price.[2]

The French prisoners confined in Sisinghurst Castle, in Kent, in the middle of the last century, suffered much from Scurvy. They had eaten no salt provisions, but had been served daily with fresh meat and bread.[3]

At the close of the Punjaub campaign of 1848–49, the troops located in the country suffered from the disease. They had abundance of fresh meat and bread, of excellent quality, but no fresh vegetables.[4] For some years after stations for troops had been found in the Himalayas, fresh vegetables were not procurable in sufficient quantity. Though the soldiers were provided with good fresh meat and bread, Scurvy was not only present, but was attended with its full mortality.[5]

In the second Burmese war, a detachment at Meanday was dieted for several months on fresh beef in unlimited quantities, biscuit, rum, and rice. The men had, however, no fresh vegetables, nor any substitute for them. After three months, Scurvy made its appearance. Lime-juice was procured and issued freely, and the disease rapidly abated.[1]

Dr. Hammond[2] describes having seen many cases of Scurvy among troops who had fresh-meat rations on four days in the week, and game of their own procuring on the others.

The Turks, as noticed above, in the Mingrelian campaign of 1855–56, had no salt meat, and suffered much more severely from Scurvy than did their French and English allies at a time when the latter were dependent upon it for animal food.

So, also, during the previous winter, "They fed on good sound biscuit, boiled rice, fresh meat twice or three times a week; salt meat was unknown amongst them: they were not overworked or idle, and were in excellent spirits at having beaten the enemy; and yet I found on examination that, on an average, three men out of four on duty in the spring of 1855 were more or less afflicted with Scurvy."[3]

In 1836, above one hundred cases of Scurvy occurred in the 75th regiment whilst quartered in Caffreland, at a time when the men had no harassing duties, and were abundantly supplied with rations of good fresh meat, without having had an ounce of salt provisions. They had no vegetables. The Hottentot troops doing duty with them were served with the same rations, but sought out for themselves pumpkins, melons, some indigenous wild fruits, and esculent roots. They entirely escaped an attack, as also did the 27th and 72d European regiments, encamped at a distance of eighteen miles, fed with the same rations, but supplied also, in addition, with vegetables.

Cases of Scurvy occur every year in North Wales, where fresh meat and milk are abundant, but where the cottagers rear little or no garden produce. Not only the inhabitants, but visitors, located in the country for a short time, are known to suffer, we are informed, occasionally from this disease.

We have ourselves met with many cases of Scurvy amongst the poor of London who had eaten no meat at all for several weeks, but had lived on tea, bread, and butter.[4]

---

[1] Histoire de Louis IX. par le Sieur Joinville. Bohn's Antiquarian Library.

[2] Dr. Budd, Library of Medicine, art. "Scurvy."

[3] Philosophical Trans. Sir J. Pringle's Address, 1776.

[4] Med. and Surg. Hist. of the British Army, 1858.

[5] Ibid.

[1] Dr. Crawford, Med. and Surg. Hist. of the British Army, 1858.

[2] Mil. Med. and Surg. Essays, p. 192. Philadelphia, 1864.

[3] Dr. Bird, On Scurvy. Lond. 1858.

[4] See also Dublin Med. Press, vol. xviii. Dr. Bellingham; "Observations on the Scur-

The occurrence of Scurvy so frequently, and in such well-defined form, in cases when salt meat has been absolutely wanting in the dietary, is sufficient to prove that this substance is not a necessary antecedent of the disease, and cannot therefore be properly termed a cause. But it is alleged, sometimes, that it is to the use of salt provisions, combined with the absence of fresh vegetable food, that the disease is owing. If the facts recorded above are insufficient to negative this view, there are two other circumstances which certainly leave the matter in no doubt:—

1st, There is no case of Scurvy on record, as occurring in a person who has been adequately supplied with fresh succulent vegetables of good quality.

2d, The occurrence of Scurvy in persons living upon salt meat may be prevented by the regular administration of fresh vegetables, or the juice of lemon.

In connection with this subject there is an important point to be considered, as regards the relative rapidity with which Scurvy will appear in persons fed upon fresh meat, or salt meat, or who have had no animal food at all. There is great difficulty in arriving at conclusions upon this point. In the crews of vessels, for example, which have quitted a port during the winter, it often happens that a scorbutic taint has been acquired on shore, and before the men were exposed to the limited dietary of ship life. So also on land, before the supply of vegetables is entirely cut off by the failure of a crop, or by the poverty of the patient, a certain amount continues to be taken, although insufficient for the preservation of health. From these and other causes, reliable statistics as to the exact interval which elapses before Scurvy makes its appearance are wanting. The conclusions at which we have arrived, from a careful consideration of recorded outbreaks, as well as from our own personal observation of the disease, are that under a salt-meat diet, Scurvy will appear sooner than when fresh meat has been taken in the ordinary quantity, and that the disease will show itself more rapidly when the patient has been deprived of all animal food than when he has obtained supplies of salt meat. Liebig has shown that the process of salting deprives flesh of a large proportion of its most important constituents, so that the remainder is deficient in nutritive properties, and the altered and hardened character which it acquires renders even such nourishment as it contains difficult of assimilation. As a result, a diet of salt meat represents only a less degree of starvation than the total absence of animal food, and starvation has been agreed universally to intensify Scurvy. Dr. Kane,[1] the Arctic explorer, speaks highly of the improvement in strength which took place amongst his crew when they succeeded in procuring fresh walrus meat, as a substitute for their salt provisions. He arrived at the conclusion that raw walrus meat was powerfully anti-scorbutic; but his descriptions of the sufferings undergone by his party from Scurvy, even when abundantly supplied with this food, fail completely to justify this opinion. Mr. Whymper[2] expresses an equally favorable opinion of walrus and seal meat, but the details which he gives are not sufficiently elaborate for the purpose of scientific discussion. It seems most probable that fresh meat of any kind, although satisfactorily proved by the instances we have recorded to be incapable of preventing Scurvy, will yet, by its powerful nutritive properties, help considerably to sustain animal strength and retard the development of scorbutic symptoms. So a man, deprived of all food, and dying of starvation, will have life considerably prolonged if he can obtain water. Water, however, we know to represent only a portion of the requirements of man, and to be incapable, by itself, of preserving his existence for more than a short period.

From the limited variety of food which so often accompanies conditions in which Scurvy has appeared, such as besieged towns, encamped armies, and on board ships in long sea-voyages, monotony of diet has been frequently urged by writers as an important cause of Scurvy. But probably one of the most monotonous dietaries in the world is that upon which the poor inhabitants of Ireland thrive,[3] and which consists almost entirely of stirabout, milk, and potatoes. So long as they can obtain this food in sufficient amount, Scurvy is unknown; but when the monotony was broken by the failure in the potato crop, the disease appeared extensively.

The relation, indeed, which such an alleged cause, as well as others which have been most insisted upon—namely, cold and damp, idleness, and mental depression—bears to Scurvy, seems to us simply that which obtains generally in modifica-

vy," by Dr. Mertans; Phil. Trans. vol. lxviii.; Lond. Med. Gazette, vol. iv. Dr. Shapter; Lancet, 1851, p. 519, Dr. Boyd.

[1] U. S. Grinnell Expedition. Second Voyage.

[2] Travel and Adventure in Alaska. 1868.

[3] "The general conditions of these populations, although wearing an aspect of great wretchedness to English eyes, is not now unfavorable to health. Throughout the country I found them a fine, well-built, and often athletic race, with children sufficiently fleshy and rosy, and bearing all the marks of health."—Dr. E. Smith, *Sixth Report of the Medical Officer of the Privy Council*, 1863.

tions of health. The fact that these conditions are certainly not essential to its production must exclude them from the category of true causes. That they are frequent concomitants, and hasten as well as intensify the symptoms of the disorder, is just what is seen in many other diseased conditions which, depending upon a special cause, are yet capable of being injuriously influenced by circumstances known to affect the assimilation of food and the consequent nutrition of the animal economy.

SYMPTOMS.—The earliest symptom of Scurvy is a change in the color of the skin, which becomes pale, sallow, or of a greenish tint, according to the variety of the natural complexion. Succeeding, and indeed often contemporary with this, is a peculiar listlessness of mind, an aversion to exercise, and a condition, not so much of anxiety as to the state of health, as of indisposition to take any trouble regarding it. The patient lounges where formerly he has displayed energy in his occupation; he does not care to speak unless addressed. In reply to inquiries he will usually complain of flying pains about the limbs and back, which he generally refers to rheumatism. The change of aspect, where several individuals are exposed to the same circumstances, will be noticed by them of each other, whilst the observer is unconscious that he, too, is presenting the same appearance.[1] Up to a certain period the appetite remains good, and digestion continues tolerably perfect; usually, however, there is some constipation. There is no fever. Sleep is obtained readily enough. It is sometimes described as accompanied by dreams, in which the luxuries of fruits and vegetables are vividly pictured.[2] Gradually petechiæ are observed, especially about the legs and thighs. They are small, of a reddish-brown color, fading away at the edges, and are especially apt to occur at the points where hairs perforate the skin. They are usually not elevated above the surface. Besides this there may be larger maculæ, apparently formed by the convalescence of several petechiæ, of irregular outline, and particularly common about the lowest part of the legs and on the feet. As the disease advances still larger markings will be noticed, so much resembling bruises as often to be mistaken for the results of violence. Like the eruption of variola, they are apt to fix upon weak portions of the frame for their situation, parts where there has been a blow, strain, or other injury some time previously. Accompanying these external signs there is breathlessness, for which the ear applied to the chest fails to discover any adequate cause. The expression of the countenance is dejected, or it wears an aspect of indifference. The lips are pale. By degrees the face assumes a bloated appearance. In some cases, however, about this period, the eye and its surroundings are the only parts exhibiting signs of Scurvy. The appearance presented is then very remarkable. The integument around one or both orbits is puffed up into a bruise-colored swelling. The conjunctiva covering the sclerotic is tumid and of a brilliant red color throughout, "about the eighth of an inch in thickness or elevation above the cornea, leaving the cornea at the bottom of a circular trench or well."[1] There is nothing inflammatory about this condition; it resembles very violent ophthalmia in the color presented, but there is no pain or discharge. We have seen many cases in which this appearance, together with pallor of the complexion and listlessness, constituted the only evidences of Scurvy, and they have generally been of the most serious character, often terminating fatally. The gums so generally present a remarkable alteration in Scurvy that their condition has been often described by writers as a perfect test of its presence or absence. Our own experience does not correspond with this, and other observers have recorded a similar opinion.[2] All the other phenomena may be present and yet the gums continue in an unaltered condition, except that they are paler than ordinarily. But usually, at an early period of the disease, the gums, first of all pale and contracted, begin to show a swelling at their free margins. This gradually increases so that the teeth are encroached upon, and eventually, in some cases, almost disappear from sight in the huge fleshy masses which encompass them. The swollen gums are then spongy, of a dark-red or livid hue, not sensitive to the touch, and disposed to bleed, sometimes slightly, at others profusely, when irritated. Under these circumstances the teeth become loosened in their sockets, and often fall out. There is a sickening fetid odor from the breath. This is only observed as an accompaniment of the swollen state of the gums, and is evidently due to the sloughing which usually occurs in them.

[1] This phenomenon is graphically described by Dr. Kane: U. S. Grinnell Expedition.

[2] U. S. Grinnell Expedition, p. 267.

[1] Bird, On Scurvy, p. 38. See also Dublin Medical Press, vol. xviii., a paper by D. Bellingham.

[2] Dublin Med. Press, Dr. Bellingham; "Scurvy in Exeter," Dr. Shapter, Lond. Med. Gazette, vol. iv.; L'Union Médicale, 1857, p. 419: "Scurvy in the French Army in the Crimea," M. Perrin; Dict. de Médecine, art. "Scorbut" (Paris, 1865), 12th edit.

So severe is the affection of the gums in many cases, that fleshy masses like huge granulations are often seen to protrude between the lips. Chewing is completely impossible, and there is some difficulty even in taking fluid nourishment. It is impossible to describe the fearful appearance presented by the sufferer under these circumstances. His skin harsh, dry, dirty-looking, and discolored with bruise marks, bloated and puffed up in parts by swellings, his whole manner apathetic and helpless, the condition appears to a novice more irremediable than is seen in almost any other disorder. And yet it is remarkable that these cases, where the external manifestations of the disease are so strongly marked, are frequently just those which yield most rapidly and surely to treatment. The change wrought in a few hours by the administration of lemon-juice or vegetables, coupled with general care, is the most extraordinary thing in therapeutics, and of itself furnishes a powerful argument in favor of the cause of Scurvy existing in the absence of such food.

Besides the petechiæ and other larger ecchymoses under the skin which we have described, a most frequent and highly characteristic symptom appears in the occurrence of swellings in the flexures of the joints. A favorite seat of this condition is the ham. The well between the insertions of the flexor muscles is filled up more or less completely by a mass which is hard, but not so unyielding as to be incapable of pitting on pressure. It requires, however, more force to produce this effect than in œdematous swelling, and the impression is retained for a longer period. As this swelling increases, the limb is gradually more and more flexed, doubtless because extension, by stretching the skin over the tumor, is attended with great pain. If the effusion occurs, as it most commonly does, in the lower extremities, the patient is unable to walk. A similar swelling is sometimes noticed at the bend of the elbow, and still more frequently beneath the muscles of the jaw. In the latter position the movements of mastication are very painful. Another common seat of such effusion is under the muscles in front of the tibia, or between the periosteum and that bone where it is subcutaneous. Such tumors have often been mistaken for syphilitic nodes, and mercury has been administered with the result of increasing the scorbutic condition to an alarming extent. It is one of the facts most universally noted, that mercury has a powerfully injurious effect in Scurvy. The skin covering such swellings may retain its color or present an ecchymosed aspect according as the subcutaneous areolar tissue is invaded or not by the effusion.

The breathlessness which we have noted as an early symptom in Scurvy becomes more confirmed as the disease progresses. On auscultation we find the respiratory murmur louder than natural, but otherwise unaffected. It is very frequently accompanied by occasional faintings, especially when the body is made to quit the horizontal posture. These attacks of syncope are highly perilous. It has happened to us on more than one occasion to witness death from this cause. The patient, previously recumbent, has suddenly sat up in bed to receive our visit, and speedily fallen back in a fainting fit, from which he could not be restored. At Trebizond, notwithstanding that great care was used in the transport of sick from the ships to the hospital, many died whilst being carried up. The danger from fainting is well known in the Dreadnought hospital-ship; and Mr. Harry Leach, the resident medical officer, informs us that no scorbutic patient who is severely affected is allowed to walk up the steps, but is carefully hoisted up the ship's side in a recumbent position.

There is an affection of the chest in Scurvy which, especially when the disease occurs during the prevalence of cold and damp, is very apt to be mistaken for pneumonia. Faint rigors, followed by a certain amount of feverishness, and accompanied by lancinating pain in one or both sides, usher in this condition. The pain is felt only in coughing, and a very viscid mucus is expectorated. The dyspnœa increases, and a constriction as though from a cord bound tightly round the chest is described. Although it occasionally happens that these pulmonary symptoms are dependent upon true inflammation, they are much more commonly associated with effusion of sanguineous fluid into the cavity of the pleura, or into the substance of the lung itself, these structures sharing with every other organ in that tendency to effusion which is the dominant feature of Scurvy. When the lung is thus invaded the expectoration after a short time becomes dark and sanious, with all the horrible fetor which is ordinarily associated with gangrene of the lung, but which is here dependent upon decomposition of the bloody fluid poured into the lung substance. There are now cold sweats, increasing dyspnœa and anxiety, a pulse small and frequent, softer than in inflammatory pneumonia,[1] and death takes place. In other cases there is no pain or cough; but the breathing rapidly becomes short and laborious, and death occurs suddenly. Auscultatory signs of mischief in the lungs are usually wanting; but now and then there is localized dulness on percussion,

[1] M. Aug. Haspel, Gazette Médicale. 1859, p. 70.

with bronchial breathing. Or mucous râles are heard; sometimes also gurgling sounds at certain parts of the chest.[1] The symptoms of gangrene of the lung, when it occurs, are indistinguishable from those arising from effusion of fluid which becomes decomposed. The mere occurrence of very fetid and dark sanguinolent sputa is not necessarily an indication of either condition, as its source may exist in the sloughing and bleeding gums. But constant and increasing oppression of the breath, frequent syncope, and great anxiety point unmistakably to pulmonary mischief; and cases in which these occur are amongst the most hopeless which are ever encountered.

Dulness on percussion may sometimes be noted under circumstances when it probably may be correctly referred to sanguineous effusions into the muscles of the chest, and unconnected with lung mischief. The diaphragm also is sometimes invaded by effusion, and great difficulty of respiration may be thus produced. It is not usually practicable to distinguish the dyspnœa arising from this condition from that caused by lesion of the pulmonary substance, but its significance is not so serious, and it will generally subside with rapidity as the scorbutic state is remedied by dietetic treatment.

As regards the digestive system, the tongue is usually clean and moist. The color is sometimes red, at others pale, with a violet tinge. It is often large and flabby, showing the teeth-marks at the edges.[2] In the early stages of the disease there is tendency to constipation. Later there is usually more or less of painless diarrhœa, often sanguineous in appearance, but unaccompanied by the other symptoms characteristic of dysentery. Scurvy, when it occurs in camps, is so frequently, if not constantly, complicated with the dysenteric diarrhœa which commonly prevails under those conditions, that disturbances of the digestive system are amongst the most frequent concomitants of the disorder. It does not appear, however, that apart from the exciting cause, dysentery is to be considered a symptom of the disease. The dejections usually consist of undigested food, with a quantity of colorless fluid somewhat resembling the evacuations of cholera, or they may be accompanied by a considerable flow of dark blood. The slimy, bright, blood-stained, and offensive fæces of dysentery are wanting, unless that disease be present as an accidental complication. A fatal result is not unfrequently due to this exhaustive diarrhœa, the patient becoming worn out by the frequent discharges.

The intellect of patients suffering from Scurvy is usually remarkably free from impairment. The listlessness, however, to which we have referred above, is constantly present, and is sometimes accompanied by great depression of spirits. As a rule, there is complete coherence of ideas, but we have seen cases occasionally in which symptoms of excitement of a maniacal character were present. Ritchie notes this, and describes also tinnitus aurium, muscæ volitantes, vertigo, and deafness as being occasionally complained of.[1]

In confirmed Scurvy the slightest pressure suffices to open the skin and to give rise to an ulcer, whose edges are hard, thick and shining, and the surface fungoid and bleeding. Its tendency is to increase rapidly in size, and to invade the neighboring structures. An intolerably offensive odor is emitted from it. Ulcers, such as these, will often eat their way into the soft tissues with great rapidity, exposing and invading large vascular trunks, from which dangerous hemorrhage may occur. Sometimes the disorganization of the flesh is sufficiently complete to expose the bones and produce caries. The lips and nostrils are occasionally the seat of this ulceration, and the patient then presents a ghastly appearance, much like that of an aggravated case of lupus.[2] The exhaustion attendant upon these spreading ulcers is often fatal. Wounds and even slight scratches become invaded by this process. Its influence in cases of frost-bite is most disastrous.

An affection of the sight, to which the title hemeralopia (sometimes also nyctalopia) has been given, is frequently observed in Scurvy. In some cases, recorded by Dr. Bryson,[3] it was the first symptom of the disease noticed. The patients can distinguish objects well enough during daylight, and even at night can read a book held close to a candle, but the moment they pass from the influence of the light they become absolutely blind, and require to be led about. Mr. G. Lawson informs us that several such cases have fallen under his notice at the Moorfields Hospital, but in none has the ophthalmascope revealed any signs of mischief. The pupils he finds sometimes dilated and sluggish, in other cases natural. The condition rapidly subsides under an anti-scorbutic regimen. Mr. Soelberg Wells

[1] Haspel, op. cit

[2] There was in general some ptyalism, and then the tongue was indented on its sides, and the swelling of the parotids and of the gums gave the patient the look precisely of a mercurialized person."—Ritchie *On Scorbutus*, Edin. Monthly Journal, July, 1847.

[1] Edin. Monthly Journal, July, 1847.

[2] Bird, On Scurvy, p. 9.

[3] Ophthal. Hosp. Reports, July, 1859, p. 40.

has met with the symptom in cases of great depression of the nervous system, after severe illnesses, and in badly-fed and cachectic subjects. It sometimes occurs in prisons. "It ought," he writes us, "to be carefully distinguished from that which depends upon retinitis pigmentosa, in which the ophthalmoscopic appearances are most marked, which is not amenable to treatment, and generally leads, sooner or later, to almost, if not complete, blindness

Diagnosis.—In typical cases of Scurvy, and especially when, from circumstances, the occurrence of the disease is probable, the diagnosis is very easy. The dirty pallor and bloated condition of face, remarkable lethargy and indisposition to exertion, ecchymosed state of the skin generally, with contraction of the limbs from effusions, and spongy bleeding state of the gums, form a combination of symptoms which readily distinguishes Scurvy from every other disease. But in the commencement of an outbreak, in sporadic cases, or when the attack is slight, the nature of the ailment is very often mistaken. The patients who present themselves for medical assistance complain of weakness, of pains in the limbs and back which are almost always referred to rheumatism, or of pain in the stomach. They say nothing probably of the state of the skin covering their legs, or of any sponginess of the gums, so that the condition of these structures is not perceived by the attendant, who applies himself accordingly to the relief of the symptoms detailed to him by the patient, and the true nature of the case is consequently overlooked. The color of the skin (especially when dirty) in ordinary chlorosis, strongly resembles that of a scorbutic patient, and in such a case there is often a dull heaviness of manner which might tend to mislead an observer. In fact, the early stage of Scurvy is really a form of chlorosis, produced, however, by special circumstances, and remediable only by a correction of these. A careful examination of the skin and gums and the history of the illness will serve to distinguish the conditions.

The red and purple spots, livid blotches, and bruise-like stains which occur in purpura, and closely resemble those found in Scurvy, may possibly cause some hesitation in diagnosis. But there is little difficulty in distinguishing the two disorders. In purpura these appearances often present themselves suddenly in a patient previously in fair health. This is never the case in Scurvy. The latter disease is always gradual in its progress, and it will be found, on inquiry, that a period of increasing pallor, debility, and listlessness, preceded the appearance of petechiæ or blotches. The very peculiar dirty pallor of complexion so characteristic of Scurvy, is absent in purpura. So also is the tendency to effusions about the joints, causing contraction of the limbs, and the spongy and bleeding gums. Lastly, the occurrence of purpura is entirely independent of the defect in diet which produces Scurvy, and it is not cured by fresh vegetable juices. There would never indeed be any difficulty in the diagnosis of Scurvy but for its comparative rarity, at least in an advanced form, in civilized life. Under circumstances of privation, as in winter campaigns of armies, Arctic expeditions, and the like, the disease is usually expected and recognized as a matter of course. Not so, however, in ordinary civil practice. Amongst the classes which furnish the out-patients to our hospitals, dispensaries, and unions, pallor and debility are more often present than absent. Even bruise-marks from accidental injuries are not at all uncommon, so that unless the possibility of Scurvy be pretty constantly remembered, and the symptoms be tested by a reference to that disorder, there is great probability of a number of cases being overlooked. There would be less liability to such errors if the alteration of the gums commonly attracted the patient's attention, and was expressly pointed out to the medical attendant. This very rarely happens. In persons of the class referred to the teeth are rarely or never brushed, and the gums, consequently, are often in a more or less unhealthy condition, so that a little additional discomfort in this respect is scarcely regarded. It is considered a trivial matter in comparison with the so-called rheumatic pains from which they suffer, and and for which alone they ask relief. Were scorbutic patients voluntarily to tender a history of all their symptoms, and the dietetic conditions under which they have been living, there could be little chance of any instructed person, even though he had never seen the disease, coming to a wrong conclusion. But this, it may safely be said, never occurs. The complaints made by the patients of debility and pains in the limbs are just those symptoms of the disorder which are the most likely to be referred to other causes, and it is needful, therefore, that the medical attendant should himself institute the inquiries necessary to prove the presence of Scurvy in his patient. This, of course, he fails to do unless an idea of the probable nature of the disease has presented itself to his mind. We are induced to dwell upon this point from a conviction that there are still many members of the profession who, because no patient has ever consulted them for a swollen and bleeding state of the gums, are under the impression that they have never met with a case of Scurvy

—a conclusion, it will be seen, by no means well founded.

PATHOLOGY.—Great obscurity still involves the question of the ultimate cause of Scurvy. That the proximate cause is an alteration in the quality of the blood, induced by the absence from the ingested food of fresh vegetable juices, is sufficiently manifest from the history of the disease. But we are still in the dark upon three points of importance—

1st. What is the essential element contained in fresh vegetable material by the deficiency of which in these cases such remarkable changes are produced?

2d. Is the influence of this element exerted upon the chemical or the physical quality of the blood?

3d. By what physical law does the blood so altered in quality exhibit such changes in its relation to the tissues?

The essential feature of Scurvy, upon which is based nearly the whole series of organic lesions which takes place, is this—that the relation between the blood flowing in the capillaries and the tissues is so altered as to permit of the diversion of some or all of the blood constituents from their natural receptacles into tissues from which they are excluded in a state of health. Beyond this, however, there are the cases of fatal syncope, which do not admit of such an explanation. In such instances it would seem, either that the muscular structure of the heart is so weakened by mal-nutrition as to lose the power of efficient contraction, or, what is still more probable, the phenomenon is due to embolism.

It is impossible to offer any certain solution to the second and the third questions. Owing to the absence of a perfectly satisfactory mode of analysis of the blood, not only in cases of Scurvy, but in a state of health, no theory can be safely based upon such accounts of the alterations manifested by scorbutic blood as have been published. The only positive modification that can be detected in the blood is a very considerable diminution in its density.[1] It is quite conceivable that the exudations of sanguineous fluid may depend upon the increased tendency to exosmose which such an alteration would necessarily produce.

With regard to the first question, we are able to arrive, principally by a process of exclusion, at something which probably approaches the truth. We have seen that Scurvy will occur where there is no deficiency in the albuminous, oleaginous, or saccharine elements of food, but where there is a want of something which fresh vegetables can alone supply. Fresh lemon-juice may be taken as a convenient instance of a material which is able to prevent the development of Scurvy, and to cure it if it has already appeared. Lemon-juice contains free citric acid, mucus, vegetable albumen, and sugar,[1] with small quantities of malic acid, and acid salts, especially of potash.[2] The only ingredients which it is necessary to consider attentively in this analysis are citric and malic acid, and potash. The other substances are found abundantly in food which is not anti-scorbutic.

In 1848 Dr. Garrod brought forward a very ingenious view of the cause of Scurvy, which has attracted much attention.[3] From examinations of food under the use of which Scurvy was capable of occurring, he was led to the conclusions, "that in all scorbutic diets potash exists in much smaller quantities than in those which are capable of maintaining health," and "that all substances proved to act as anti-scorbutics, contain a large amount of potash." It is, then, to the absence from the food of a requisite amount of potash that he attributes the occurrence of Scurvy, and it is to the presence of potash in lemon-juice that he ascribes the anti-scorbutic power of that material. But for his argument to have been complete, the converse of his second proposition should have been true also. It ought to have been shown that all substances largely containing potash are anti-scorbutic. This is not the case. By reference to Dr. Garrod's table of analysis, we find that one ounce of boiled mutton contains 0·637 grain of potash, whilst one fluidounce of lemon-juice contains but little more, 0·846 grain. So that two ounces of boiled mutton added daily to a dietary under which Scurvy is occurring should be even more efficacious than one ounce of lemon-juice. The numerous instances on record (to some of which we have referred) of fatal Scurvy occurring in persons abundantly supplied with fresh meat, and on the other hand the indisputable power of lemon-juice in preventing the disorder, are sufficient to disprove this. Again, in the records of Scurvy disasters on board merchant vessels, pea-soup always figures as a most important part of the dietary of the crew. They have often been driven to subsist almost entirely upon this food, owing to the bad quality of the meat. One ounce of peas, according to Dr. Garrod's table, contains 0·529 grain of potash—a large proportion, considering that in an ounce of white flour only 0·1 grain is found. Yet peas are well known to be utterly useless in the prevention or cure of Scurvy.

[1] MM. Becquerel and Rodier, Lancet, 1847, vol. ii.

[1] Witt. Chem. Soc. Quart. Journal, vii. p. 44.

[2] Garrod, loc. cit.

[3] Edin. Monthly Journal, January, 1848.

The crucial test of administering nitrate of potash to Scurvy patients has been applied, and found wanting.[1] Moreover, Dr. J. O. Grant describes the occurrence of Scurvy amongst the Ottawa "lumberers" living upon pork salted with nitrate of potash. In one shanty he found twenty-five men out of thirty-six attacked with the disease.[2]

We are thus led to the conclusion that it is either to the free organic acids which exist in lemon-juice, or to the acid salts, that the efficacy of this material is owing. Now, the influence of the citric acid of commerce in Scurvy is by no means certainly proved. Statements and opinions on this point are very contradictory, but the bulk of evidence is certainly opposed to the utility of citric, as well as of tartaric and acetic, acid. There seems reason to believe that the bitartrate and citrate of potash have some influence as anti-scorbutics, though their power is certainly far less than that of fresh vegetable juices. It is probable, therefore, that although the organic acids and potash separately do not represent the requisite material, it is to be found in the chemical combination of the acid and base. Very possibly the form in which these salts exist in lemon-juice renders them more easily absorbed and decomposed by the digestive organs than when exhibited separately. The Materia Medica gives numerous analogous examples of the superior efficacy of a medicine in its natural combinations.[3] No artificial imitation of mineral waters is equal to the supply from their natural source. All anti-scorbutic juices contain salts of citric, tartaric, or malic acids, and we have no evidence of any substances which contain these materials in considerable quantity, and are yet deficient in the power of preventing Scurvy. The mode by which they act is still involved in obscurity.

Morbid Anatomy.—The body of a patient who has died of Scurvy is generally emaciated, but this is by no means always the case. Where the diet has been absolutely deficient, or of such a nature that its mastication was almost impossible from the condition of the gums, there is much wasting. But Scurvy, as we have seen, may occur when there has been not only no lack of food, but the nutriment has been of a kind easily taken by the patient, although from its quality it has not been able to prevent the disease. Under such circumstances the general bulk and weight of the body are preserved, whilst the tissues are found to present the appearances characteristic of the disorder.

Externally, the body presents the same general aspect as was observed during life. Decomposition is more than ordinarily rapid. The extremities are usually rigid. Blood is sometimes observed to flow from the mucous passages. Blood, or fibrinous effusion, more or less strongly blood-colored, is found extravasated under the skin, into the subcutaneous areolar tissue, and into the aponeurotic sheaths of the muscles, sometimes bruising and breaking the muscular fibres. The lower extremities, and especially the hams, are generally the most severely affected, but the same condition may be found in the arms, particularly about the bend of the elbow and under the pterygoid muscles of the jaw. These effusions, when they take place, under the periosteum, sometimes lead to death of the bone beneath, and this has not unfrequently occurred in the jaws. Simple serous effusions, besides, depending apparently on the obstructed circulation, occur, especially about the feet and ankles, so as to give a peculiarly clumsy appearance to the lower extremities.

The condition of the brain varies considerably. It is often free from any appearance of lesion. Sometimes there is effusion of serum under the arachnoid and into the ventricles, whilst the vessels on the brain surface are empty, and the general aspect of its substance is pale. In other cases the cerebral vessels are gorged with very dark fluid blood, or coagula; and there may be ecchymoses upon the surface of the brain, and sanguineous effusion into its substance.

Serous fluid, sometimes in large quantities, is frequently found in the pleural cavities. The lungs may be pale, shrunk, and bloodless in appearance, or gorged with serous fluid, and, sometimes, with very dark blood. When grave symptoms of mischief in the chest have presented themselves during life, stains, or violet marblings, like to those on the skin, have been found upon the surface of the lungs after death. On cutting into these they are found to be of varying depth, but usually superficial. Internally, the vesicles and small bronchi contain a muco-sanguinolent product; there is, besides, slight or severe bloody infiltration into the cellular interstices of the pulmonary vesicles, occupying especially the bases of the lungs, and characterized by a red-winey tinge, with impregnation of black blood. In certain cases the lung offers in some points all the characters of the most complete engorgement, loss of elasticity, crepitation and permeability, increase of volume and of weight. These characters,

[1] See Med. Times and Gazette, vol. xx, Dr. Murray; Med. Times, March 23, 1850, Dr. Bryson, R. N.

[2] Med. Times and Gazette, December 26, 1863.

[3] Parkes, quoted by Aitken.

however, are never carried to the extent seen in ordinary pneumonia. It is principally at the diaphragmatic aspect inferiorly and posteriorly that this engorgement is seen; physical laws tending materially to the choice of this site. Little deposits of blood, not coagulated, are found in different parts of the lungs. These are of variable volume, and constitute, in fact, a species of ecchymotic collections, which compress and obliterate little by little the pulmonary vesicles. They form sometimes largish, fluctuating tumors, composed of liquid blood without clots, contained in cavities of irregular form, which are not lined by any membrane. The sudden rupture of such tumors causes considerable hæmoptysis. Occasionally the lung will be found gangrenous; it is then characterized by the usual greenish-gray color of its structure, mixed with darkened fragments, and imbued with air-bubbles and an ichorous bloody liquid, breaking up under slight pressure and emitting a most offensive odor.[1]

An equal uncertainty attends the condition in which the heart may be found. It is sometimes pale and flaccid, with the cavities quite empty. In other cases it is filled with black liquid blood, and its cavities are dilated. Occasionally its lining membrane, as also that of the aorta and pulmonary artery, is stained with a reddish tinge.[2] Black fluid blood is sometimes found in the pericardium. The muscular substance of the heart may be ecchymosed.

The muscular and mucous coats of the stomach and intestines are invaded usually by sanguineous effusions, and the deposits present all that variety of color which is characteristic of bruises in their various stages, varying from a pink to a blackish green tinge. The intestines themselves may contain fluid blood. Dr. Ritchie describes an enlarged condition of the solitary glands in the lower part of the ileum. The mucous surface of the intestine is sometimes abraded, with minute and superficial ulceration, or it may present detached black ulcers of considerable size. There is a tendency to increase of severity in these towards the lower extremity of the bowel.[3]

The liver and spleen are often, but by no means always, enlarged, gorged with dark blood, and their structure softened and friable.

With all the variety which may present itself in the post-mortem appearances, there is one appearance which is constant in all cases of death from Scurvy. In some part or other, sanguineous effusion into the tissues will be discovered. Considering the delicate structure of the brain, it is remarkable that lesions of this organ occur by no means so commonly as in other and less vital parts of the economy.

As regards the nature of the effusions which play so important a part in the fatal results of Scurvy, minute observations would seem to show that they are essentially fibrinous in character, more or less colored by blood corpuscles. They are sometimes gelatinous in consistence, marked with streaks of a pale yellow color, somewhat resembling the fibrinous clots so often seen in the heart. They occasionally exhibit a higher degree of organization. Deposits of this kind occur in the form of layers of from a quarter of a line to a line in thickness, composed, apparently, of fibrin of a bright yellowish-red color, firm and elastic, affording no fluid on pressure; they are, in fact, false membranes, and are quite distinct from the surrounding muscles, to which they adhere. This kind of deposit has been termed "scorbutic formation." The stiffness of the joints, and especially of the knees, appears to be caused by the firm consistence of these effusions.[1] Upon injection, capillary vessels have been discovered, which in their character and mode of distribution are similar to those met with in other recently organized adventitious tissues. It seems most probable that the hypertrophy of the gums proceeds from the deposit of a plastic material. They are firm, and bleed when wounded. The rapid absorption which they undergo upon treatment is inconsistent with the supposition that they are distended with blood, but is explicable upon the view of the fibrinous character of the enlargement.[2]

Prognosis.—When a patient affected with Scurvy is placed under favorable circumstances as regards food, shelter, &c., his recovery may be safely expected, provided that irreparable mischief has not already occurred in organs essential to life. It is remarkable that the severity of the external manifestations of Scurvy is by no means an accurate guide to a safe prognosis. The fact has been noticed by many observers, and we have ourselves repeatedly witnessed it, that when the patient, from the extent to which his skin, muscles, and gums were involved, appeared most severely affected, recovery

[1] Gazette Médicale, 1859, p. 70: M. Haspel. The description of the morbid appearances in the lungs is mainly derived from M. Haspel's minute and graphic account.

[2] Ritchie, loc. cit.

[3] Ibid.

[1] Dr. Himmelstiern, Brit. and Foreign Med. Rev. vol. xx. p. 150.

[2] See also Budd on Scurvy, Lib. of Med. vol. v. p. 86.

would very frequently be much more certain and rapid than in cases in which such serious external signs were absent. Were it not inconsistent with what we know of the pathology of the disease, it would appear that a degree of safety to internal organs was procured by this severe affection of the outside of the body; as though, indeed, the fury of the disorder expended itself on parts of the economy the least essential to life. In general terms the prognosis will be favorable, or the reverse, according to the degree to which such structures as the brain, lungs, and heart have escaped serious lesion, or are manifestly much involved.

THERAPEUTICS. — The treatment of Scurvy almost entirely consists in supplying the patient, in the most easily assimilable form, with that material by the deficiency of which his disorder has been produced. Combined with this there will, of course, be needed such a judicious arrangement of general diet as will most easily contribute to his general nutrition. The choice of this will much depend upon the condition of the patient's gums and digestive organs. It is very important that his diet should be varied as much as possible, consistent with the avoidance of diarrhœa. Fresh lemon-juice, in the form of lemonade, should be administered, as the ordinary drink, *ad libitum*. The existence of diarrhœa should be no reason for withholding this treatment. The looseness of the bowels in Scurvy will be uninfluenced by any medical appliance, so long as the scorbutic condition of the blood remains uncorrected; and the fresh juice of the lemon has been proved to be more easily digested than any other form of vegetable food. Professor Maclean[1] speaks highly of the use of the Bael fruit in the dysentery which is often associated with Scurvy. The fruit contains a large quantity of tannin with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal. Professor Maclean has seen it useful when all other measures had failed. According to circumstances, the food may consist besides of good beef-tea, with eggs beaten up with wine, or, if the patient can bear it, solid fresh meat roasted or boiled, mashed potatoes, cabbage, milk, salad, or sauer-kraut. The diet will require careful observation; but the great general principle is to be borne in mind that the anti-scorbutic principle must be received by the patient in one form or other if his treatment is to be successful. When diarrhœa is persistent, the trisnitrate of bismuth with opium may often be given with great advantage, the use of fresh lemon-juice being, however, continued at the same time. When the gums slough and bleed very much, they should be brushed over daily with solid nitrate of silver. An amount of relief is thus afforded which it is difficult to explain. Our Turkish patients were so alive to this that they used to indicate to us by pantomime their desire to have the application repeated at every visit. In effusions under the periosteum the iodide of potassium has been found very useful in relieving pain and hastening absorption.[1]

For the offensive fetor of the breath, washes of chlorine may be used, and probably Condy's fluid freely diluted would be a good application. For the hard swellings in the hams and legs, friction with warm soapsuds and water several times a day, was employed with most success in Turkish hospitals. Scorbutic ulcers may be dressed with lint steeped in lemon-juice and covered with oil-silk. But all local remedies are but palliatives, and are inert as substitutes for the constitutional treatment of the disorder.

Amongst the vegetables which may be used as preventives of Scurvy are oranges, lemons, limes, cabbage, lettuce, potatoes, onions, mustard and cress, dandelion, sorrel, scurvy-grass, the *Agave americana* (cactus), grapes. An ounce of lemon-juice should be issued daily, when vegetables run short, and, as on board ship, should be begun ten days after the deprivation of vegetables.[2] Potatoes may be conveniently preserved in casks, with the addition of molasses.

Of late years a very ingenious form of preserved vegetables has been prepared by MM. Masson and Chollet, of Paris. It consists of cauliflower, carrot, lettuce, peas, &c., dried and compressed into solid slabs, which are very portable, and keep good for a length of time. The preparation requires soaking for four or five hours in water, before use, and then should be cooked very slowly. The experience of the Crimean and American wars has shown it to be convenient and palatable enough; but as a preventive of Scurvy, far behind the fresh vegetables or lime-juice in efficacy. It may be added to these with advantage, but cannot replace them. Dr. A. Marroin,[3] in his record of Scurvy in the French fleet, says, "Preserved vegetables retard the outbreak of Scurvy: they slacken its march without stopping it altogether, when it has thrown its roots deeply into the economy." They were liberally supplied to the crews of the French ships in the Black Sea during 1855. Yet, in the second quarter of the

[1] Aitken's Science and Practice of Medicine, vol. i. p. 624.

[1] Bird, On Scurvy, p. 16.

[2] Parkes, Practical Hygiene, p. 248.

[3] Histoire Médicale de la Flotte française, p. 104. Paris, 1861.

year, thirty cases of Scurvy are recorded, whilst in the third quarter (the summer month) no less than 531 cases occurred. In October, 427 cases are mentioned. "In November, during the first fifteen days, we numbered more than 1000 scorbutics on the six vessels in Katcha Roads."[1] One vessel alone, the *Friedland*, had no less than 400 cases on board. Some of the ships were then sent down to the Bosphorus, with all the scorbutics on board. On their arrival the mess tables were well supplied with salads, fruits, &c. "The change effected by this was instantaneous. Those who presented symptoms of Scurvy, saw them disappear; those (and they were even still more numerous) who experienced that muscular debility and horror of movement which constitute the warnings of it, were, after a few days, restored to perfect health."

In the Confederate army (Dr. Darby informs us) the yam, which is generally cultivated throughout the south, was found very beneficial. Syrup from the Chinese sugar-cane (sorghum), abundantly manufactured in the last two years of the war, was issued as a ration with decided benefit. When badly made it is liable to fermentation, and deranges the bowels, yet even in this condition it was of great service in scorbutic cases, as was also the extracted juice before being boiled.

Apples certainly possess anti-scorbutic properties, but they are inferior to lemons and oranges. Cider would appear to be deficient in the property. Dr. Boyd[2] relates two cases of Scurvy which occurred in lunatics, who had lived for several weeks exclusively upon bread and cheese, three times daily, with a pint of cider at each meal!

Sauer-kraut has long been recognized as very efficacious. It was employed by Captain Cook with signal advantage.

It is probable that the light French wines possess considerable anti-scorbutic power.

It is not often that the power of milk, as an anti-scorbutic, is able to be tested in adults, but the immunity from Scurvy of infants subsisting entirely upon this food shows that it possesses the property to a very large extent. We have lately had under our observation an adult patient, who, in consequence of organic disease of the pylorus, was unable to take ordinary diet, and who subsisted for seven months upon Oswego boiled with milk, without any symptoms of Scurvy occurring.

As regards the preservation and use of lemon-juice, the following practical suggestions have been recently issued by the Board of Trade for the information of shipowners and shipmasters:—

"Every ship on a long voyage should be supplied with a proper quantity of *lime* or *lemon-juice*.

"The juice having been received in bulk from the vendors, should be examined and analyzed by a competent medical officer. All measures adopted for its preservation are worthless, unless it be clearly ascertained that a pure article has been supplied.

"Ten per cent. of brandy (sp. gr. 930), or of rum (sp. gr. 890) should afterwards be added to it.

"It should be packed in jars or bottles, each containing one gallon or less, covered with a layer of oil, and closely packed and sealed.

"Each man should have at least two ounces (four tablespoonfuls) twice a week, to be increased to an ounce daily if any symptoms of Scurvy manifest themselves.

"The giving out of lime or lemon juice should not be delayed longer than a fortnight after the vessel has put to sea."

[Fresh, or even desiccated, vegetables, as potatoes, onions, &c., have proved, in American army experience, much more efficacious in the prevention of Scurvy than lemon-juice, or even than fresh lemons and oranges.—H.]

[1] Marroin, loc. cit.

[2] Lancet, 1851, vol. i. p. 519.

PURPURA.

By Thomas Hillier, M.D.

Revised by Tilbury Fox, M.D.

The term Purpura is derived from *purpureus*, purple, and, as generally used, it signifies a discoloration of the skin, of small size, due to the occurrence of hemorrhage into its substance, and unaccompanied by any serious disturbance of the general health, or any appreciable organic disease. The hemorrhage into the skin is, in fact, the disease. Sometimes, however, the term is applied to cutaneous hemorrhage occurring in diseases in which the general condition is the more important, as in "purpuric" fever.

The occurrence of hemorrhage in the cutis, in fact, from whatever cause, has been considered sufficient to warrant the use of this term. Cutaneous hemorrhage is often seen in typhus fever, in measles, in smallpox, in scurvy, and as a result of injury, as well as in cases known as Purpura proper. Typhus fever has been described as Purpura contagiosa, but this name is now obsolete; the cutaneous hemorrhage which often accompanies it being properly regarded as a symptom of secondary importance. In scorbutus or scurvy we have another well-defined disease, in which hemorrhage into the skin constitutes an important symptom. Purpura has been, and often is, confounded with scurvy, but should be carefully distinguished from it. With the advance of medical knowledge it is very likely that the cases even now classed together under the name of Purpura, will be further distributed under several distinct categories according to their real pathological character. It might be well to use the term cutaneous hemorrhage for extravasations of blood connected with general diseases, or violence, and to limit the use of the term Purpura to those forms in which the skin alone seems to be at fault.

Willan described four varieties: Purpura simplex, P. hæmorrhagica, P. urticans, and P. senilis, and classed the disease with the exanthemata. Purpura, however, is readily distinguished; the patches are not transitory, and do not enlarge, they undergo absorption, changing color day by day, like a bruise; the color of true exanthems disappears on pressure, the blood still remaining in the vessels, whilst in Purpura the coloring matter, and sometimes the blood-corpuscles, are out of the vessels, and the stain is, therefore, not effaced by pressure.

In the present day, however, Purpura, in its more limited and proper rendering, is commonly described under two main divisions: Purpura simplex, and Purpura hæmorrhagica. In Purpura simplex, hemorrhage is confined to the skin; in Purpura hæmorrhagica blood escapes also from the mucous surfaces, the alimentary, the genito-urinary, and the respiratory.

Purpura urticans is a compound disease, made up of urticaria and Purpura. Purpura senilis is merely the disease Purpura in old people, in which the patches are large.

Symptoms.—On the skin, a number of spots or patches make their appearance; these vary in size from mere points, when they are called *stigmata*, or the size of pins' heads or peas (*petichiæ*), to large patches of considerable dimensions, even several inches in diameter. The term *vibices* is applied to the patches whose length much exceeds their breadth, so that they resemble a bruise left by a whip or stick, and *ecchymosis* is the name employed when the shape is more irregular. The color varies from a bright red, especially at the early stage of the disease, to a violet, deep purple, or blackish tint. When the eruption first appears, the spots generally have an abrupt, well-defined margin; but as they fade, their outline is gradually lost in the surrounding skin. Occasionally in the early stage, if examined with a lens, the outline may be irregular. In some cases, the discoloration of the skin is preceded by subcutaneous swelling and induration. The discolored spots are not themselves raised. Occasionally, there is an escape of blood-stained fluid under the cuticle, giving rise to blebs on the affected patches. Some writers express this fact in other words, by stating that Purpura is complicated with pemphigus. When the hemorrhage infiltrates the skin very closely, the derma is found, on the removal of the cuticle, to be thickened, hard, and of a purple color. It speedily dries, and has lost its sensibility; it is, in fact, dead. Aggravated cases of this kind are of rare occurrence.

Spots of Purpura are distinguished from simple exanthems by not disappearing or fading on pressure. This is a symptom common to all cutaneous hemorrhages. In a few days the eruption of Purpura changes color, passing through various shades of red, orange, and yellow, until it completely disappears. Whilst some spots are fading, fresh ones appear, or successive eruptions may occur, separated by intervals in which no eruption is seen. Together with the cutaneous phenomena, hemorrhages often occur from various mucous surfaces, giving rise to epistaxis, hæmaturia, melæna, hæmatemesis, or hæmoptysis. It might be said that Purpura simplex, when severe, may be accompanied by internal hemorrhage, and then passes into Purpura hæmorrhagica. This will be noticed in speaking of Purpura hæmorrhagica.

Varieties of Purpura.

Purpura Simplex.—Purpura in its mildest form is commonly seen on the legs in the form of small bright-red spots, which make their appearance without any other symptom beyond slight lassitude, and, in some cases, aching of the limbs. The first eruption soon disappears, but is often speedily followed by a second, and this again by a third. Its appearance is favored by standing, and the cure is promoted by rest, and by the use of elastic bandages. An attack of this kind often occurs during the convalescence from rheumatic fever, and in persons whose general health is impaired from any cause. A special variety, Purpura rheumatica, has been indeed named by Schönlein. In this form of the disease there are antecedent pains about the body, especially the knees; the spots are freely and widely developed, and have a border oftentimes of a somewhat bright hue; they are of the size of hemp seed or thereabouts. A fresh crop and fresh pains go together. This variety is observed to occur in the strong; it is most frequent in those between the ages of twenty and thirty; it is never seen in children, and is more frequent in males than females. It is only Purpura occurring in rheumatic subjects.

Purpura senilis is a form of cutaneous hemorrhage especially frequent on the upper extremities of old women whose arms are much exposed to weather and local irritants. The degenerate state of the tissues of the old predispose to its occurrence, and the extravasation of blood often follows more than ordinary exertion or direct injury of the skin, or undue pressure of some kind. Bateman thus describes it: "It appears principally along the outside of the forearm, in successive dark purple blotches of an irregular form and various magnitude. Each of these continues from a week to ten or twelve days, when the extravasated blood is absorbed. A constant series of these ecchymoses had appeared in one case during ten years, and in others for a shorter period, but in all the skin of the arms was left of a brown color." It is not attended with any constitutional disturbance.

In diseases of the heart causing obstruction to the capillary circulation, petechiæ are occasionally seen. Five interesting cases of the kind are to be found in the Trans. Path. Society, vol. xvii. 1860, p. 422, by Dr. Ogle. In one case the purpuric spots vanished whenever the patient kept his bed. If he were allowed to get up and go about, the spots would again appear.

Purpura Hæmorrhagica.—So far as the skin is concerned, the phenomena are the same as in Purpura simplex, except that the morbid appearances are commonly more aggravated. The term "hæmorrhagica" is given when there is, in addition to cutaneous hemorrhage, a flow of blood from the free surface of mucous membranes The most common hemorrhage is epistaxis, which may be slight in amount or very profuse. It occurs alone or in connection with hemorrhage from other mucous surfaces. Hæmatemesis is of less frequent occurrence; it is sometimes very profuse; melæna also occurs from hemorrhage either into the large or small intestines. Bleeding may take place from the gums, but is not nearly so frequent as in scurvy. Besides the blood which escapes from the free surfaces there is sometimes extravasation into the substance of the mucous membranes: this may be often seen on the palate, inside the cheeks, or on the gums. Small blisters containing bloody serum are sometimes found on the tongue and other parts of the mouth; these soon break, and their contents escape.

Hemorrhage into the conjunctiva is not uncommon. Hæmaturia is a frequent symptom; the blood may come from the pelvis of the kidneys, the ureters, or the bladder. Hæmoptysis, dependent on hemorrhage from the bronchi, is not so often met with. Menorrhagia and hemorrhage from the external auditory meatus are occasional symptoms. It is not often that extravasation occurs into the parenchyma of organs; it has, however, been seen in the brain and lungs; and death has resulted from cerebral or pulmonary apoplexy. Blood is sometimes extravasated into the substance of the muscles. Purpura hæmorrhagica, like the simple form, is either ushered in by fever or occurs quite independently of febrile disturbance. It may occur in the midst of apparently good health, or it may be preceded for some weeks by great lassitude,

faintness, and pains in the limbs and joints. It is usually accompanied by a sensation of great debility and depression of spirits. The pulse is generally weak, and may be quickened; it is sometimes, on the contrary, full and not frequent; occasionally it is intermittent. Paroxysms of fever resembling hectic have been sometimes observed. As the disease advances, signs of anæmia manifest themselves, great pallor of the mucous membranes, a venous hum in the neck, tinnitus aurium, and shortness of breath. Œdema of the face and feet may sometimes occur. Some patients are liable to attacks of slight faintness or complete syncope. The digestive functions are in some cases quite unaffected; in others they are more or less disordered. There may be tenderness in the epigastrium, with a sensation of fulness, nausea, and either constipation or diarrhœa. Deep-seated pains in the abdomen, chest, or back are also sometimes present; these are either caused by congestion of the liver, lungs, kidneys or other viscera, or are merely of a neuralgic character. The urine sometimes contains blood; at other times, casts of the tubes and albumen. This condition may first appear and subside with the cutaneous phenomena, or Purpura may occur in the course of chronic Bright's disease, or the renal changes may follow Purpura. The duration of the disease is very various, from a week to several months.

Dr. Graves in his Clinical Medicine (vol. ii. p. 362), describes two cases having some of the characters of Purpura hæmorrhagica, but presenting important points of difference; he proposes for them the name of "Exanthema hæmorrhagicum." There was an eruption of red spots, somewhat resembling that of measles, but without the crescentic outline usually seen in this disease, and more nearly resembling that of typhus. The patients were strong and healthy previous to their attacks. There was but slight febrile excitement, and this only at the onset; the pulse was slow (in one case 70, and in the other 50 in the minute), but it was peculiarly hard and thrilling, almost dicrotous. There was no headache, delirium, or loss of sleep. Bleeding took place from the intestines and urinary organs, from the gums, the nares, stomach, and other mucous surfaces; it gradually became more profuse, resisting all treatment. The tongue was dry and brown in both cases. The exanthems appeared on the skin on the seventh day of illness, in one case, and after seventeen days in another; the spots never became petechial, and disappeared in about five days. Both cases proved fatal in about four weeks. In one case the eruption was ushered in by a tingling sensation, resembling the sting of nettles, and was diffused over the trunk and extremities; in the other, there was no such tingling, and it was limited to the limbs. One man, aged 34, had been a great spirit-drinker, and the symptoms commenced immediately after drinking cold water, whilst in a state of profuse perspiration. The other man, aged 29, was of temperate habits, and no probable cause could be thought of. The cases appear to have been like acute Purpura as regards the mucous membranes, but unlike it in presenting an exanthem but no hemorrhage in the skin. Whilst the pathology of Purpura is so imperfectly understood, these cases may be classed with it; hereafter, they may be more clearly distinguished from it. Dr. Graves states that, if cases of this kind should again come under his notice, he would freely resort to blood-letting, as affording the best chance of cure.

Dr. Lingen (in the British Medical Journal, July 27, 1867, p. 61), refers to a group of cases of "Irish Purpuric disease." A pig was taken ill, it was drowsy, "stiff," and before death exhibited purpuric spots on its belly, face, and nose. A servant girl who attended to the pig, was attacked with febrile symptoms, and was ill a few weeks. The owner of the pig had suffered, and had large purpuric spots on the loins, the thighs, and smaller ones on other parts. The cowman died in three days of a similar attack. "Purples" in pigs are common in America.

Purpura urticans is a complication of abortive urticaria with Purpura simplex. It is characterized by "rounded and reddish elevations of the cuticle, resembling wheals, but which are not accompanied like the wheals of urticaria, by any sensation of itching or tingling." They gradually form and subside within twenty-four or thirty-six hours. They are usually seated on the legs, and are interspersed with petechiæ; they are also seen on the thighs, breast, and arms. Whilst some spots fade, others appear in succession for three or four weeks. This form of disease is not usually attended with fever; it is most common in warm weather; it is often accompanied by œdema, and some stiffness and pain in the legs. It has affinities with erythema tuberculatum.

This variety of Purpura illustrates an important clinical fact, viz., that cutaneous hemorrhage may occur, under certain circumstances, at the seat of ordinary eruptions, accompanied by congestion; the changes in the tissues predisposing to the escape of blood from the vessels. Under these conditions, the eruption which is supplanted by the hemorrhage is abortive. The writer has seen, for instance, a purpuric patch take the place of a cluster of vesicles in zoster. The same thing is seen sometimes in erythema, and in con-

nection with lichen, constituting *lichen lividus*, which Hebra describes under the term *Purpura papulosa*.

A form of Purpura attended with pyrexia and painful œdema of the subcutaneous cellular tissue is sometimes seen in children. In one case reported by MM. Barthez and Rilliet, a boy three years old was suddenly seized whilst in good health with pains in the feet, which soon became swollen. His skin was hot; on the third day there was general anasarca, and large round red patches appeared on the skin. Two days later, the skin of the legs became shining and tender. It was then covered with patches, some red and some yellowish, round, and varying from half a line to five lines in diameter. The red spots were very slightly elevated, surrounded in most cases by a pale rose-colored areola, whilst the centre was of a claret color, and did not disappear on pressure. The child was quite well at the end of a week.

Another case is described by Ollivier (Archives de Médecine, vol. xv. 1867, pp. 206–216). Here the disease was more severe, the ecchymoses being larger and more numerous; enteritis was also present. There were several successive crops of eruption; each one accompanied by the appearance of œdema. These cases are considered by Barthez and Rilliet to be allied to Purpura urticans. No mention is made of the presence or absence of albuminuria in either of the cases. They seem to have been cases of erythema, complicated with extravasation into the cutis.

Causes.—These are not properly understood. Cutaneous hemorrhage may arise under diversified conditions, many of which have been very illogically assigned as causes. Impure air, indigestible or scanty food, and continued fatigue, by impairing the process of sanguification, are supposed to help on the development of the disease. Purpura frequently occurs as a sequela to smallpox, measles, scarlatina, and rheumatic fever. Intemperance appears to have induced it; damp lodgings and miasmatic influence favor the occurrence of hemorrhage, which has also occurred in the course of cirrhosis, acute atrophy, and cancer of the liver, of ague, of Bright's disease, of amyloid disease of the viscera, of syphilis, and of long standing suppuration of bone. Aggravated jaundice is often attended with Purpura. In fact the old writers recognized some connection between it and disease of the liver, in the use of the term *Purpura hepatica*. It has also been seen in persons not exposed to any specially debilitating causes, and apparently in good health. Amenorrhœa has been assigned as a cause; it is more probable that it was a common effect of some unknown cause. Purpura has been known to disappear after the occurrence of profuse menorrhagia. Ricord mentions the case of a syphilitic patient who suffered from Purpura hæmorrhagica, whenever he was treated with iodide of potassium. Virchow observed the same effect from the administration of this drug to a cancerous patient; and other writers have also recorded the occurrence of Purpura in patients when under the influence of the iodide of potassium. The want of vegetables in food, and an excessive quantity of salt meat, have been assigned as causes of Purpura; but this has arisen from confounding Purpura and scurvy. It may occur at any age, but is especially frequent in children and old people. There must be some condition common to these various diseased states leading to perhaps more or less softening of the tissues of the capillaries, and certainly rupture; but before referring to the exact nature of the changes in Purpura, it is necessary to say a few words on the character of those morbid conditions which have been found to accompany cutaneous hemorrhages.

Pathological Anatomy.—In the first place the petechias and ecchymoses in cutaneous hemorrhages are due to extravasation of blood,—not merely of its coloring matter,—into the cutis. There is sometimes hemorrhage into the subcutaneous cellular tissue, and into the muscles. Smaller or larger extravasations are also often found in the mucous membranes of the mouth, pharynx, stomach, small and large intestines. They are less frequently met with on serous surfaces, the pleura, pericardium, and peritoneum. Hemorrhage in the lungs or in the cerebrum is not often seen. Blood is often found in the pelvis of the kidney. The blood in the body is in some cases quite natural, coagulating readily; in other cases it is unusually fluid, and indisposed to coagulate. The liver may be healthy, or the seat of incipient or advanced cirrhosis, of acute atrophy, or of amyloid degeneration. The spleen is either normal or enlarged. Dr. Habershon (Guy's Hospital Reports, Third Series, vol. iii. 1857) describes cases in which the spleen was large, of a red color, studded throughout with pale yellow spots from one to three lines in diameter, which were connected with the capillary circulation, and consisted of cells, nuclei, and granules. The white corpuscles of the blood were not in excess, but rather deficient in number. Dr. Ogle (Path. Soc. Trans. vol. xi. p. 269) describes cases of enlarged spleen containing adventitious material, of which white corpuscles formed a considerable proportion, and alludes

to the circumstance that in several of them there was a tendency to purpuric hemorrhage. The kidneys may be healthy, or the seat of chronic degeneration, either amyloid, or of some other character. The capillaries of the skin have been examined, and pronounced healthy; but this statement must be received with caution, owing to the difficulty of the examination. In the case of Purpura recorded by Dr. William Fox,[1] occurring in a case of secondary syphilis with severe ulceration of the pharynx and larynx, amyloid degeneration was detected in the spleen, kidneys, liver, and intestines; the same change was also observed in the muscles and the capillaries of the skin.

Dr. W. Fox states that "sections of the skin near, but not in the parts affected with hemorrhagic extravasations, gave either with Schultze's solution (chloride of zinc and iodine), or with iodine alone, or iodine and sulphuric acid, a most intense reddish-brown, in portions between the fat, besides corresponding to the course of the capillaries. This coloration did not pass much into the papillæ. The color, with Schultze's reagent, was somewhat evanescent; but that with iodine lasted from forty-eight to seventy-two hours, and in some preparations the marking out of the capillaries was beautifully effected in this manner. This change was not constantly met with in all portions of the skin tested; but was best marked in portions taken in close proximity to the affected spots. In some of these parts, in which I succeeded in isolating portions of the capillaries and smaller arteries, I found that they broke up very easily, that some presented a peculiar glistening, waxy look, while others had a non-granular appearance, in no degree corresponding to the appearances observed in health."

In the same case, there was hemorrhage into many of the muscles, and peculiar appearances, very similar to those described by Zenker, were seen in other parts of the muscle. "Patches of muscle, from a quarter to half an inch in diameter, are whitish gray, contrasting markedly with the normally-colored tissue around. The fibres so affected resemble those of fishes. They are dry, friable, and break with a granular fracture. They have also a strong refraction. These portions of muscles stain of an intense reddish brown, with iodine."

Under the microscope these pale patches presented a mixture of two appearances. (*a*) Some fibres were excessively pale, had a uniform waxy look, and had lost nearly all appearance of striation. Many broke up very easily, and in many places ruptured within the sarcolemma. Some presented an appearance as if made up of innumerable refracting particles, but I could not succeed in breaking up these fibres, so as to examine separately the constitution of these particular particles. These fibres were not distinctly enlarged. (*b*) Other fibres in the same field, much paler than natural had not the glistening, waxy look, but were very finely granular. The transverse and longitudinal striation was indistinct; but these fibres, in many cases, split up very easily into fibrillæ. The granules disappeared for the most part under liquor potassæ or acetic acid, leaving a few scattered fat drops in the fibre. The nuclei appeared about as distinct as usual in the more waxy specimens; they were indistinct, but not enlarged in those which were granular.

"The exceedingly pale spots in the rectus abdominis presented the waxy appearance in the most marked degree. The heart showed, in a few parts, similar waxy fibres, but in the affected spots the more general appearance was that the fibres were finely granular, with indistinctness of the transverse striation. The granular character disappeared as a rule with acids and alkalies; a few fibres here and there were distinctly fatty."[1]

An attempt was made to inject the vessels of the forearm after death; but although great force was used, the injection failed for the most part to reach the smaller vessels. The blood coagulated firmly; at an early stage of the disease, the white corpuscles of the blood were seen to be in excess, whilst at a later period they had decreased in number, and bore relatively to the red an apparently normal proportion.

Dr. Dickinson (Trans. Path. Soc. vol. xvii. p. 13) describes a case of meningeal apoplexy associated with Purpura. In a cachectic patient, sudden effusions of blood occurred in the skin of the arms and thighs, one being the size of an egg. Subsequently the man was seized with pain in the neck, rigors, vomiting, and want of sleep. He was restless and semi-delirious, but not paralytic. He could not speak, and death occurred the third day.

At the post-mortem examination the vessels of the pia mater were very carefully examined under the microscope, and they were found to be apparently healthy; the blood in the body was fluid, and like the juice of cooked cherries or plums. The liver was cirrhotic. No doubt bile products had been plentifully circulating in the blood. This case contrasts with that of Dr. Wilson Fox, in which the capillaries were diseased. The occurrence of Purpura in connection with so-called amyloid deposit has, however, been no-

[1] Brit. and Foreign Med. Chir. Review, October, 1865.

[1] Wilson Fox, loc. cit.

ticed in other cases by Dr. Dickinson. The same gentleman has also (Trans. Path. Soc. vol. xiii. p. 104) recorded a case of Purpura in a girl aged 16, who was jaundiced from the pressure on the hepatic duct by an hydatid cyst, the blood containing a large amount of bile, but in which there was no appreciable lesion of tissue.

NATURE.—Much uncertainty exists as to the exact nature of the disease of which cutaneous hemorrhage is the outward evidence. There must be rupture of the vessels to permit the escape of blood, and the latter does not take place in health, be there ever so much pressure on the capillaries. When hemorrhage does occur, it might—considering the matter *à priori*—be due to a weakened and diseased state of the capillaries, the result of changes in the blood it may be; or to some change in the blood which originates a softening of the tissues outside the vessels as well; or it might be due to a loss of nerve control over the vessels, so that the normal force of the heart's action could not be withstood. Now the blood has been several times analyzed; it has sometimes been found to contain a deficiency of fibrin, sometimes a normal quantity, and sometimes an excess of that ingredient. Becquerel and Rodier have given the name of *scorbutic* condition to all cases in which the fibrin is less than 0·2 per cent. in the blood. In scurvy proper, however, and in Purpura, the fibrin may even be increased, so that a deficiency in fibrin is certainly not the property of the blood on which hemorrhage always depends. Becquerel found in 1000 parts of blood 803·44 water, and 196·56 of solid matter.

In an examination of the blood in two cases of Purpura, by Dr. Parkes, the only remarkable result was an excess of iron with a general deficiency of the solid constituents. The relative proportion of the different organic solid constituents to each other was not much affected. In an analysis by Routier, quoted by Simon (Animal Chemistry, vol. i. p. 319), there was no general deficiency of solids, but a small proportion of fibrin, ·09 per 100. Simon examined the bloody fluid discharged from the mouth of a girl, aged twenty years, suffering from Purpura hæmorrhagica, and found it to contain no fibrin, a few blood-corpuscles, and some bile.

It was long a favorite doctrine, that change in the *quality* of the fibrin, rendering it liable to coagulate in a peculiarly gelatinous manner, existed in cases of Purpura and scurvy. Some modern observers (Magendie, Marchal, and Poggiale) have adopted this view, but it is one to which little importance can be attached; in the first place, because this form of coagulum is often absent, and in the second place, because nothing is known of the change which has taken place in fibrin which does thus coagulate. Deficiency of fibrin might help out the disease.

It has been proved by experiments on animals, conducted by Magendie, Virchow, and others, that the injection of putrid matter into the veins will cause ecchymoses of the endocardium, of the lungs, liver, and kidneys, &c., effusion of blood from the intestinal mucous membrane—this may be by the production of thrombosis. The injection of ammonia, however, into the blood, has also been found to produce hemorrhages. This shows that extravasations may follow changes in the blood, but proves nothing as to the nature of Purpura. Dr. Garrod has suggested that a deficiency of potash in the blood may be the cause of scurvy, and this disease has been cured by the administration of salts of potash; in this respect, Purpura is entirely different from scurvy. Purpura is not cured by the administration of salts of potash or of fresh vegetables.

The occurrence of cutaneous hemorrhage or Purpura in the more general sense of that term, in acute atrophy of the liver, as well as in cirrhosis and other diseases of that organ, has been variously explained by different writers. At one time it has been ascribed to debility of the heart's action, the general want of tone in the system, and the defective nutrition of the bloodvessels (Buhl); at another time, to the deficient formation of fibrin (Monneret); and at another, to an overdistended condition of the bloodvessels in consequence of a deficiency in the secretion of bile. Frerichs suggests that there is an abnormal attraction between the walls of the vessels and the blood which has become altered in its composition, from which arise obstruction and rupture of the capillaries (Clin. Treatise on Diseases of the Liver, vol. i. p. 232, New Sydenham Society). The presence of a large quantity of bile in the blood has been stated to cause a solution of the walls of the blood disks, favoring the transudation of hæmatine into the tissues. It is questionable, however, whether change in the blood alone will account for the escape of blood-corpuscles from the capillaries. If the blood be charged with bile, or morbid products, or be deficient in fibrin, and obstruction occur, then rupture might more readily take place. In Purpura there is probably always an actual escape of the blood disks; it is not merely an extravasation of the coloring matter, owing to a solution of the walls of the disks.

The sympathetic nervous system may

possibly, besides the blood, be concerned in the recurrence of Purpura, for Simon produced cutaneous hemorrhage by destroying several of the great sympathetic ganglia in the neck of the frog; softening of the tissues first occurring.

To explain satisfactorily an escape of blood-globules from the capillaries, one imagines that lesions of the vessels must be assumed to exist. A blood change may be the first morbid condition leading to these lesions. The nature of the changes in the capillaries is not yet ascertained; sometimes it may be simple rupture from over-distension; but more frequently there is probably a degenerative change in the capillaries, making them specially liable to give way as in the case of Purpura hæmorrhagica, above referred to, observed by Dr. Wilson Fox, where the capillaries were in process of lardaceous degeneration.

Dr. W. Fox observes: "Another very important question arises as to how far this affection of the capillaries can be logically considered to have been the cause of the hemorrhage. Any direct association of the two changes will probably be considered doubtful by many who know that lardaceous affections of tissues are rarely associated with hemorrhage; and further, that the change in the parenchyma of organs and in mucous membranes thus affected is often preceded by a similar change in the smaller vessels.[1] The evidence as it stands at present is decidedly against such a theory of causation, unless one or two possible hypotheses may be admitted to explain the connection of the phenomena observed. (1) May this lardaceous degeneration, which we know chiefly as a chronic disease, occur occasionally in a more acute form, and in this manner so rapidly alter the elasticity of the vessels, before their diminished calibre can have retarded the flow of blood in the part, that rupture and hemorrhage ensue? (2) Is it possible that this lardaceous change, occurring only in *tracts* of tissue, may throw such a stress on the collateral capillary circulation of the tissue around, that adjacent but comparatively unaffected capillaries gave way? Both theories derive some support from the observations made on the dissemination of the degeneration in this case, and also from the observations of Zenker, in a similar degeneration of the muscles in typhoid fever."[2] But then we have the testimony of such cases as those of Dr. Dickinson to the contrary, in which the capillaries seemed to be quite healthy.

[1] Virchow, Cellular Pathology. Chance's Translation, p. 374.

[2] Ueber die Veränderungen der willkührlichen Muskeln in Typhus Abdominalis. Leipzig, 1864.

Dr. W. Fox's case of Purpura, though not an uncomplicated one, seeing that the patient was suffering also from an aggravated form of secondary syphilis, must be regarded as a valuable contribution to the pathology of Purpura; it may serve as an indication of the direction in which inquiries as to the nature of the disease should be instituted.

Purpura senilis is probably due to a degeneration of the capillaries, in parts exposed to rough usage; we are not aware that this has been confirmed by actual observation.

The general result at which we arrive as regards cutaneous hemorrhage, then, is this: that it occurs in many diseases in which the blood is disordered. When—this is Frerich's view—in consequence of the altered relation between the blood and the tissues, the former passes with difficulty through the capillaries, and the tissues are more or less badly nourished in common with the whole body, rupture of the capillaries is likely to occur, and especially if there be, in addition, amyloid degeneration of the capillaries or obstruction to the general circulation, as in liver disease. This applies to cutaneous hemorrhages as a whole. In the disease more properly called Purpura, there is no evidence of diseased capillaries, nor of blood-change. Hebra, however, asserts that the capillaries must be weak, and that Purpura is "due to change in the cohesion of the tissues surrounding the ruptured capillaries," as in inflammations. Hebra thinks that temperature and clothing have much to do with the occurrence of Purpura. It still remains a question how far in progress of time a common pathological origin will be assigned to all cutaneous hemorrhages, Purpura included.

Prognosis.—In Purpura simplex recovery usually occurs, but relapses are very frequent. Purpura hæmorrhagica is always a grave disease; it has often proved fatal from exhaustion, or, more rarely, from cerebral or pulmonary apoplexy. Recovery ensues in a certain proportion of cases under judicious treatment. The prognosis of cutaneous hemorrhage is that of the general disease in which it occurs.

Diagnosis.—The characteristic features of Purpura are the extravasation of blood in the cutis, and, in the hemorrhagic variety, loss of blood from other parts. The eruption in the skin is known from other non-hemorrhagic eruptions, by its not disappearing or fading on pressure. *Typhus fever* and *hemorrhagic measles* will be distinguished by the constitutional symptoms and history peculiar to those diseases. In the former, a history of con-

tagion, destitution, or overcrowding, cerebral symptoms, dryness of the tongue, and the fact that the spots are small, generally spread over the body, and at first disappearing on pressure, will prevent any mistake; in typhus, too, there are no hemorrhages from mucous surfaces, unless it be complicated with scurvy. In measles, the rash is brighter, and at first disappears on pressure; there are also signs of coryza and bronchial congestion, preceding the rash for forty-eight hours or more. The only disease with which Purpura is often confounded is *scurvy*. The latter disease is always caused by a want of fresh vegetables; it may be always prevented, and generally cured by a proper supply of vegetables. This is not true of Purpura. In scurvy, the gums are usually swollen, soft, and sore, which is not the case in Purpura. There is a tendency to painful swellings of the limbs, and stiffness of the joints in scurvy, not observed in Purpura. The gums are swollen, spongy, discolored, and bleeding in scurvy. The eruption of Purpura is often bright at the onset, and the lips and tongue are frequently of a good color when the disease sets in. In scurvy, the complexion is pale and sallow, and the patient is obviously out of health before the hemorrhages take place; this is very often not the case in Purpura. Fresh vegetable diet at once prevents the occurrence of hemorrhagic spots, and does not influence Purpura.

Ecchymoses from violence may be known by their fixed local character, their size, and the absence of small petechiæ. Fleabites in unhealthy persons often retain the petechial character for many days, or even weeks; they may be known by their nearly uniform size, and the presence of a central minute point where the skin was punctured by the insect.

The *hemorrhagic diathesis*, in which there is a tendency to excessive loss of blood from very slight causes, must be distinguished from Purpura.

In persons of a hemorrhagic diathesis, there is usually some wound, ulcer, or contusion of the part from which blood escapes; there is not the tendency to general bleeding in all parts of skin and mucous membranes which characterizes Purpura. Signs of the diathesis generally make their appearance at an early age, and there is very often a history in the family of an hereditary tendency to the same condition.

TREATMENT. — According to the different views entertained of the nature of Purpura, different lines of treatment have been recommended and adopted. The cases brought together under the name of Purpura, in its wider signification, being of very varying nature, agreeing only in the circumstance of presenting hemorrhage in the cutis, obviously must not be all treated alike. Regard must be paid to the diathesis of the patient, whether he be rheumatic, gouty, cancerous, or scrofulous. The antecedent conditions must be ascertained — whether his diet has been suitable or deficient in any nutritive elements, whether he have resided in damp, ill-ventilated places, or been exposed to malaria.

At one time, blood-letting was recommended for Purpura, as for nearly every other disease. Its use is now universally discarded. If there be signs of plethora, with a full, strong pulse, and the mucous membranes of a good color, the use of free saline purgatives, preceded by a dose of calomel, is indicated. The purgative may in rare cases be combined with a small quantity of antimony. Cases of this kind are seldom met with in the present day.

If there be any sign of hepatic congestion, the sulphate of magnesia or soda,[1] with dilute sulphuric acid, should be given two or three times a day; a good dose of jalap, with a grain or two of calomel, may be first given. The perchloride of iron has been much extolled in France and elsewhere. M. Pize (Journ. de Méd. et Chir. pratique, August, 1860) regards it as an almost infallible cure administered in quantities of one drachm in twenty-four hours (equal to about four drachms of the tincture of the sesquichloride of iron of the London Pharmacopœia). If iron be given at all, it should be used in full and frequently repeated doses, half a drachm or a drachm of the tincture of steel every two hours. In the ordinary cases of Purpura simplex, the dilute sulphuric acid, with quinine and sulphate of magnesia, may be given.

Dr. Neligan has strongly advocated the use of turpentine in large doses, in Purpura, with or without castor oil. He gave from an ounce to an ounce and a half to adults, and from two to four drachms to children. He reports several cases in which it certainly seems to have exerted a most remarkably beneficial influence.

I have myself administered it with very satisfactory results to two children affected with this disease, one in a very severe form. I have seen it used in two fatal cases in which it appeared to check the hemorrhage, but its use was resorted to at so advanced a period of the disease as not to afford any reasonable hope of preventing death.

In one child, aged 11 years, who had numerous petechiæ and ecchymoses of the skin, with profuse epistaxis, and was losing blood from the kidneys, the bowels,

[1] This salt has been strongly recommended in persons of the hemorrhagic diathesis.

and the stomach, I gave at first two drachms of oil of turpentine, with half an ounce of castor oil, once a day; on this treatment there was slight improvement. She was, at the end of two days, ordered olei terebinthinæ, ʒj; mucilaginis, ʒj; syrup ad ℥ss; ol. menthæ pip. ♏ij twice a day; and an enema consisting of olei ricini, ℥ss; olei terebinthinæ, ℥ss; decocti hordei, Oss, once a day.

In four days a marked improvement had taken place; all hemorrhage had ceased, and the ecchymoses were rapidly disappearing. The turpentine was then given once a day for about a week, at the end of which she was quite well, regaining her color and strength.

Dr. Hardy, of Dublin (Dubl. Hosp. Gazette, 1859), advocates the use of the tincture of larch bark, in doses of about ten or fifteen drops every hour, or less frequently, according to the severity of the case. This remedy, probably, acts in the same way as turpentine; it is said to be an agreeable medicine to take.

When the hemorrhage is very profuse and the patient is exhausted, astringents, such as gallic acid and acetate of lead, are usually resorted to, but they are very often inefficacious.

Mr. Hunt recommends arsenic in Purpura simplex, and in this recommendation he is supported by Dr. Habershon, upon what ground it is difficult to say.

In treating a case of Purpura, regard should always be had to the circumstances of the patient prior to the attack. If the diet have been deficient in quantity or quality, such deficiency should be made up by a well-regulated diet, a due proportion of animal and vegetable food being administered, with a moderate allowance of light stimulant, such as claret, marsala, or some pure wine. If the pulse be full and firm, and the color of the lips good, an emetic of ipecacuanha, or even antimony, at the outset may be a good introduction to the turpentine treatment. Digitalis has been recommended in cases where the pulse is very frequent, but not strong enough to justify blood-letting. This drug has been found useful in menorrhagia and in pulmonary hemorrhage; it may be worth a further trial in Purpura hæmorrhagica.

Local measures may be resorted to where there is great loss of blood from parts within reach. For epistaxis and uterine hemorrhage, injection of cold lotions containing alum, tannin, or acetate of lead, may be used, or plugging may be adopted in extreme cases. In hæmatemesis, ice may be given by the mouth. Purpura of the extremities may often be checked by careful bandaging of the part.

In Purpura simplex, following rheumatism, quinine is often of use.

Hebra advises in all cases that the patient should take *rest*, and it is always desirable to keep the patient, if possible, in the recumbent posture for some days.

In convalescence, the administration of bark, as recommended by Werlhoff, Behrend, and Jeller, who first described the disease, may be had recourse to if there is any symptom of debility.

[CHLOROSIS.

By Henry Hartshorne, M.D.

Synonyms.—Green Sickness; Chloroanæmia; Icterus Albus; Leucopathia; Cachexia Virginum; Morbus Virgineus.

History.—Allusion to Green Sickness was made by very early writers, even as far back as Hippocrates. Sydenham refers briefly to it. Hoffman, in the eighteenth century, gave a very good account of it. Many more recent writers have paid attention to its pathology; especially Blaud, Andral, Becquerel, Rodier, and Trousseau, in France, and Popp, Hirsch, and Virchow, in Germany. All who have dealt with it, however, are not agreed as to its nature; especially in regard to its relations with anæmia. It is said to be a particularly common disease in the West Indies, Algeria, and Turkey in Asia. Huss asserts that it has considerably increased in Sweden during the last half century.

Symptoms. — Occurring at about the age of puberty, in girls, Chlorosis shows itself mainly by changes in the condition of the skin, digestive system, circulation, reproductive apparatus, and nervous system. The complexion becomes pale, with a yellowish or green tinge. Often, a tendency to œdema of the face exists, with bluish or blackish circles around the eyes.

Morbid appetite is a characteristic symptom. The patient has a strange craving for ashes, slate pencils, or chalk; occasionally, instead, for vinegar or other acids. The breath is foul; the bowels are apt to be constipated. Palpitation of the heart is frequent; always the pulse and respiration are accelerated by slight exertion. A murmur, "bruit de diable,"[1] is heard on auscultation, from the base of the heart towards the arteries in the neck. Fainting upon the occasion of any nervous excitement is not unusual.

Amenorrhœa nearly, but not quite, always attends Chlorosis. The disorder most frequently occurs in girls whose menstruation has not yet been established, but is retarded beyond the normal period.

The temperature of the body is commonly, in Chlorosis, lower than natural; particularly in the extremities.

Languor and debility exist, with vertigo, headache, *tinnitus aurium*, lowness of spirits, irritability or capriciousness of temper, and imperfect sleep. Neuralgic or myalgic pain is common, especially in one or both sides, near the margins of the ilia. Tenderness of the spine, in the upper dorsal region, is sometimes observable. Syncope, or even convulsions, may occur from fatigue or excitement.

CAUSATION.—This is hard to trace in many instances. Trousseau and Hammond have reported cases supervening upon fright. Insufficiency of nutrition, of exercise, and of wholesome mental occupation, are probably promotive of it. Whatever much disturbs the nervous system, in a female of slender constitution, about the period of puberty, is likely to produce Chlorosis.

PATHOLOGY.—Formerly it was a current medical opinion that Chlorosis consisted in a sort of fibrinous plethora; and, for the supposed excess of fibrin in the blood, venesection was prescribed. Late writers generally agree that, in most cases, deficiency in the number of red corpuscles exists, without any characteristic change in the amount of fibrin. Virchow holds that the total number of blood corpuscles is lessened in Chlorosis; while in leukæmia, many of the red corpuscles are substituted by white corpuscles, and the whole number is not diminished. Leichtenstein,[2] examining the blood by Vierordt's method of quantitative spectral analysis, asserts that a diminution of hæmoglobin in the blood appears constantly in Chlorosis. This observation confirms the report of several of those who have at different times chemically analyzed chlorotic blood. A deficiency of iron has been reported by Foedisch, Hoefer, Andral and Gavarret, and others. The term *oligochromæmia* has been used by some writers to designate a reduction in the amount of coloring material in the blood; *oligocytæmia* signifying deficiency of the corpuscles.

Yet, upon the testimony of many practitioners, anæmia, in either form, cannot be said to be invariably present in Chlorosis. The term Chloroanæmia is, therefore, regarded, especially by several of the French pathologists, and by Dr. T. G. Thomas, of New York, as inexact. Becquerel and Valleix have considered Chlorosis rather as a *neurosis* of the ganglionic nervous apparatus. Rokitansky, Virchow, and other German authors, have not accepted this view. It has, nevertheless, great probability in its favor. Retarded development, at the period when menstruation ought to be established, may have much to do with its origin in many cases; such organic debility resulting in a ganglionic ataxia or asynergia, involving all the functions of the vegetative, and some of those of animal life.

Virchow has proposed the assertion, or hypothesis, that a congenital imperfection of the vascular system, especially of the aorta and arteries generally, is fundamental at least in cases of obstinate or recurrent Chlorosis.[1] Certainly such a defect of development cannot be supposed to exist in the majority of cases, from which recovery occurs in a few months.

DIAGNOSIS.—Accepting the view as correct, that Chlorosis is not a mere variety of anæmia, but a distinct cachectic disease, we may follow Dr. T. G. Thomas's[2] enumeration of the diagnostic differences between these two affections. These are comprised in the statements that "Chlorosis is a disease of the nervous system, and may occur with or without its most common symptom, anæmia. Chlorosis cannot (while anæmia can) usually be accounted for by discovery of special cause. Chlorosis occurs in true type usually to girls about the time of puberty. Is affected favorably only by remedies which act upon the nervous system, as alteratives and tonics. Produces a light-green color, instead of the puffy and pale appearance of simple anæmia. Commonly produces sadness and nervous disquietude. Is constantly accompanied by visceral neuralgia. Iron often fails to benefit;

[1 This term, first applied by Bouillaud, is derived from the French name (diable) for the humming top. Skoda gave to the murmur, for like reason, the name "nonnengeräusch."]

[2 Lancet, April 12, 1879.]

[1 See Immermann on Chlorosis; Ziemssen's Cyclop. of Pract. of Medicine, vol. xvi.]

[2 Diseases of Women, 4th Edition, Phila., 1876, p. 772.]

while, in anæmia, iron always does good. If the supposed cause be removed, the patient will often improve but slowly; the removal of the cause of anæmia being followed commonly by rapid improvement."

Some of these characteristics, and particularly the absence of improvement, in certain cases, under the use of iron and other recuperative remedies, will not distinguish cases of Chlorosis from examples of progressive pernicious anæmia. What needs to be remarked upon this last subject, however, will be reserved for another place.[1]

Prognosis. — Chlorosis does not, of itself, tend towards a fatal termination. Not rarely, however, the congenital defect or early impairment of constitution, which brings on the chlorotic cachexia, may result also in phthisis. The continuance of the symptoms of Chlorosis is usually protracted at least through several months, sometimes for years. Favorable signs are the return of regular menstruation and normal appetite and digestion, tranquil sleep, and a healthy color. Especially, whether as a cause or a consequence, amenorrhœa is the most significant of the usual features of this disease; and its removal almost always heralds approaching recovery.

Treatment.—We have evidently here to deal with—first, a cachexia; and, secondly, certain symptoms and conditions affecting the functions and apparatus both of organic and of animal life.

Anæmia being present in a large majority of cases, the blood-making power must be recuperated. If possible, the cause of its curtailment or depression must be ascertained and removed. Unfavorable mental impressions often have to do with this causation, as grief, anxiety, domestic unhappiness; also, sedentary habits, with deficiency of sunshine, pure atmosphere, and exercise. All that can be done towards improving any of such conditions will promote the convalescence of the chlorotic patient.

Whatever the causation, however, it is certain that hygienic influences will be of the greatest importance in the cure of this cachexia. Change of residence will often do a great deal of good. If the patient has been living in town, let her, if practicable, make a prolonged visit to a healthy locality in the country. Mountain air, in the summer, will agree well with many such patients; *camping out*, in good, cheerful company, the best of all; provided, that is, a sufficiently nutritious supply of food can be obtained. The sea-shore will suit, perhaps, a greater number, with sea bathing. Care must be taken, however, that a chlorotic subject does not remain too long in the surf. A sluggish circulation will not easily react from the depressing influence of cold water. Headache, diarrhœa, &c., may result from a too prolonged cold bath, and the incipient *nisus* of menstruation may thereby be suppressed. From five to fifteen minutes at a time will be sufficient for any invalid or valetudinarian to remain in a cold bath, even at the highest temperature of the water in midsummer at Atlantic City or Cape May.

Diet is of the utmost consequence in Chlorosis. Shall we endeavor to prohibit altogether the indulgence of the morbid cravings of the patient? Trousseau thought otherwise. It will, indeed, be almost impossible, without bodily restraint unsuited to the case, to prevent the taking of indigestible things into the stomach. It is not unreasonable to suppose that some indication of nature exists in the disposition to eat unusual articles. Analogy favors this, in the history of various diseases. Especially is this familiar in regard to the desire for acids on the one hand, and alkaline substances on the other. Most chlorotic patients crave earthy or alkaline materials; a few, vinegar, lemons, &c. Dr. Barnes's suggestion is rational, that the *salts* of the blood may be deficient. If we can interpret, at least approximately, the natural meaning of the extraordinary tastes alluded to, they may aid in guiding our medication of the case, to which attention will be presently given.

Of course a highly nutritious regimen is called for, and, in anæmic cases, one containing a large proportion of animal food. Variety is very desirable. Yet, in the enfeebled condition of the digestive organs, ordinary meals may not be appropriated so fully as to furnish enough for the needs of the system. Then extra portions must be given between meals, in the form of beef-tea, milk, &c. The lack of appreciation of the value of beef-tea, shown of late by some physicians and others, is probably due to its being so often made in a manner which wastes the greater part of its albuminoid material. When prepared by the maceration of chopped beef for a couple of hours in tepid water, then boiling for less than half an hour, and pouring off without filtering or straining, merely removing the scum which rises to the top, and salting it moderately, beef-tea contains all that is nourishing and available in meat.[1] It

[1] See Progressive Pernicious Anæmia, vol. iii.]

[1] More concentrated, and more acceptable to some feeble stomachs, is beef *essence;* made by putting finely chopped beef in a large bottle without water; placing the bottle,

should have a rich brown color, and should be stirred, after standing, before it is taken. The clear liquid from which the finely divided brown portion settles when at rest, is, like Liebig's extract, only the serum, so to speak, of beef-tea; except that, unlike the serum of blood, it contains little or no albumen, only dissolved gelatin and salts. Liebig's view of the real function of his preparation is now familiar to the profession. He never ascribed to it the qualities of a complete food; it is, rather, a subtle nutritive stimulant; often serviceable as such, but requiring other food-stuff to be added to it, to make the complement of needed diet.[1]

Milk should be, in the diet of Chlorosis, a standard article. If slow to be digested, lime-water may be added. There will be no danger of inconvenience if so much as one or two fluidounces of lime-water be added to each pint of milk.

Tea will be better than coffee as an appetizing and refreshing beverage at breakfast, but even tea should be taken only of moderate strength, to avoid increasing nervous excitability. At times of special depression, the exhilarating effect of coffee may be tried; but the tendency to palpitation of the heart in Chlorosis makes it more than usually objectionable in that complaint. Cocoa or chocolate will be innocent and nutritious, if it does not prove too heavy for the stomach.

For the peculiar anorexia (or cachorexia) of Chlorosis, benefit may be expected, as in scurvy, from the moderate use of raw vegetable food. Best of all will be fresh fruits in season; also, celery, lettuce, radishes, &c.; anything, indeed, edible and relishing, which "has never seen the fire." While we do not know what are the chemical elements existing in such articles which are destroyed or altered by cooking, there is no room to doubt their value as a part of every dietary, especially when the appetite is deficient.

Light gymnastics may be resorted to, when practicable, to systematize the development of the muscles, with equable distribution of nervous irritability; besides favoring an improvement in the circulation and secretions. But more spontaneous and varied exercise, as in excursions, riding on horseback, rowing, sailing, &c., will be still more beneficial; giving refreshment to the mind as well as the body. All exertion, however, must be kept in moderation, short of exhausting fatigue. Worst of all, no doubt, for chlorotic patients, will be detention, day after day, within doors; either, with one class, working in the ill ventilated rooms of factories, or, with another, lying on sofas and reading exciting works of fiction, day and night.

Of medicines, in Chlorosis, iron claims the first place. Yet Dr. Barnes is right in urging[1] that it may not be well tolerated at the beginning; even when decided anæmia exists. Headache and a feverish condition may attend its too early or free administration. Where the skin is dry, and especially if it be above the normal temperature, saline medicines should be given at first. Aitken prefers carbonate of potassium; Barnes, the freshly prepared effervescent solution of acetate of ammonium, or *spiritus mindereri*. Either will answer very well; and so will cream of tartar, if the bowels need relaxation. Hyposulphite of sodium is a favorite with some practitioners under similar indications.

Gentian, columbo, or other vegetable bitters may be called for to improve the tone of the stomach. Nothing will be likely to meet this indication better than the compound tincture of gentian, in fluidrachm doses, just before meals.

Coindet has recommended iodine in Chlorosis; Trousseau frequently gave iodide of potassium. Liquor ferri iodidi, in fifteen or twenty-drop doses, will combine the effects of the alterative with the roborant action of iron, so generally required.

Of other chalybeate preparations, the tartrate and citrate of iron seem to be especially appropriate to the treatment of this disorder; and the same may be said, perhaps, of the phosphate. Vallet's pill of the carbonate, however, will do very well. Dialyzed iron has disappointed some, at least, of those who have used it. The tincture of the chloride is rather astringent for most cases of Chlorosis.

Shall we attempt especially to medicate the amenorrhœa? The confidence in emmenagogues, as such, which was once entertained, has latterly very much lost ground. The absence or deficiency of menstruation is, most probably, only the effect, not the cause, of the low state of development, or of sanguification; a symptom of cachexia. Restore the functions of the nutritive apparatus, and enrich the blood, and menstruation will return. No doubt, however, the "vicious circle" of deranged actions will be more easily broken, if we can bring about the ovarian

loosely corked, up to the neck in cold water, which is then brought to the temperature of ebullition, and boiled for two or three hours. This, also, should not be filtered or strained; but merely poured off, skimmed carefully, and salted to taste.]

[1 This has been especially proved by the high nutritive power shown in South America, with animals, to belong to the *residuum* from the manufacture of Liebig's extract.]

[1 Diseases of Women, Philada. edition, p. 193.]

and uterine molimen with normal periodicity. If there is any sign of effort at monthly uterine hyperæmia and flux, it should be encouraged. Warm hip baths near bedtime, and moderate doses of aloes, by the mouth or by enema, will be amongst the most efficient measures for this end. Warner's cordial (aromatic tincture of rhubarb and senna) also is gently laxative, and promotive of determination of blood towards the pelvic organs. Electricity, applied with moderate energy to the abdomen and spine, may promote the establishment or return of menstruation. Where "serous plethora" exists, Aitken advises the abstraction of blood, according to Vogel's plan, in very small amount (one or two ounces) at a time, by venesection, cups to the back, or leeches to the anus or inside of the thighs. In chlorotic cases not anæmic this treatment may do good, when headache coexists with fulness of pulse and other signs of plethora; but such cases are rare. Saline purgatives will most generally do all that is called for in reducing intravascular pressure in Chlorosis. Some physicians employ digitalis, in order to tranquillize the often agitated heart. The palpitation, however, is commonly only a part of the generally unstable equilibrium of the system; to be rectified chiefly by restoring constitutional strength.

For this end, nervine tonics are appropriate, in combination with iron, and, if the stomach bears it, cod-liver oil. Quinine may here serve an excellent purpose; given in merely tonic doses, six or eight grains *per diem*. Extract of nux vomica, or strychnia, may be prescribed in small, tentative doses, watching carefully the effect produced. It will be better, in their use, especially at first, rather to fall short than to exceed, in the impression made by the drug upon the nervous system. With yet greater caution, in cases of a depressed character, phosphorus may be employed; preferably in the form of phosphide of zinc. Arsenic is a favorite medicine in Chlorosis with some practitioners; its use in obstinate cases is certainly altogether justifiable. It will not need, however, that large doses should be given; three or four drops of Fowler's solution of arsenite of potassium two or three times a day will suffice. Even with such doses, in some cases, headache, nausea, diarrhœa, or facial œdema may warn the practitioner to withdraw it, or reduce the amount.

When tardiness of digestion or general debility suggests the administration of alcohol to chlorotic patients, careful directions should be given as to the quantity and frequency of its use. Otherwise, indiscretion on the part of the patient or her friends may easily make way for the habit of excess. Women, and especially girls, when unaccustomed to alcoholic drinks, are generally extremely susceptible to their stimulating effects. For Chlorosis, malt liquors, and especially ale, will usually answer best; a small wineglassful (two fluidounces) of this will be enough, taken at dinner, and, if sleep be uncertain, again on going to bed. With young girls, even a still smaller dose may be better; the test being whether or not it flushes the face, quickens the pulse, or is felt at all in the head. Either of these signs will show that the dose has been too large to do good, and should be reduced.

On the whole, the most important part of the treatment of Chlorosis is, not the employment of medicines, notwithstanding their frequent advantage and importance; but rather that which is hygienic; in diet, clothing, atmosphere, and, perhaps above all, personal surroundings, social circumstances, and mental impressions and influences. The nature and therapeutic indications of the malady may be summed up in the expression, that it is a *neurotic cachexia*.]

RICKETS.

By W. Aitken, M.D.

Definition.—A constitutional disease, characterized by an unhealthy state of the system, which precedes for several weeks or months a peculiar lesion of the bones and of some of the solid visceral organs. The lesion in the bones is characterized by irregularity in their growth, by non-solidification of the growing layers of the bone, by the progressive formation of medullary cavities in the older or more mature bone, thus rendering the bony laminæ thin and brittle (Virchow). There

is generally albuminoid (amyloid ?) degeneration of the spleen and liver.

SYNONYMS.—Morbus Puerilis Anglorum, the Rickets;[1] Fagellum Angliæ seu Tabes Angliæ; Rhachitis;[2] Rachitis;[3] Atrophium infantum Anglicum;[4] Cyrtosis Rachia;[5] Cyrtonosus; Tabes Pituitosa; Morbus Anglicus; Osteomalachia infantum;[6] Tabes Pectorea; Spina Nodosa; Rachitismus; Osteosarcosis; Innutritio Ossium;[7] Osteomalakia;[8] Scrofula Rachitis;[9] Rachite, Rachitisme, Riquets, Noruire; Englische Krankheit, Zweiwuches; Rachitide.

CAUSES.—As with other constitutional diseases so with Rickets, the transmissibility of a defective constitution from parent to offspring is the first link in the chain of causation which brings about the cachexia or unhealthy state of the system peculiar to Rickets. Wiltshire, from personal experience, is of this belief. Herring affirms true Rickets to be, in the highest degree, hereditary; while, according to Schönlein, too early marriages, and, according to Küttner, intermarriages, mainly conduce to its transmission. Sir W. Jenner, on the other hand, knows of no facts to prove that Rickets is hereditary.

The history of the disease, however, appears to show that there are *predisposing causes* derived from the parents or the nurse, which are so capable of influencing the health of the child as to lead in course of time to the establishment of the disease now under consideration. This is quite different from a child inheriting Rickets as a disease which its parents had, and which they transmitted to its constitution.

Of predisposing causes derived from the parents, the influence upon children begotten of bodies exhausted from chronic disease, venereal excesses, or age, has been recognized from very early times. An innate or congenital debility is thus imparted to the infant constitution; and when Rickets becomes a frequent disease (as, at one period, it seems to have been in this country), its occurrence would often seem to be hereditary. Vogel says, the father of a rickety child has acknowledged to him that his system was contaminated by syphilis at the time the child was begotten; and Vogel is also of opinion that the existence of constitutional syphilis in the parent may frequently explain the occurrence of Rickets in the children of the better classes. Moreover, it is an ancient belief which regards Rickets as a degenerate form of syphilis. Nevertheless, Sir W. Jenner is opposed to the belief that Rickets is a form of congenital syphilis, mainly by a consideration of the following facts: "The parent who infects his offspring [with syphilis] has usually contracted syphilis before marriage, and the children first begotten after infection are those who suffer [from inherited syphilis]; while, as a rule, it is only the younger children of the family that suffer from Rickets—the first-born being commonly healthy, though the later born are highly rickety." Sir W. Jenner is also very sceptical as to the influence of the father in affecting the health of the children; but he believes that the health of the mother has a decided influence in the development of Rickets in the child. Of this he is sure, that where the mother is of delicate health—in a state in which anæmia and general want of power form the prominent features, without being the subject of actual disease—that such mothers give birth to children which are often apt to be affected with Rickets to a most decided degree, even although the father is in robust health, and when the hygienic conditions under which the children are placed may be most favorable.

With reference to the development of Rickets after birth, the experience of Sir W. Jenner further leads him to state that it is very common for the first, or the first two or three children, to be free from any signs of Rickets, and yet for every subsequent child to be rickety; and, if a woman bear one rickety child, in the majority of cases all her subsequent offspring will be rickety. This he explains upon the following grounds: "That, among the poor, the parents are generally worse fed, worse clothed, and worse lodged, the larger the number of their children—for the man's wages remaining stationary, the calls on his means are increased. And among the rich and poor alike, the larger the number of children, the more has the mother's constitutional strength been taxed, and the more likely is she to have lost in general power." (Medical Times and Gazette, March, 1860.)

The causes of the peculiar ill-health which precedes the expression of Rickets in the child are the subject of great differences of opinion; but generally it may be stated that whatever external or intrinsic circumstances are favorable to the formation of watery blood (hydræmia) in a child, seem favorable to the development of Rickets. Deficient or improper diet taken daily, impure air constantly breathed, deficient exposure to solar light, want of cleanliness, cold, moisture, want of exercise in the open air, are all, more or less,

---

[1] Whistler. [2] Glisson.
[3] Sauvages, Vogel, Boerhaave, Cullen.
[4] Chuden. [5] Good.
[6] Cumin. [7] Darwin.
[8] Swediaur. [9] Young.

influential in the production alike of Rickets and of other constitutional diseases. But details relative to the specific kind of improper food, although of most interest, are the least known. The influence of improper food has been fully proved by the experiments of Guérin on animals. On the other hand, Dr. W. Cumin, in his article on Rickets in the Cyclopædia of Practical Medicine, observes, that whole broods of young geese and ducks, young pointer and greyhound puppies, and young pigs, have Rickets or the "krinckets," when they have been continually exposed to cold and wet, or have been kept in damp kennels and sties. Again, M. Trousseau states that of a hundred rickety children, ninety-eight were either never suckled at all, or were weaned very early; while the experience of Sir W. Jenner and of Mr. Lonsdale confirms the belief that the improper and unsuitable feeding of children is by far the most efficient cause of Rickets, the nourishment being first of all deficient during the period of infancy, when the child does not get sufficient from the mother, either in the quantity or in the quality of the milk secreted; and Mr. Lonsdale has invariably found that in all rickety children the parents have had little or no milk for their supply, and have been obliged to feed the children either partially or wholly with food other than the milk of the mother. The mothers observe that the children never grow properly from the first, and it is mainly the *improper* nature of the supplementary food "given by hand" which impairs the health of the child. Here the evidence of Sir W. Jenner corroborates and completes the evidence of Mr. Lonsdale. Among the poor, Sir W. Jenner observes that the children are dosed with *improper* food even from their birth; and the common mode of rearing the children of the poor in London is thus described by him:—"For the first two or three days after birth their tender stomachs are deranged by brown sugar and butter, castor-oil and dill-water, gruel and starch-water; as soon as the mother's milk flows they are, when awake, kept constantly at the breast. And well for them if they are not again and again castor-oiled and dill-watered, and even treated with mercurials,—for the poor have learned the omnipotent virtues of gray-powder.

"After the first month, bread and water sweetened with brown sugar is given several times a day, and during the night the child is, when not too soundly asleep, constantly at the breast. As soon as the little ill-used creature can sit erect on its mother's arm, it has at parents' meal-times 'a little of what we have'—meat, potatoes, red herring, fried liver, bacon, pork, and even cheese and beer daily, and cakes, raw fruits, and trash of the most unwholesome quality, as special treats, or as provocatives to eat, when its stomach rejects its ordinary diet. Then, instead of being weaned when from ten to twelve months old, the child is kept at the breast when the milk is worse than useless, to the injury of the mother's health, and to the damage of its after-brothers and sisters, in the hopes that thus keeping it at the breast may retard the next pregnancy. The children are sacrificed that the passions of the parents may not be restrained." (Med. Times and Gazette, May 12, 1860.)

Nevertheless, the causes of Rickets are still extremely obscure, as are the causes of all the constitutional diseases. The extensive exciting causes referred to are common antecedents of many affections, and seem to induce one or other of these diseases, according to circumstances of which, as yet, we know nothing.

The subject requires and merits extensive investigation, for, primarily or secondarily, Rickets causes more deaths than any other disease of childhood; and, looking to what we do know of the unhygienic circumstances under which it has been developed, it is one amongst the most preventible of diseases. Without doubt, Rickets is "the most common, the most important, and, in its effects, the most fatal of diseases which exclusively affect children." (Jenner.)

[This is far from being the case in the United States. The language of Drs. Meigs and Pepper[1] corresponds with the general experience of American practitioners: "Rickets must be a vastly more common affection among the poorer classes in London than among the same classes in our large American cities; nor can we say that our own experience agrees with that of Jenner, who further says that he has 'very often seen it in the children of the wealthy.'" "Owing to the better accommodations and larger size of the houses, the better quality and the greater abundance of the food of the lower classes in American cities as compared with European or English, we escape to a great extent the ravages of this fatal disease." In nine years, ending with 1870, the mortality records of Philadelphia reported but two deaths from Rickets. This is, however, less important as evidence in regard to the existence of the disease, than the general observation of physicians showing its comparative rarity in this country.[2]—H.]

[1 Treatise on Diseases of Children, Phila., p. 633.]

[2 See an elaborate paper upon Rickets, by Dr. J. S. Parry, Amer. Journal of Med. Sciences, April, 1872.]

SYMPTOMS. — The commencement of Rickets is said to have been recognized in the fœtus. Bordenave and Pinel have mentioned such cases. The latter describes the case of a rickety fœtus at the eighth month, in which the distortion of the bones was confined chiefly to the lower limbs. Meyer also more recently records a case of *intra-uterine* rachitis. (Henle and Pfeuffer, Band. vi. § 151, quoted by Wiltshire.) Glisson, Henckel, Klein, Lelletier, and other authors referred to by Dr. Cumin, have also given examples of congenital Rickets.

Sir W. Jenner, however, in his extensive experience has never seen congenital Rickets, but he has often heard the mother say that the rickety deformity of her child had existed from its birth; although he properly attaches no value to such assertions if unsupported by strong confirmatory evidence.

The earliest recognition of the cachexia associated with Rickets has been variously stated, and these statements have no doubt varied with the opportunities of the observers for seeing cases at an early period of the disease. Opinions, therefore, on this point are somewhat conflicting.

According to Jenner, the impairment of the general health (cachexia) rarely becomes apparent before the fourth month, and usually between the fourth and twelfth month. (Med. Times and Gazette, April 28, 1860.)

Rickets, however, is rarely observed before the seventh month (Cumin); "and most commonly does not declare itself until the child first begins his attempts to walk, or until he suffers from the effects of the first dentition." (Cyclop. of Pract. Med., vol. iii. p. 616.)

Dr. Mason Good records that "Rickets seldom appears earlier than the ninth month of infancy, and not often later than the second year." (Study of Medicine, vol. v. p. 346.)

Dr. Copland, who has had considerable experience of the disease, is still less definite. He states that "the complaint has been met with from the earliest months, till approaching puberty; but it is most commonly observed to commence during the first dentition, or from six or seven months to three years of age. (Med. Dict., vol. iii. p. 643.)

Dr. Dunglison believes that children are unquestionably born with a *predisposition* to Rickets, although they rarely exhibit any evidence of the disease till towards the termination of the first year; and at first the progress of the disease is so very slow as almost to be imperceptible. (Practice of Medicine, vol. ii. p. 704.)

All these observers agree, however, that the complaint is one peculiar to infancy; and, as such, it is materially associated with the development of the child—perhaps, the conception of the embryo—at all events, its phenomena are *inbred*, and undoubtedly, therefore, constitutional. Some few cases occur of children born with disproportionate development of the bones, which have led to the belief that Rickets has been observed in the fœtus; but, as Dr. Copland justly observes, "It is doubtful whether or not the imperfect ossification, and consequently softened state of the bones observed congenitally, should be viewed as Rickets; as, in this disease, there is a change in the state of the affected bones, different from a mere delay or simple imperfection of osseous formation. (Loc. cit.)

Out of 346 cases of Rickets observed by M. Guérin there had origin—

Before birth	3	cases.
In the First year	98	"
" Second year	176	"
" Third year	35	"
" Fourth year	19	"
" Fifth year	10	"
" Sixth year	5	"
	346	

Of these, 148 were males, and 198 were females.

From these records, the number of cases happening in the first and second years of life very greatly exceeds those of other periods of life; and in the subsequent years the numbers diminish very rapidly.

It seems to take a considerable length of time before the phenomena characteristic of Rickets become so fully expressed as to leave no room for doubt regarding the nature of the affection. According to Guérin, there is a period of at least six months during which a marked series of deranged actions succeed each other. But while many of these are common to other diseases, there are a few characteristic phenomena which, while they occur in sequence, are so peculiar as to be sufficient to determine the specific characters of the disease now under consideration.

The phenomena and symptoms of Rickets, therefore, may be arranged under four classes, namely:—

First, those which are common to many diseases, and in which there is nothing diagnostic of Rickets—symptoms which might arise from deranged digestion, from improper food, or from tuberculosis; and, as Sir W. Jenner observes, they are symptoms too often referred to "the irritation of teething," or to the so-called "infantile remittent fever." This has sometimes been described as the precursory or incubative stage. (Guérin, Guersent, Copland.)

Second, those which "at once mark the nature of the disease, render the diagnosis

easy, and enable us to predicate that the true affection will show itself."

Third, the stage of characteristic deformity.

The *Fourth* class of phenomena may be of favorable or unfavorable import; and may characterize a period of restoration to health, of irremediable atrophy, or of approaching dissolution. The symptoms peculiar to each of these stages are described from the writings of physicians who have had extensive experience and opportunities of observing this remarkable disease—namely, MM. Guérin and Guersent in France, and Copland and Jenner in this country; the latter of whom has given, from his own observation, the most detailed and original account of this disease that has ever been given by any physician. That account will be found in the pages of the Medical Times and Gazette, vol. i. for 1860.

During the precursory or incubative period, the most ordinary symptoms of impaired general health are those which indicate gastro-intestinal irritation. The bowels are irregular in their action, sometimes confined, or only relaxed, but more commonly there is diarrhœa, with tumidity or enlargement of the abdomen, when emaciation generally becomes more or less obvious.

The stools are usually unhealthy-looking, pale, deficient in bile, or of a dirty-brown or leaden color, and of a most offensive odor. In some respects this odor is peculiar in its resemblance to rotten, half-decayed meat. Appetite is feeble, capricious, or entirely lost; and digestion is difficult. The child becomes dull and languid, sad, or peevish, and adverse to play or to exert itself in any way. There is low, febrile irritation present; the skin is hot, the temper irritable; and, although drowsy, the child sleeps but little. It is thirsty, and will drink large quantities of water; if it has begun to walk, it "is taken off its legs;" it lies about, and is unwilling to play or to be amused, or to indulge in any kind of action, preferring to sit or lie, and appearing to be feeble or indolent, and unable to use exertion of any kind. It complains of pains in the joints or along the bones; the face becomes pale, and the flesh becomes soft and flabby; the pulse becomes quick, and indicates irritability of the heart's action; the superficial veins become large, and the jugular veins especially are much dilated; the hair continues thin upon the scalp, and the fontanelle remains widely open.

But in each or all of these phenomena there is, as yet, nothing diagnostic of Rickets. They may accompany or usher in other maladies. The transition from apparent health to the expression of these phenomena is always gradual, more or less slow, and as Dr. Copland has observed, some other disorder or malady may mark the incubative stage of Rickets, and, overlaying as it were that disease, may render the possibility of diagnosis still more protracted and difficult. He refers especially to bronchitis, hooping-cough, and lobular pneumonia, and thus, instead of beginning abruptly, Rickets may commence and progress most insidiously, so that the mother cannot say when her child first began to suffer, and often the deformity and change in the shape of the bones are the earliest abnormities she observes.

On the other hand, Sir W. Jenner, from very extensive experience, has at last been able to identify three very distinct symptoms as commonly present during the approaching development of Rickets, which at once mark the nature of the disease and which render the diagnosis easy, and enable us to predicate that the bone affection will show itself.

These symptoms ought, therefore, to stand as they here stand—by themselves in the *second* class of phenomena. The most remarkable of these symptoms are as follow:—

(*a*) *Profuse perspiration of the head; or of the head, the neck, and upper part of the chest.* Not uncommonly, says Sir W. Jenner, "it is because this symptom has arrested the mother's attention that she seeks medical aid. She uses the strongest terms to express the amount of perspiration: 'It stands in large drops on his forehead!' 'It runs in streams down his face!' 'His head is all of a reek!' 'The pillow is soaked!' It is especially when the child sleeps that these copious perspirations of the head occur, but they are not infrequent at other times, as when the child is at the breast, or even resting its head on the mother's arm. A little increased exertion or a little increased temperature may induce them at any time." (Med. Times and Gazette, April 28, 1860.) Such perspirations are extremely weakening and colliquative during sleep, and when they occur the superficial veins of the scalp are generally large and full, the jugular veins much dilated, and sometimes the carotid arteries may be felt strongly pulsating. (Copland, Jenner.) While the skin generally may be moist during the day, during the night the abdomen and extremities are usually dry and hot, at the time when the head, face, and neck are bathed in perspiration.

(*b*) The second symptom recorded by Sir W. Jenner as specially indicating the general derangement which is the precursor of the rickety deformity of the bones, is *the desire and the efforts made by the little patient to be cool, particularly at night.* The child kicks the bed-clothes off, or throws its naked legs on the counter-pane. "He is always catching cold, because he

will lie without any clothes at night," is the constant language of the mother in such cases. Sir W. Jenner says he has often gone into the wards of the Hospital for Sick Children, after the children have been some time asleep, and seen the rickety ones lying exposed; he has been assured by the nurses that they had put the bed-clothes over them again and again, to but little purpose; and this even in cold weather, when the other children were well covered. "During this restlessness at night the little patient may be seen frequently to bore its head into the pillow, or to rub the back of the head on the place where it rests. When it wakes in the morning, it will cry if the occiput is pressed, or when the head is lifted up and washed. If the head is examined it will generally be found more or less denuded of hair. The whole skull feels thinner than usual, as if distended like a bladder, or it is flattened behind, at the vertex, and protuberant in front." The cranial bones may now be found "very soft in certain points, yielding elastically, like card-board, beneath pressure, and giving the feeling as if the skull might be bent upwards by the finger-points upon the brain." (Wiltshire.) This rachitic softening of the cranium has been now shown to be extremely common; and, in extreme Rickets, is generally the first manifestation of the disease, so far as the bones are concerned. (Elsäser, Widtman, Wiltshire.)

(c) The third characteristic symptom referred to by Sir W. Jenner is *general tenderness*. "The child cannot be moved without its uttering a cry; pressure on any part of its body is followed by like evidence of suffering." "He is tender all over," says the mother; or, "I can't think what has come to the child; if I do but touch him he cries." The condition contrasts most forcibly with that of a child in health. "A child in health," says Sir W. Jenner, "delights in movements of every kind. It joys to exercise every muscle. Strip a child of a few months old, and see how it throws its limbs in every direction; it will raise its head from the place on which it lies, coil itself round, and grasping a foot with both hands thrust it into its mouth as far as possible, as though the great object of its existence at that moment were to turn itself inside out. The child suffering severely from the general cachexia which precedes and accompanies the progressive stages of the bone-disease in Rickets, ceases its gambols, it lies with outstretched limbs as quietly as possible, for voluntary movements produce pain." But not only is the child unwilling to be moved, it is often in terror lest it may be moved, and it will cry at the approach of those who have been accustomed to dance it, and whose presence was wont to afford the child extreme pleasure. (Stiebel, Jenner.)

Dr. Copland, however, would be still more convinced if a series of symptoms which may be regarded as a fourth set of pathognomonic signs of Rickets, were superadded to the three characteristic sets of phenomena just described.

When with the phenomena already mentioned, the urine is more abundant than in health, and when it deposits a copious calcareous sediment, or abounds in the phosphates, then the early or precursory stage of the complaint may be considered as already present; and it is in this stage especially that the salts are most abundant in the urine.

As the disease progresses, the general appearance of the child becomes more and more significant of the disease. It acquires a peculiarly staid, steady, or sedate aspect. Its natural lively expression is replaced by one of languor, of pensiveness, and of age. Its face grows broad and square; and when the child is placed upright on its mother's arm, it sits (as she says) "all of a heap." Its spine bends, and its muscles are too weak to keep it erect. The head thus comes to sink between the shoulders, and the face turns a little upwards. These symptoms, which Sir W. Jenner has so carefully and minutely detailed, as a painting from the life, are so very rarely correctly interpreted, and their significance so little appreciated, that he has subjoined the following case in illustration of the characteristics of this remarkable disease:—

"A. V., aged 3½, a male. His present ailment commenced about four months since, shortly after a 'severe cold on the chest,' with the following symptoms:—heat of skin, especially at night; thirst; loss of appetite; profuse sweating about the head; extreme tenderness of the whole body, so that he could not be touched without crying from the pain it caused him; relaxed bowels, the stools being, to use the mother's own words, 'stinking,' a 'rotteny smell;' a desire to lie exposed at night—again to use the mother's words, 'even in that bitter cold weather he would never lie covered over; in the previous winter he liked to lie warm.' Although he had long run alone, he was soon 'taken off his legs.'"

Present State.—"Rather thin; muscles very flabby; evident tenderness of head, trunk, and extremities. The muscles seem to partake of the tenderness, and the abdominal muscles are as tender as those of the thighs. Sits in his chair, unwilling to move from morning to night. Cries if his brothers or sisters approach him. Feverish at night; throws his clothes off; sweats over the head profusely; the perspiration is limited to the head; appetite

very small; bowels act once a day, but stools very offensive. Intellect decidedly less acute than that of his brothers and sisters was at the same age. Head large, square. He cut all his teeth long before his illness commenced. Spine curved backwards from about the first dorsal vertebra to the sacrum, and forward from first to last cervical vertebræ. Ribs very soft, so that there is great recession of each rib where it joins the costal cartilage at each inspiration. Physical signs of trifling catarrh. Very little enlargement of the ends of the long bones. No enlargement of glands, liver, or spleen." (Med. Times and Gazette, April 28, 1860, p. 416.)

The next set of phenomena which challenge attention in cases of Rickets are those which are associated with the characteristic deformity of the skeleton, when the consequences of the bone disease are superadded to the general derangement of the system. If the general ill-health be severe enough to attract the notice of parents, it will not seem to have lasted long before the deformities of the bones begin to show themselves; and if the general ill-health be very much expressed, the softening of the bones will be so great as to render the deformities more remarkable than the mere enlargement of the ends of the long bones—usually so characteristic an appearance; and the younger the child the softer usually are the bones. With increasing pallor of the skin and flabbiness of the muscles, the extremities of the long bones (such as those of the ankles and wrists, and the sternal ends of the ribs) indicate, by a swollen and knobby, or double-jointed appearance, that the lesions of the skeleton are advancing so as to cause the deformities characteristic of Rickets. The lowermost ends of the long bones then begin to yield, especially those of the lower limbs; and hence, if the child has been able to walk, it can no longer do so. The bones gradually change their form. Those of the lower extremity become bent, so that the convexity of the curvature is forwards and outwards, and, of course, the concavity inwards and backwards. The femora are sometimes curved forwards, so that the convexity is forwards; and this forward curvature is produced before the child walks, simply by the weight of the legs and feet—the lower portion of the limbs hanging pendent from the knee-joint, as the child sits in its mother's lap or on a chair. The soft femur then yields, so that it is curved forwards. After the child walks, the weight of its body mainly determines the curvature of the thigh-bones; the curvature is then apt to be mainly outwards, and the curve which existed before walking will be exaggerated. In some cases the heads of the femora will be bent at an obtuse or acute angle to the shaft. More often, therefore, like the tibiæ, they are curved so that the convexity is forwards and outwards. When the tibiæ curve before the child walks, it is an exaggeration only of the normal curve of the tibiæ in the young child, and is produced as the child sits cross-legged, leaning on to the floor or bed with the outer malleolus. The bones which, in health, seem to be the strongest, may be bent by the most trifling force; and if this force is constantly applied, so will the bending and deformity become more and more apparent, and the direction of the curves will depend on circumstances which determine the point on which the chief amount of pressure is brought to bear.

If the disease becomes developed during the later periods of childhood, the knees are then generally bent inwards, and the feet thrown outwards. The knees thus press against each other, and the child rests on the inner aspect of the foot; but the continued curvature outwards of both femora and tibiæ may cause the knees to be separated to an unnatural distance. The whole of the lower limbs then form irregular curvatures, with the convexities outwards, and generally greatest at or near the knees. (Copland.)

The spine is so bent that the cervical anterior curve is increased. The face is thus directed upwards, and the head falls backwards; and this deformity becomes the more strongly marked according as the muscular debility is the greater. When this muscular debility is extreme, the head is no longer supported. It therefore falls forwards or backwards as circumstances may determine—waggles about, in fact, not unlike a button loosely attached to a garment.

There are two characteristic curvatures of the spine to be distinguished, according as the child is able or unable to walk. If the child is unable to walk, there is a posterior curvature of the spine, commencing at the first dorsal, and extending to the last lumbar vertebra. If, on the contrary, the child is able to walk, then this posterior curvature is limited to the dorsal region, but is combined with an anterior curvature in the lumbar region. This posterior curvature in the child yet in arms is sometimes so extreme, that Sir W. Jenner has known it to be mistaken for angular curvature; and, as it may be so easily mistaken, he gives the following details as to how the curvature of Rickets may be distinguished from angular curvature:—

"If a child be held by the upper part of its trunk, the weight of the lower limbs will usually remove the rickety curve, and it may certainly be straightened, if the nurse hold the child by the upper part of the trunk, and the phy-

sician raises the lower limb with one hand, and at the same time places the other on the curved spine." (Medical Times, March 17, 1860.)

These curvatures in Rickets are merely exaggerations of the natural curve of the spine, which always more or less exists when a child of three or four months old sits unsupported on the nurse's arm. Lateral curvatures, in the young child, are thus less common than those which are antero-posterior. Their direction in Rickets, however, is mainly determined by the position which may be accidentally assumed by the child; and, as Sir W. Jenner points out, if the child be carried constantly on the left arm, there is a disposition to lateral curvature, and the convexity of the curve will be towards the left. Dr. Copland gives the result of his experience in favor of the curve of the spine being in general outwards; but it is sometimes also lateral, outwards in the back or between the shoulders, where the curvature is also to one side, and to the opposite side in the lumbar region, where also there is sometimes a curvature inwards.

The curvatures of the spine, especially those outwards, are generally associated with a flattening of the ribs laterally. (Dict. of Medicine, vol. iii. p. 644.) The deformity entailed upon the thorax thus comes to be that which is of the greatest interest to the physician, because it is the one which is associated with the greatest distress and impediment to the functions of the heart and lungs. The back is flattened, the ribs being at an acute angle where the dorsal and lateral regions unite. Beneath each axilla there is a large concavity or hollow, instead of the normal rounded form. At this part the lateral diameter of the thorax is the greatest, and the ribs pass forwards and inwards from their angles to the points where they unite with their cartilages, so that on the line of junction of the ribs with their cartilages the lateral diameter of the thorax is the least; the cartilages curving outwards before turning in to unite themselves with the sternum. The sternum is thus thrown forwards, and the antero-posterior diameter of the thorax comes to be abnormally great; the sides of the chest are compressed, the dorsal spine pushed outwards (backwards ?), and the sternum outwards (forwards ?); and the diameter of the chest from left to right being thus diminished, while the antero-posterior diameter is increased, the deformity known as "pigeon breast" is formed. (Jenner, Copland.) The general aspect of the thorax is also otherwise changed, so that grooves are formed on each side of the sternum, where the ribs and cartilages unite. These grooves (as Sir W. Jenner describes them) pass from above downwards, on the antero-lateral aspect of the chest, and extend from the first to the ninth or tenth rib; and the deepest part of the furrow is just outside the knobs which are formed where the ribs and cartilages unite. The furrow extends further down on the left than on the right side; but it is deeper over the fifth and sixth ribs on the right than on the left side; the heart and the liver respectively supporting, to some extent, their corresponding ribs. The points of maximum recession correspond to the fifth, sixth, and seventh ribs. The chest expands again considerably a little below the level of the nipple, the chest walls being borne outwards by the liver, stomach, and spleen. At each descent of the diaphragm during the act of inspiration, the ribs recede where they are softest, at the part where ossification is deficient, and the furrows on the chest, described by Sir W. Jenner, are the consequence; and, just in proportion as the ends of the ribs are forced inwards, so is the sternum carried outwards. It is also a characteristic sign of the deformity in the rickety thorax, as Sir W. Jenner has pointed out, that the line of recession corresponds to the upper margin of the liver, spleen, and stomach, these organs preventing recession during the act of inspiration. The precordial region thus also apparently bulges, and the chest-walls covering the heart do not recede so much as on the opposite side. Therefore, the left side appears much fuller than the right, and the precordial region appears abnormally full. (Jenner.)

The curvatures of the humerus, clavicles, ulna, and radius, require some notice. Although the bones of the upper extremity are said to be much less frequently curved than those of the lower, still the humerus is sometimes bent at an angle just where the deltoid is inserted; simply in consequence of the weight of the arms bending the softened bone when the limb is raised by the action of the deltoid. The curvature of the humerus is still further increased by the efforts which the child makes to support itself by the aid of its arms while it sits. It rests on the open palms of its hands, and thus throws a large share of its weight off the trunk on to the bones of the arm and the forearm. Thus also the radius and ulna come to be *twisted* by pronation of the palms as well as curved outwards.

After the bones of the lower extremities, the clavicles are the bones next frequently deformed. They are the subject of extreme angular curvature, mainly at two places. The greater curvature is always at the spot just outside the part where the *sterno-cleidomastoideus* and the *pectoralis major* muscles are attached. The lesser curvature is about half an inch

from the acromio-clavicular articulation. The first curve is forwards and somewhat upwards; the second curve is backwards. These curves are produced partly by the weight of the arms on the humeral end of the clavicle—the sternal end being supported by the muscles just mentioned, and by its ligaments. But the main agent in effecting the curves of the clavicle is the force which bears upon it when the weight of the trunk is thrown on the upper extremities, as the child sits with the palms of its hands resting on the ground; or as it crawls about on the floor. (Jenner.) Many of these deformities are thus traceable to the manner of carrying or placing the child, or to the weight of its body, acting on different parts of the softened skeleton, when the child attempts to stand, walk, or sit, or when it crawls about on its hands and knees. Some deformities, again, like those of the thorax, are mainly influenced by the acts of respiration and atmospheric pressure, counterbalanced by the pressure outwards of the intra-thoracic and abdominal solid viscera. (Jenner.)

The flat bones, like those of the head and the growth of the teeth, the scapula, and the pelvic bones, are also more or less affected, and give rise to characteristic deformity in Rickets.

The deformities of the head in Rickets are thus distinguished by Sir W. Jenner:—

"1st. By the length of time the anterior fontanelle remains open. In the healthy child, it closes completely before the expiration of the second year. In the rickety child, it is often widely open at that period.

"2d. By thickening of the bones. This is usually most perceptible just outside the sutures—the situation of the sutures being indicated by deep furrows.

"3d. By the relative length of the antero-posterior diameter of the head.

"4th. By the height, squareness, and projection of the forehead.

"The first two of these peculiarities of the rickety head are the result of the affection of the bones: the last two are due chiefly to disease of the cerebrum."

As a whole, the head of the child in Rickets is generally unusually large, the vertex flattened, and the forehead prominent, broad, and square, with considerable expansion at the centres of the parietal bones. The sutures also are sometimes expanded, or they remain open. Th forehead, however, seems to project more than it really does, in consequence of the arrest of growth of the bones of the face and expansion of the frontal sinuses or of the nasal and ethmoidal cavities. The bones of the upper jaw, and the malar bones, are also arrested in their growth, while the under jaw appears elongated.

The process of dentition is invariably arrested or delayed in rickety children; and if the teeth have formed, they soon decay; or they early fall from their sockets. Sir W. Jenner has seen the incisor teeth fall from the jaws before the second molars of the first set had made their way through the gums. So important is the knowledge to be got from the progress of dentition, that Sir W. Jenner lays down the following rule of practice, namely:—

"If a child pass over the ninth month without teeth, you should carefully inquire for the cause. It may be that an acute illness has retarded dentition. It may be (but this is very rare) that there is some condition of the gum which interferes with the advance of the teeth. It may be (and this is infinitely the most common cause of late dentition) that the child is rickety; fail not then, when called to a child in whom the teeth are late in appearing, to look if it be rickety, for if you do fail to look for Rickets, you will most likely attribute to the irritation of teething symptoms which are the consequence of the rickety diathesis—the late dentition in Rickets being in itself merely a symptom of the general disorder. The rickety deformities may be very trifling, and yet the teeth considerably retarded in their development." (Medical Times and Gazette, April 7, 1860.)

The scapulæ are in a few instances so deformed as to embarrass more or less the movements of the shoulder. (Copland.)

With regard to the pelvis, its form varies in rickety children according to the direction in which its component bones are compressed by the spine and superincumbent parts on the one side, and the heads of the thigh bones on the other. The direction of the forces which are thus constantly influencing the form of the pelvis and shape of its component bones, vary as the child is the greater part of its time lying, sitting, crawling on all-fours, walking, or shuffling along on the floor. On the other hand, the form of the pelvis varies also according to the age of the child when the compressing forces are brought to bear on the walls of the pelvis, and the consequent differences in the degree of ossification of the pelvic bones—the cartilages being less yielding than the bones. The rickety pelvis is therefore extremely variable as to shape, and is much more frequently triangular than oval. (Jenner.)

Next to the deformity of the thorax, that of the pelvis comes to be of the greatest practical importance, especially in the after-life of the female, relative to the functions of generation.

The sacrum and pubis may be carried either backwards or forwards; the ilia may be directed inwards, or otherwise altered; the lower part of the sacrum may

be pushed upwards; and the outlet of the pelvis may thus be variously altered in form, and diminished in its diameters.

The progress of deformity, in relation to the order in which the individual bones are affected, has been attempted to be laid down by Guérin; but when it is understood that Rickets is a constitutional disease, and therefore one affecting the whole system generally, it will readily be seen that the bones are affected as one organ, and are variously deformed according to the predominating influence of the circumstances already mentioned. No one bone, therefore, is ever affected without all of them suffering; and the deformities may manifest themselves by enlargement of the ends of the bones, or by softening of the bones, or both, in a variable degree. If a child is the subject of Rickets before it walks, the ribs, clavicles, and upper extremities become deformed; while the tibiæ escape bowing, unless the child sits so as to press upon them. (Jenner.)

In a child suffering from Rickets, the large size of the wrists is generally the first deformity which attracts attention. The costal ends of the ribs, the malleoli at the ankles, the olecranon process of the ulna, and the ends of the long bones generally, are all similarly enlarged. This enlargement of the ends of the bones, and the softening, however, do not progress in an equal degree, either condition being often out of proportion to the severity or progress of the other; and Sir W. Jenner has also observed, that it is not uncommon to see the thoracic deformity lessen at the time the legs are bending—a circumstance which he considers due to the disease having greatly diminished, and the muscular power having increased, so as to permit of the child walking before the bones of the leg are strong enough to bear the weight of the body. (Med. Times, April 7, 1860.)

The extreme deformity which results from Rickets is not only very common among the poor, but it is not so uncommon as has been supposed among the rich. (Jenner.) "All degrees of softening of the bones may be seen, from that in which the ribs only yield to extraordinary forces (as during bronchitis, and then only sufficiently to flatten the antero-lateral aspect of the chest), to that in which the ribs yield at every inspiration; all degrees of enlargement of the ends of the ribs and of the long bones—from that where one might maintain that the enlargement was only that proper to the child, to that in which the projections on the anterior wall of the thorax and the enlargement of the wrist would strike the most careless observer." (Jenner, loc. cit.) The consequences of the bone-disease are often attended with extreme distress to the child, and are thus described in the eloquent word-painting of Sir W. Jenner: "It is strange to see a little child sitting placidly on the bed, without moving for hours together—its legs placed so as to escape pressure, its spine bowed, its head thrown backward, the chief weight of its body cast on its arms; and to know that, notwithstanding the apparent calm, the tiny thing is indeed fighting the battle of life: for it is striving with all the energy it has to keep in constant action every one of its muscles of inspiration—endeavoring so to supply the mechanical defects of its respiratory apparatus, due to the softening of the ribs. It wants no toys. It is the best of children if you only leave it alone; move it, and you inflict pain on its tender frame; show it the horse or the doll that was once its delight, and it turns away its head or stares vacantly: to notice would divert its attention too much from the performance of those respiratory movements which are essential to its existence." (Med. Times and Gazette, 1860, p. 416.)

Amongst the phenomena which are significant of Rickets, the condition of the intellect has been generally described as precocious. Both Copland and Jenner, however, agree that such is not always the case. On the contrary, the child continues dull, taciturn, or stupid, or even idiotic; and, in children who are the subjects of extreme Rickets, intellectual capacity and power are always deficient; and the mental, like the muscular power, retrogrades as the constitutional ill health of Rickets continues to progress. The error regarding precocity of intellect is variously explained away. Thus, on the one hand, it has been believed that the openness of the sutures—by allowing the circulation within the cranium, and the development of the brain, to advance unimpeded, and even at an increased rate—has permitted the faculties of the mind to expand unduly; whilst on the other hand, the closing of the sutures, and the consequent unyielding state of the cranial bones, has been thought to confine and embarrass the growth and functions of the brain, and so occasion deficiency of the intellect rather than precocity. But again, according to the experience of Dr. Copland, precocity of intellect has not always been found to exist in Rickets in connection with openness of the sutures, nor has stupidity been concurrent with their closure. Sir W. Jenner's explanation is, perhaps, the more satisfactory. Speaking generally, he believes that the mother's opinion must be well weighed before it is received as correct. She is apt to believe her child is very clever—quite a prodigy—when it is only a few degrees removed from an idiot. "The rickety child, separated, in consequence of its

physical defects, from other children, and thrown necessarily much into the society of adults, catches their tricks of expression, their phrases, and even perhaps some of their ideas: and hence it is thought, by the mother especially, to have a larger intellect than other children." (Med. Times and Gazette, loc. cit.)

General Symptoms of Rickets during the Progress of the Bone Deformity. — The morbid conditions which existed during the developmental stage of the disease continue throughout the period when the bones soften and the body becomes deformed. The abdomen continues tumid, sometimes increases in size, and is often tympanitic. Emaciation progresses; the muscles of the limbs become flabby; they lose their power; they waste and diminish in bulk and volume. This loss of power, however, is infinitely greater than can be accounted for by the mere diminution in the size of the muscles. In proof of this Sir W. Jenner instances the case of a girl, six years of age, who was brought to the Hospital for Sick Children, in whom the loss of muscular power was so extreme, that she was not only unable to stand, but even unable to support herself in the least possible degree. She lay across the arms of the person who carried her like a large half-stuffed rag doll. When placed in bed, she was incapable of changing her position without assistance; nay, she could not raise her arm an inch from the bed. Even long aftewards, when greatly improved, she could not feed herself, and had to be tied in a chair with her head placed on a pillow at its back. If her head fell forward, the nurse had to raise it; for, unaided, she could not lift her chin from her breast. This child recovered so much as to walk about without assistance; but after her return home she unfortunately fell down stairs and was killed by the fall. (Med. Times and Gazette, 1860, vol. i. p. 416.) Although such is an extreme instance of muscular debility during the acute stage of Rickets, yet it is very common, as Sir W. Jenner observes, to see children of two, three, or even four years of age, who are quite unable to support themselves in an erect position; and if a child has commenced to walk before it has become the subject of extreme Rickets, it loses the power of walking. The child continues to become more and more languid and weak. The perspirations continue free, and are readily excited to increased flow on the least attempt merely at exertion. The thirst increases; and although the appetite is often good, the bowels are deranged, irregular, and the stools are often loose, pale or white, and devoid of healthy bile. They are fetid, and the food is often passed as it is eaten. The pulse becomes quick, small, and weak, so that slight hectic symptoms are developed, and pains are complained of in all the bones and joints. There is complete cessation of growth, particularly of the bones. Sickness and emaciation increase, and the child appears to suffer pains except when lying on the back quite still. This stage of softening of the bones and progressive deformity may last for two or three months; and under unfavorable circumstances, when the disease is neglected, it may continue for years, the deformity slowly increasing or remaining in abeyance.

The fourth class of phenomena which may be recognized in the history of cases of Rickets embraces symptoms which may be of favorable or of unfavorable import. They are phenomena which characterize a period of restoration to health, of irremediable atrophy, or of approaching dissolution.

During this period various intercurrent affections are apt to supervene when the disease does not tend towards recovery. Under such circumstances emaciation progresses, the abdomen becomes more distended and tumid, and the bowels more disordered. The softening and deformity of the bones continue to increase, and eventually some visceral affection of the thorax or of the abdomen, or of both, terminates existence. Death occurs in Rickets mainly under one or more of the following conditions:—

(1) Intensity of the general cachexia, which, however, rarely proves fatal directly, death being in general due to one or more of the following morbid states:—

(2) Catarrh, with general congestion of the lungs, general bronchitis, or effusion into the pleura. Mechanical difficulties to respiration contribute greatly to the danger of death from these affections. "The softening of the ribs renders the mechanical power by which inspiration is performed so defective, that the impediment offered to the entrance of air by the mucus in the bronchial tubes cannot be overcome, and collapse of large portions of the lungs follows." (Jenner.)

(3) Gastro-intestinal irritation, enlargement of the mesenteric glands of the spleen and of the lymphatic glands. The enlargement of these glands is generally the result of albuminoid (amyloid?) infiltration or degeneration; and the cachexia which is most significant of these affections is characterized by extreme emaciation and pallor of the surface of the body. Anæmia is extreme. Serum is often effused into the subcutaneous areolar tissue, and the surface of the skin has that "peculiar transparent, waxy, greenish-yellow tint which is sometimes seen in the anæmia of young women." Sometimes under these circumstances there is general ana-

sarca—the face as well as the extremities, the hands as well as the feet, being œdematous. The deformity of the bones in such cases may be either extreme or but very moderately expressed, and may, according to the experience of Sir W. Jenner, either precede or follow the infiltration of the organs. The lymphatic glands, when so affected, vary from a small size to the size of a pea, are not tender, and never inflamed. They may be felt in the groins, in the axilla, and in the neck, rolling under the finger, and free from adhesions to the surrounding tissue. They are hard to the touch, and of a globate form. The spleen is usually at the same time, with the glands, the seat of albuminoid infiltration. As its enlargement, according to the experience of Sir W. Jenner, is often overlooked, he has given the following directions for the examination of children in all obscure cases of this description:—"In every obscure case of early childhood the *absence* of enlargement of the spleen should be established. If we place the fingers of the right hand directly under the left twelfth rib, just outside the mass of the lumbar muscles, and the fingers of the left hand a little to the left of the middle line, in front and half-way between the umbilicus and the ensiform cartilage, and then press the parts forward with the right hand, and backwards and to the left with the left hand—the enlarged spleen may always be readily felt in the left hypochondriac region. We know the hard mass we feel to be the spleen, by the sharpness of its anterior margin, by the anterior margin passing from under the cartilage of the eighth, ninth, or tenth ribs obliquely downwards and inwards towards the median line. The obliquity of this line is such that usually, if continued downwards, it would cross the median line about half-way between the umbilicus and the symphysis pubis; the anterior edge is usually nearer the middle line in front in the child than it is in the adult, because in the child there is a fold of peritoneum, not usually, if at all, described in English books on anatomy, extending from the left side of the arch of the colon to the left twelfth rib, and over the anterior edge of this the spleen must pass before it can extend low enough to be detected by the hand. This fold of peritoneum causes the enlarged spleen to lie more forward, as well as to have a more oblique position, in the child than in the adult."

The enlarged spleen is still further distinguished by its movability: "If the enlargement be great, and the parietes of the abdomen be thin, the notch in the anterior margin can often be felt." Sir W. Jenner says that the liver is seldom so greatly enlarged but its edge feels harder and sharper to the touch than natural. In all these respects the liver and spleen correspond in their characters to what belongs to the amyloid degeneration of these organs; and, as in that degeneration also, the number of the white corpuscles in the blood is not increased.

(4) Sir W. Jenner has specially called attention to the connection of *laryngismus stridulus* with Rickets; and that while it is an affection so constantly referred to the irritation of teething, it ought rather to be regarded as one expression of the constitutional disease now under consideration. In every case of *laryngismus stridulus* (save two) which has come within the experience of Sir W. Jenner, the child was the subject of Rickets. Dr. Wiltshire, also, in his account of "some cerebral affections of children," says that he has had abundant evidence of Rickets in cases of *craniotabes;* and that in such cases tetaniform convulsions were easily propagated to the respiratory muscles, giving rise to the disorder frequently known under the names of *thymic* or *laryngeal asthma.* Dr. Wiltshire also examined the body of a child who, at the age of six months, died of general convulsions following *laryngismus stridulus. Cranio-tabes* was diagnosed, and Dr. Wiltshire was able to cut off the calvarium with a small pair of scissors as easily as he could cut cardboard. The long bones could be bent like soft wax, and the bones of the skull could be folded on themselves without breaking. From the soft and yielding state of the bones, the brain is liable to pressure and other irritation; hence—

(5) Chronic hydrocephalus may supervene and prove fatal. The effusion may be within the ventricles or between the membranes, and may, or may not, be associated with tubercles on the membranes of the brain. A form of *acute hydrocephalus* has been described as due to Rickets. (Portal and Naumann.)

(6) Convulsions (*eklampsia*) may prove fatal, and nothing be found in the viscera, or within the cranium, to account for death. Hypertrophy of the brain is not uncommon; and it is often strikingly soft and anæmic. (Meric, Wiltshire.)

(7) Persistent diarrhœa may cause death; and in such fatal cases it would be well to examine the mucous surface of the intestines as to whether or not it has undergone amyloid degeneration. When the case is prolonged, and becomes chronic, none of these intercurrent affections proving fatal, the deformity continues with no abatement in the general symptoms; but oftentimes the softness of the bones diminishes, they lose their flexibility, become atrophied, and are readily broken. Deformity still progresses, recovery rarely takes place, the morbid state of the blood

and the general cachexia increase, and death usually follows from some structural changes in the internal organs.

Such are the phenomena of unfavorable import, and which characterize the progressive stage of Rickets, terminating in an irremediable atrophy, or in death; but there is another set of phenomena which characterize a period of restoration, and which are of favorable import.

Such favorable changes are mainly indicated by the condition of the excreta. "The urine assumes a more natural appearance and composition; the stools are more healthy, and colored more deeply by bile; the abdomen appears less tumid and less tympanitic; the pulse is less frequent, and pain in the limbs is not so much complained of. The countenance presents more animation, and the hectic or remittent febrile symptoms, and thirst, subside gradually. The appetite is less capricious, and more natural; and with the continuance of these changes, the flesh becomes firmer, and voluntary motion is made with greater activity. The growth of the limbs, which had been suspended till now, proceeds with remarkable vigor; the bones are gradually restored; and, if the deformity is not very great, it disappears by degrees: the curvatures are either diminished or altogether removed; the swellings of the epiphyses of the bones subside, and ossification proceeds with great rapidity—the affected bones acquiring greater density and strength than usual. The muscles also acquire a more powerful development, so that persons who have been rickety in childhood have afterwards become remarkable for strength." (Copland's Medical Dictionary, vol. iii. p. 645.)

The Urine in Rickets.—The earthy phosphates (constituents of bone) have been found in greatly increased amount, both by Lehmann and Beneke, in cases of Rickets—a phenomenon no doubt connected with the increased metamorphosis of bone. "It has not yet been shown, however," writes Dr. Parkes, "that this is universal, and Rickets may be connected sometimes with simply deficient ingress of the earthy salts without altered egress." The urine is commonly pale; and, according to Dr. Copland, the urea and uric acid are diminished, while the salts are increased. A free acid has been sometimes observed—said to be phosphoric—and a considerable sediment of oxalate of lime is not uncommon, while urinary calculi are frequent in rickety children. The increase in the fixed salts is most considerable during the advance in the first stage, and when the deformity begins to appear in the bones. It is less remarkable when the disease is far advanced, and when the softenings and flexures are the greatest. The phosphate of soda and the earthy phosphates are then most abundant. (Copland.) Lactic acid has also been found by Marchand, and by Gorup-Besanez, associated with lactates and a great excess of the earthy phosphates. An hypothesis has been raised on these observations, namely, that "an undue amount of lactic acid in the body dissolves and carries out the earthy salts." In a case recorded by Mr. Solly (Medico-Chirurg. Trans., vol. xx. p. 448) three or four times the usual amount of phosphate of lime existed in the urine. (Parkes, Copland.)

These phenomena have been described with reference to the extreme manifestation of the disease, but far less intense forms of the constitutional affection and disease of the bones are frequently seen. Enlargement of the carpo-radial epiphyses and curvature of the tibia, are sometimes accompanied with so plump and fresh a condition of the child, that it is with great difficulty the parents can be induced to think there is anything amiss with it. (Wiltshire.)

Diagnosis.—The constitutional manifestations of Rickets are to be recognized as distinct from those of scrofulosis, tuberculosis, or syphilis; although they have by some been all regarded as mere modifications of one and the same disease. To determine whether similar maladies are identical, four things are essential to be known:—(1) a knowledge of causes; (2) a knowledge of symptoms; (3) a knowledge of the effects of remedies; (4) a knowledge of the morbid appearance of the diseases in question. On comparing, therefore, the phenomena of Rickets in these respects with the corresponding phenomena in each of the diseases mentioned, it will at once be seen how different Rickets is from either of those affections. The general phenomena of each disease respectively are perfectly distinct from the phenomena of the others; and the pathological tendencies of each are dissimilar. As shown also by the investigations of Sir W. Jenner, the pathological tendencies of any one of these affections are rarely manifested by those who are the subject of either of the others; and although Rickets does not by any means exclude tubercle, yet it is absolutely unfavorable to tuberculization. Nevertheless, rickety children may be tubercular, just as syphilitic children may be so. Tuberculosis, also, is well known to be hereditary; but from what has been already stated under the *causes* of Rickets, it does not appear that Rickets is hereditary in the sense that phthisis is hereditary. The facts collected by Dr. Edwards and Sir W. Jenner point to the conclusion that, while more than forty per cent. of tuberculous children are born of phthisical parents, about nine per cent. only of rickety children come of phthisical parents. The

converse, also, may take expression in the fact, that the children of phthisical parents are not found to be especially prone to Rickets or to scrofulosis; and although several members of one family may be the subjects either of Rickets, or of tuberculosis, or of scrofulosis, it is comparatively rare for members of the same family to be the subjects of more than one of these constitutional affections. Lastly, as will appear from what has gone before, and from what is to follow, the cause, the prognosis, the pathology, the morbid anatomy, and the treatment of each of these affections are different. (Jenner, Trousseau, Merei.) Dr. Wiltshire, however, entertains a modified view, inasmuch as he regards Rickets and tuberculosis as two distinct forms, or local manifestations, of one general dyscrasia—namely, the scrofulous. (Med.-Chir. Rev., July, 1856, p. 75.)

From what has been written, it may be conjectured that the diagnosis of Rickets in the precursory or incubative stage of its development will mainly lie between tuberculosis, especially of the lungs, the peritoneum, or of the brain, or cerebro-spinal membranes. A careful observation of symptoms of records of temperature, as contrasted with those given by Dr. Sidney Ringer[1] relative to the recognition of tuberculosis, will soon show whether or not the phenomena are similar to those described under the symptoms of Rickets. When the sternal extremities of the ribs begin to swell, and become rounded in a club-shaped form; when the softened sternum begins to project more than it ought; when, with increasing pallor of the skin and increasing flabbiness of muscle, the carpal epiphyses of the radius and ulna become enlarged, and greatly so in proportion to the metacarpal epiphyses of the wrist bones, so as to give a "knotted" or "double-jointed" appearance; then the disease may be surely recognized.

Osseous tuberculosis is distinguished from osseous Rachitis in the following respects:—

The bones in both diseases are loaded with morbid blood; but the blood in Rickets differs from that in tuberculosis; it is of a brown rather than of a bright-scarlet hue, and resembles chocolate rather than blood.

The modifications in the form and direction of the bones are dependent upon the weight and pressure they have to sustain.

The state of the bone is in a great measure dependent upon a deficiency of earthy material, so that the bones are uniformly soft.

The bones tend to produce regular curves, as in the spine and thigh-bones; and the pelvis becomes peculiarly distorted. The heads and necks of the thigh-bones become depressed. The joints become distorted from a yielding of their ligaments. These deformities of the bones and joints ascend progressively from the lower to the higher parts of the skeleton—the lower limbs first yielding to the superincumbent weight. (Stanley.)

There are peculiarities also in the individual bones. They tend to form exostoses. The development and nutrition seem to be vitiated from a peculiar cause in Rachitis, different from that in tuberculosis.

Rachitis and tuberculosis are rarely associated; and tubercle is less frequently found in children who have died from Rickets, than in those who have died from other diseases. (Ancel on Tuberculosis, pp. 31, 32.)

A diagnosis is also to be established between the true Rickets and that softening of the bones in adults to which the name of *osteomalacia*, or *mollities ossium*, has been given. In true *Rickets*, if the patient survives, re-ossification or hardening of the bones is certain to take place; but *osteomalacia* is never followed by re-ossification. It rather induces fatty degeneration of the bones (never seen in Rickets), and irresistibly progresses to a fatal termination. (Vogel.)

PATHOLOGY.—The history of the development of Rickets is the history of the development of a constitutional disease. Such a disease becomes developed under the influence of agencies *within* the body itself, and acting through the continuous exercise of its functions. The disease is thus *inbred;* the constitutional or the original organization of the child being of such a kind that the continued exercise of its functions, in place of preserving the growth and development of the body in a healthy state, becomes associated, in the first instance, with slight deviations from the standard of health, and ultimately leads to well-marked diseases, often of so fixed a character and so strongly expressed, that the local lesions have sometimes been looked upon as the real disease. Hence Rickets has so frequently been classed among diseases of the bones, and described with reference to softening of the bones only and alone, which is a mere expression (and only one expression) of a general morbid state which implicates the whole system and constitution of the body. To classify Rickets with diseases of bones is therefore a mistake; for, as Sir W. Jenner has well observed, "Rickets is no more a disease of the bones, than is typhoid fever a disease of the intestines. Rickets leads to disease of the bones in the same way that typhoid fever leads to

[1] See "On the Temperature of the Body as a Means of Diagnosis in Phthisis and Tuberculosis," by Sidney Ringer, M.D., 1865.

disease of Peyer's patches." The phenomena therefore which characterize the condition of Rickets are of such a kind that they are an expression of the unhealthy state of the system, which pre-exists the development of the local lesions; and a cachectic state, a "bad habit of body," is invariably associated with the development of the disease. The change in the bones is a mere expression, and only one of many anatomical signs or characters of Rickets; just as the changes in the joints or white tissues are mere expressions of rheumatism, each of which may be regarded as so many anatomical signs or lesions developed under the influence of the constitutional disease.

The lesions characteristic of Rickets are rarely limited to one part or system; and before death ensues, or even before the constitutional disease abates, several organs, systems, or tissues become diseased or degenerated.

In all the constitutional diseases of children (as well as of adults), the seats of such lesions or structural change are characteristic of the particular constitutional affection; while, for a long time before local lesions of structure are expressed, various forms of ill-health denote the constitutional affection.

There are several well-marked diseases of childhood which are thus constitutionally developed; namely, *Rickets*, *tuberculosis*, *scrofulosis*, and *inherited* or *congenital syphilis*.

Each of these constitutional diseases is characterized by certain premonitory phenomena which, taken singly, may not justify suspicion, but which, when considered collectively, yield presumptive or circumstantial evidence that certain constitutional phenomena will terminate in the characteristic lesions of structure in systems or organs of the body, which are peculiar to each of these diseases.

The deviations from the standard of health in each case may be so slight, that it may not seem to merit the name of a disease; but when these deviations are invariably succeeded by the expression of certain well-marked pathological tendencies, the relation of such slight deviations from health to such local manifestations of disease can no longer be mistaken, ignored, or overlooked.

For example, when the condition of the skin is such that it can merely be regarded as delicate, no definite diagnosis can be come to; but when we find it so often associated with the growth of tubercle in certain parts, we cannot ignore the pathological connection between the slight deviation from the standard of health experienced by the delicate skin, and the morbid tendency of the constitution expressed by the development of tubercle in certain organs. Again, when the complexion merely excites attention by the thickness and coarseness of the features, no definite diagnosis may be pronounced; but when we know that such complexions are often associated with a peculiar form of ophthalmia, we cannot overlook the pathological connection between the slight deviation from health expressed in the complexion, and the morbid tendency of the constitution expressed by the ophthalmia of scrofulosis.

Again, when the skin attracts attention from the mere muddiness of its tint, no definite diagnosis may be arrived at; but when it is seen that such muddy skins are frequently associated with characteristic lesions in the skin and mucous membranes, we cannot fail to connect in pathological relationship the deviation from the standard of health expressed in the tint of the skin, and the morbid tendency of the constitution expressed by the lesions of the dermis and mucous membrane peculiar to syphilis.

So also, when the muscular power of a child attracts attention from its feebleness merely, no definite diagnosis may be warrantable; but when such lowness or feebleness of the muscular power is followed by softness of the bones, the pathological relationship cannot be overlooked, which obtains between the slight deviation from health expressed by the feebleness of the muscular power, and the morbid tendency of the constitution expressed by the softened bones.

The leading features of such typical pathological relationships as subsist between slight deviations from the standard of health, and peculiar deviations from the healthy structure of particular organs, have been thus concisely expressed by Sir W. Jenner, in the following tables:—

LEADING FEATURES IN TYPICAL CASES OF

Tuberculosis.

Nervous system highly developed; mind and body active; figure slim; adipose tissue small in quantity; organization generally delicate; skin thin; complexion clear; superficial veins distinct; blush ready; eyes bright; pupils large; eyelashes long; hair silken; face oval and good-looking; ends of long bones small; shafts thin and rigid; limbs straight.

Children, the subject of tuberculosis, usually cut their teeth, run alone, and talk, early.

Leading pathological tendencies.—Fatty degeneration of liver and kidneys; growth of tubercle, and consequences thereof; inflammation of serous membranes.

Scrofulosis.

Temperament phlegmatic; mind and body lethargic; figure heavy; skin thick and opaque; complexion dull, pasty-looking; upper lips and alæ of nose thick; nostrils expanded; face plain; lymphatic glands perceptible to touch; abdomen full; ends of the long bones rather large; shafts thick.

Leading pathological tendencies.—Inflammation of the mucous membranes of a peculiar kind; so-called strumous ophthalmia; inflammation of the tarsi; catarrhal inflammation of the mucous membrane of the nose, pharynx, bronchi, stomach, and intestines; inflammation and suppuration of the lymphatic glands on trifling irritation; obstinate diseases of the skin; caries of the bones.

Rickets.

Mental capacity and power small; muscular force deficient; mind and body inactive; figure short; closure of the fontanelles retarded; face small, but broad; skin opaque, often set with downy hairs.

Children, the subject of Rickets, are late in cutting their teeth, in running alone, and their teeth drop early from their sockets.

Leading pathological tendencies.—Softening of the bones; enlargement of the ends of the long bones; thickening of the flat bones, and deformities consequent on these conditions of the bones; so-called hypertrophy of the white matter of the brain; chronic hydrocephalus; pulmonary collapse; laryngismus stridulus; convulsions; albuminoid infiltration of the liver, spleen, and lymphatic glands.

Syphilis.

Adipose tissue small in quantity; muscles flabby; cutis rough, deficient in contractility; complexion muddy.

Leading pathological tendencies.—Suppurative inflammation of the mucous membrane of the nose; ulceration of the mucous membrane of the nose and the lips, mouth, throat, and anus; falling of the hair; eruptions on the skin of peculiar character; induration of the liver; suppuration of the thymus, lungs, &c.

Rickets, therefore, can no longer be regarded merely as a local disease, characterized by a mere chemical abnormity of the bones—a mere deficiency of their earthy salts. Not only is there an insufficient deposition of the lime-salts in the growing extremities of the long bones, but the amount which is deposited there is abnormally placed as regards the anatomical constitution of bone. It is found in the cartilage cells, instead of in the matrix. And not only is there a deficiency of the lime-salts, and an abnormal position of those present; but if the bones were hard before the disease began, they begin to soften, in consequence of the absorption of the lime from the shafts of the long bones, and from the substance of the flat bones. The earthy matter in a soluble form enters the blood, and is excreted by the urine. Moreover, in Rickets the growth of bone is abnormal, irrespective of the absence of lime; and the agents concerned in the nutrition of the bones remove the lime from them, in place of taking it to them from the blood. (Jenner.)

The excessive formation of acids in the stomach of the child has been assigned as the mode in which the phenomena of Rickets are brought about. It has been supposed that a superabundance of acid thus finding its way into the blood, facilitates the removal of the earthy salts from the bones; and it is fully proven that in Rickets, as in most other constitutional diseases, a superabundance of free acid is constantly generated in the *primæ viæ*. The particular acid is the subject of various statements. *Lactic acid* (Marchand), *oxalic acid* (Beneke, Ure, Schmidt), *phosphoric acid* (Weatherhead), and *hydrochloric acid*, have each in their turn been deemed the peccant agent in bringing about the characteristic lesion in Rickets. But all children who suffer from the generation of free acid are by no means sufferers from Rickets. None of the acids have been demonstrated in the blood: and altogether, there are "no facts which remove the theory from the category of pure hypothesis." The theory is also in opposition to the fact that the lime is deposited in abnormal situations.

Morbid Anatomy.—A peculiar morbid condition of the bones is the anatomical sign of Rickets, just as the growth of tu-

bercle is of tuberculosis, or as lesions of the kidney with anasarca are of Bright's disease, or as peculiar affections of the joints are of gout and of rheumatism. The most constant and striking anatomical lesions in Rickets are thus enumerated by Sir W. Jenner :—

"(1) Enlargement of the ends of the long bones—of the parts where the bone and cartilage are in contact, *i. e.*, where the cartilage is preparing for ossification, and where ossification is advancing in the cartilage.

"(2) Softening of all the bones.

"(3) Thickening of the flat bones, *e. g.*, the bones of the skull, the scapulæ [and the pelvic bones].

"(4) Deformities which follow as mechanical causes acting on the softened bones, *e. g.*, the deformities of the thorax, pelvis, spine, and long bones.

"(5) Arrest of the growth, not only of the bones, but of all parts directly related anatomically and physiologically to the bones, *i. e.*, of the muscles, vessels, nerves, and teeth.

"(6) Certain lesions of the pericardium, lungs, and capsule of the spleen — the direct consequences of the thoracic deformity.

"(7) Less constant but highly important changes, most commonly affecting the nutrition of the brain, spleen, liver, lymphatic glands, and muscles, and now and then of every organ."

The enlargement of the growing ends of the long bones is an actual hypertrophy. Sir W. Jenner has measured the circumference of several wrists, in rickety and non-rickety children, and he has found that, whether reference be made to the age, or to the height of the children, or to the length of the forearm, the circumference of the wrist was always greater in the rickety than in the non-rickety. The measurements of three children gave the following results :—

Disease.	Age.	Height.	Length of forearm.	Circumference of wrists.
Rickets	4 years	30 inches	$4\frac{1}{2}$ inches	$4\frac{3}{8}$ inches
Rickets	3 years 2 months	30 "	$4\frac{1}{2}$ "	$4\frac{1}{2}$ "
Tuberculosis . . .	3 years	35 "	5 "	$3\frac{3}{8}$ "

Similar enlargements are found to exist at the costal ends of the ribs of the ankles, of the olecranon process of the ulna, and, generally, over the growing ends of all the long bones ; and the bones that are the most covered by soft parts suffer equally with those which are less covered, and therefore more exposed, although the late Mr. Stanley taught that actual expansion occurs only in those joints which are superficial. In the healthy child, the ends of the long bones always measure more in circumference than the shafts, for as the processes of ossification are completed, the bone diminishes in girth.

The microscopical characteristics of the osseous substance in the bones of rickety children, have been investigated carefully by Kölliker, H. Meyer, Rokitansky, Virchow, and Jenner.

With regard to ossification in Rickets, it has been found that the anatomical characters are peculiar in the following respects: (1) in the disproportionately large epiphysal cartilages, the larger of the ossifying cartilage cells (those disposed in rows) measuring, instead of half a line, as much as from two to five lines ; (2) in the border of ossification being dentated, the cartilage and the bone severally interlocking and interlacing with each other ; (3) in well-marked rachitic bones, the deposition of calcareous granular particles at the border of ossification is wanting, and the cartilage cells or capsules are metamorphosed into bone cells almost invariably shortly before the matrix begins to ossify, and are also without any appearance of calcareous granules. (Kölliker.) There is thus excessive formation or hypertrophy of the structures which precede or form the nidus for ossification, while there is at the same time retardation or incomplete performance of that process. It is, in fact, an exaggeration of the condition usually seen in the first stages of ossification in the healthy subject — the completion of the process only is stayed. There is thus excessive development of the spongy tissue of the head of the bone, and of the epiphysis, and of the layer of cartilage in which the primary deposit of earthy matter takes place. (Jenner.) In the ossifying shaft of a rickety bone, Kölliker has observed that the bone cells are formed from the cartilage cells by the thickening of their wall, with the simultaneous formation in it of canalicular vacuities. If the rows of cartilage cells of the large hypertrophied ossifying border be traced from without to within, it will be seen that at the point where the deposition of calcareous salts commences, they exhibit, instead of a membrane indicated by a single tolerably strong line, a much thicker coat, which on the inner side pre-

sents delicate indentations. Kölliker believes that these cartilage cells are about to be transformed into bone cells or lacunæ. This becomes all the more evident when, further on in the growth of the bone, the thickness of the membrane, together with the simultaneous diminution of the cavity of the cell, is seen to be constantly increasing; the indentations of the interior contour line become more and more marked; and, accompanying the progress of these changes, the walls become more and more dark from the addition of calcareous matter. The very slow ossification of the matrix between the cells, in Rickets, is favorable to the observation of these changes, and permits of the alterations in the cartilage cells, and their formation into bone-cells or lacunæ, being seen, and traced step by step. Cartilage cells, inclosing secondary cells within them, are connected as a whole into a single compound bone-cell; and such cells are frequently met with having two cavities, which cells, according to their degree of development, are sometimes wide, and furnished with strong prolongations, and sometimes, from their contracted cavity and long canaliculi, resemble in all respects perfect bone lacunæ. The cartilage cells lying free, and in close apposition, though in a now ossified matrix, thus become transformed into bone-cells, with nuclei and other contents; and so an ultimate change takes place by which the bone substance in Rickets acquires pretty nearly the nature of sound tissue. These ultimate changes, so far as they affect the bone-cells, depend, in the first place, upon the commencement of ossification in the matrix, but without any primary formation of calcareous granules; secondly, upon the continuous and increasing deposition of earthy matter in it, and in the thickened cell-walls, owing to which the new bone substance becomes more and more white to the naked eye.

Kölliker's account of the lacunæ formation (of which he has given drawings) is supported by Rokitansky and Virchow. Virchow shows that the abnormity of the process of ossification in Rickets consists "not in a process of softening of the old bone, but in the non-solidification of the fresh layers of bone as they form; and that we see the process of growth better in rickety than in normal bones, because in the rickety bones the view is not obscured by the deposit of calcareous granules. The old layers of bones are consumed by the normally progressive formation of medullary cavities; and the new layers remaining soft, the bone becomes brittle. There is also a certain irregularity in the growth of the bone, so that stages in the development of bone which, when the formation is normal, ought to set in late, set in at a very early period in Rickets. In normal growth, for example, the pointed processes (in which shape the calcareous salts shoot up into the cartilage) form, along the margin of calcification, such a completely straight line, that it should almost be described as mathematically regular." This condition ceases to obtain in Rickets, and the more so the greater the severity of the case. Interruptions occur in such a way that in some places the cartilage still reaches a long way down, whilst in others the calcification has mounted up to a considerable height. These uncalcified parts sometimes become so completely separated from one another, that they remain forming specks of cartilage in the midst of the bone, and surrounded on all sides by it; and that cartilage is still found at points where the bone ought long since to have become transformed into medullary tissue.

The further the process advances, the more do we meet with isolated masses of lime scattered in the cartilage; in many instances to such a degree, that the whole of the cartilage on section appears dotted with white points. The irregularity of the process is further shown in this, that whilst in the normal course of things the medullary spaces should begin to form only at a short distance behind the margin of calcification, they exceed these limits in Rickets, and in many cases a series of connected cavities extends far beyond the border of calcification, which (cavities) are filled with a soft, slightly fibrous tissue, with vessels running up into them. Medullary spaces and vessels are therefore met with where normally and properly not a single medullary cell, and scarcely a single vessel, ought to have been found. Different histological conditions are thus found side by side, crowded into a small space: at one point cartilage, at another calcification, at a third bone or medullary tissue, and everything lying in the greatest confusion: in one place medullary tissue, above it osteoid tissue or bone, by its side calcified cartilage, and below it, perhaps, cartilage still retaining its original condition. The whole of the rachitic portion of the diaphysal cartilage acquires no real firmness, and this is one of the chief causes of the liability to distortion which rickety bones exhibit, not in the continuity of the diaphyses, but at the articular ends. This is in many cases considerable, and is the sole cause of many a deformity, as, for example, in the thorax. The curvatures in the continuity of the bones are also incomplete fractures —solutions of continuity—while those of the epiphyses are due to the proliferation of the cartilage, and constitute simple inflexions. Thus, it is easy to conceive how parts which are so entirely deprived of their regular development as in Rickets, must retain great mobility. The individ-

Fig. 19.

Vertical section of cartilage from the diaphysis of a rickety growing tibia from a child two years old. A large conical process of medullary tissue sending out a lateral band on the left side, extends from *m* up into the cartilage; it consists of fibrous basis with spindle-shaped cells, at the circumference: at *c c c* the cartilage is in a state of proliferation, with large cells and groups of cells; at *c' c'* commencing thickening and internal indentation of the cartilage capsules, which at *o o* coalesce and form osteoid tissue. 300 diameters. (After Virchow, p. 634.)

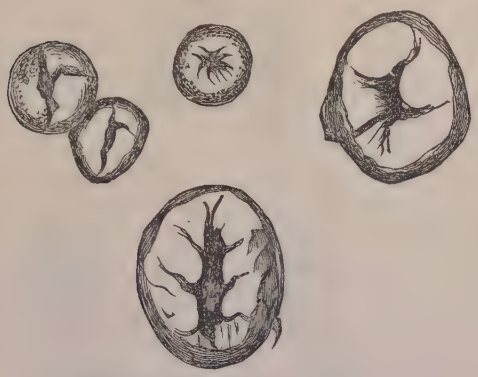
ual parts in the cartilage that ought at a later period to have become bone do not calcify, and the junction of medullary spaces often takes place a long way up above the border of calcification. Large and often very vascular conical processes of fibrous medullary tissue are seen, extending upwards from the bone into the cartilage. They do not force their way into the cartilage from without, but seem to owe their origin to a fibrillation of the intercellular substance of the cartilage itself. It is around them chiefly that the osteoid transformation of the cartilage can best be seen, and particularly that gradual conversion of a cartilage corpuscle into a bone-corpuscle. Out of the cartilage corpuscle, which has a moderately thick capsular membrane, arises a structure, provided with a capsule continually increasing in thickness, within which the space for the cell constantly grows smaller, and which, when it has attained a certain degree of thickness, acquires indentations on its inner wall, like the so-called dotted canals of vegetable cells. After this, a fusion of the capsule with the basis substance very generally ensues, and, with the production of anastomosing processes from the cells, the formation of bone-capsules is completed. At times, isolated osteoid cartilage corpuscles calcify alone, without the occurrence of any fusion; and whilst between them lies the ordinary intercellular substance of cartilage, the capsules of the osteoid corpuscles fill themselves completely with calcareous salts. In other places, on the contrary, the fusion of the capsules with the intercellular substance takes place very rapidly; the new intercellular substance formed by this fusion assumes a coarsely fibrous appearance, and in the place of several groups of cartilage cells, a fibrous mass is seen, containing jagged osseous (bone) or osteoid corpuscles. There is therefore no sharply-defined boundary in the tissue, but the condensed or fibrous substance which surrounds the jagged bodies is directly continuous with the translucent substance which holds the cartilage together. Essentially it is the same structure. At the point of transition at the zone where the conversion of cartilage corpuscles into a perfect osseous substance is taking place, numbers of corpuscles may be seen lying close to one another like hazel-nuts — distinguished from ordinary cartilage corpuscles by their dark contours, hard appearance, and unusually great brilliancy, and inclosing in a small indented cavity a little cell. These little cells are the bone corpuscles—isolated because their capsules have not yet become fused with the basis substance—with calcified capsules which they have retained from that earlier period in their existence when they were cartilage cells. (Virchow's Cellular Pathology, pp. 432–436; translation by Dr. Chance.)

Sir W. Jenner does not, however, agree with Kölliker and Virchow, that in Rickets the normal process by which the lacunæ are formed is visible. His observations have led him to quite another conclusion; the calcification of the cartilage cells in the growing cartilage in Rickets seems to Sir W. Jenner identical with the calcification of the same parts occasionally seen in *enchondromata*.

Sir W. Jenner regards it as entirely a pathological process—a petrifaction. "The spongy tissue is much more spongy in appearance than natural, and from the interstices of its meshes a deep red pulp is expressible, ... composed of colorless nucleated cells, usually containing only one nucleus, now and then two, and occasionally several blood-globules, and in some cases a very large quantity of free fluid fat—evidence of excessive preparation for the process of ossification, and arrest of the completion of the process. The periosteum is thickened over the head of the bone, as it is over the bone generally, and attains its maximum degree of thickening just at the point of junction of the bone

with the cartilage, and it is more vascular over the whole bone, as well as thicker. A crimson pulp fills the canal of the bone, and all the interstices of the tissue of the long bones." (Jenner.)

Fig. 20.



Calcification in the cells of an enchondromatous tumor similar to calcification in Rickets. (Dr. Aitken.)

On reviewing those descriptions, it will be seen that the differences in appearance and in the interpretation thereof may be merely due to the various observers having examined the bones at different stages of the disease, and perhaps also in children of very different ages. This account of the morbid histology of the rickety bone also accounts for the great softness of its texture, which is sometimes so great that the bones may be bent by the least possible force, and the thick bones may be cut with a knife or a pair of scissors. Of course the softening is obviously due to the absence of those elements which render bone hard—the calcareous or earthy salts. The animal matter, also, of rickety bones does not seem to be normal; for in the experiments of Lehmann and Marchand, rickety bones did not yield gelatine on boiling. At an advanced period of the disease, Simon also affirms that "the animal matter is so changed that its extract yields on boiling neither chondrin nor gelatine." On the other hand, perfect gluten has been obtained from rachitic cranial and thigh bones, by Schlonberger and Friedleben. They did not find that the organic basis of rickety bone had undergone a change in chemical reaction, but the unossified cartilage contained much more water. Putting together the result of the analyses of several observers, Sir W. Jenner finds that the bones of healthy children yield about 37 parts of organic and 63 of inorganic matters; whereas those of rickety children yield about 79 parts of organic to 21 parts of inorganic matters.[1] Anatomical and physiological inquiries thus clearly establish the fact that a continuous osteogenetic process is going on up to a certain point only, during the acute stage of Rickets. Layer upon layer, as in health, of new matter is deposited, the deeper seated of which layers are constantly disappearing through absorption. These layers are soft and puffy; and so long as the cachexia of Rickets persists, they never become hard.

The thickening of the flat bones is similarly due to the hypertrophy of their growing portions with increased vascularity of the periosteum. There is a great abundance of nucleated cells, which, with blood, form the pulp, and occupy all the meshes and interstices of the bones; so that the substance of the bone seems mainly to consist of diplöe. Next within the growing margin of the bones, and close to the sutures, the thickening of the cranial bones is usually the greatest, consequently the situations of the sutures are usually indicated by deep furrows on the scalp. So also round the posterior margin of the scapula, and at its acromial, coracoid, and articulating processes, the scapulæ are thicker than at their centres.

Hypertrophies and softenings of the bones do not always proceed in an equal degree — the softening being very often out of proportion to the enlargement, while the enlargement is sometimes out of proportion to the softening. In the rickety child the ribs are often softer than their cartilages; in the healthy, and still more so in the tubercular child, the cartilages are softer than the ribs.

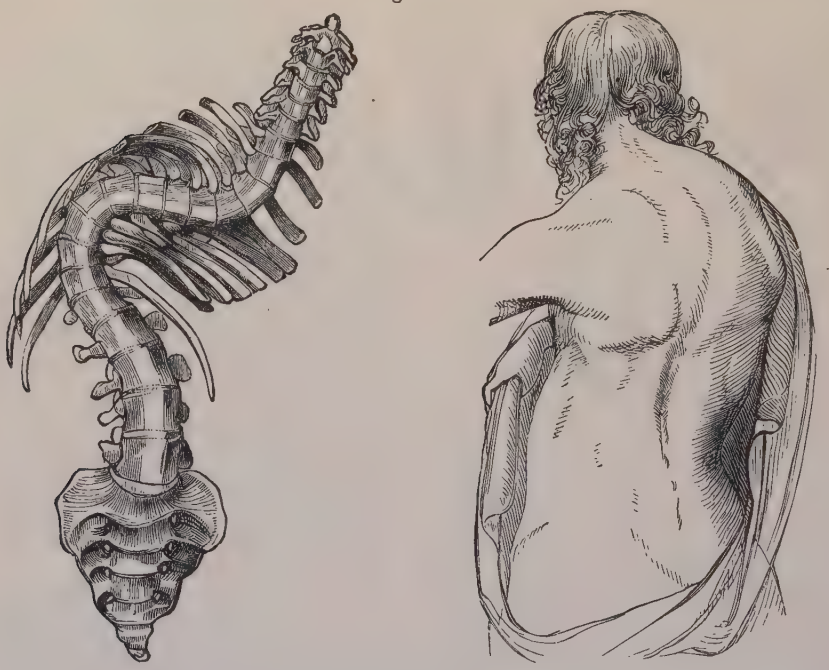
The curvature of the dorsal and lumbar spine (the symptoms of the deformities due to which have been already described) is mainly the consequence of muscular weakness and softening of the bodies of the vertebræ. The direction of the curvature is mainly determined by the weight of the head, and is generally an exaggeration of the curvature always existing when a child of three or four months old sits unsupported on the nurse's arm. Simple lateral curvatures are thus less common than antero-posterior, with more or less rotation of the bodies of the vertebræ. These become squeezed in at the concavity of the curve on the front or side, as the direction may happen to be, while on the convexity the articular processes become commensurately both thickened and enlarged. Antero-posterior bending has occurred to such an extent as to cause doubling of the aorta, adhesion of the opposed coats at the folded part, and consequent malnutrition of the lower limbs. (Miller.)

When the walls of the thorax are examined from the inside of the chest after

[1] See details of several analyses in Med. Chir. Review, July, 1856, pp. 70 and 71.

death from Rickets, the projections where the ribs join with the cartilages are much greater than on the outside; but the eleventh and twelfth ribs, which are not inflexed (as described previously), have the same enlargement in the inside as on the outside. (Jenner.) The great determining cause of thoracic deformity,

Fig. 21.



Permanent curvature of the spine, with rotation, produced by Rickets in early life. (Miller.)

according to Sir W. Jenner, is atmospheric pressure, aided by the elasticity of the lungs: and he explains as follows the mode of its operation: "Suppose," says he, "the external thoracic parietes were made of unyielding material, then the diaphragm could descend only so far as the air could enter at the orifice of the larynx, and overcome the elasticity of the lungs. The thoracic parietes however, in their normal condition, are not absolutely unyielding, but there is a due relation between their strength, the power of the diaphragm and the rapidity of its contractions, the size of the orifice of the larynx, and the elasticity of the lungs.

"The chest-walls being healthy, and the orifice of the larynx of normal size, if the young child sobs violently—*i. e.* contracts the diaphragm with abnormal rapidity and force—the most flexible parts of the thoracic parietes will yield or fall in during inspiration.

"If the orifice of the larynx be narrowed, and if the diaphragm contract with only normal rapidity and force, there will be recession of the softer parts of the chest-walls at each inspiration. Again, if the orifice of the larynx remain normal, the diaphragm acting energetically, as in a healthy child, and the chest-walls be softened (as in Rickets), then, at each inspiration, there will be recession of the most yielding part of the thoracic walls."

These are the conditions which we obtain in Rickets. The part of the rib where ossification is imperfect and incomplete is so soft that, at each descent of the diaphragm, it recedes, and the furrows on the thorax already described at page 479 are produced. In proportion as the ends of ribs are forced inwards, the sternum is carried forwards. Sir W. Jenner rightly excludes muscular action from all direct share in the production of these deformities of the thorax or curvatures of the long bones in Rickets. He does not agree with Rokitansky that the deformity of the thorax is the consequence of any want of power of the respiratory muscles; for on dissection he finds no correspondence between the points of insertion of the muscles of inspiration attached to the outer surface of the chest-walls and the points of recession. Nor does he find that the diaphragm causes circular recession by its direct contraction, as by drawing in the receding parts at each contraction; for dissections, compared with cases and models, prove that the line of recession does not correspond to the points of attachment of the diaphragm, but it cor-

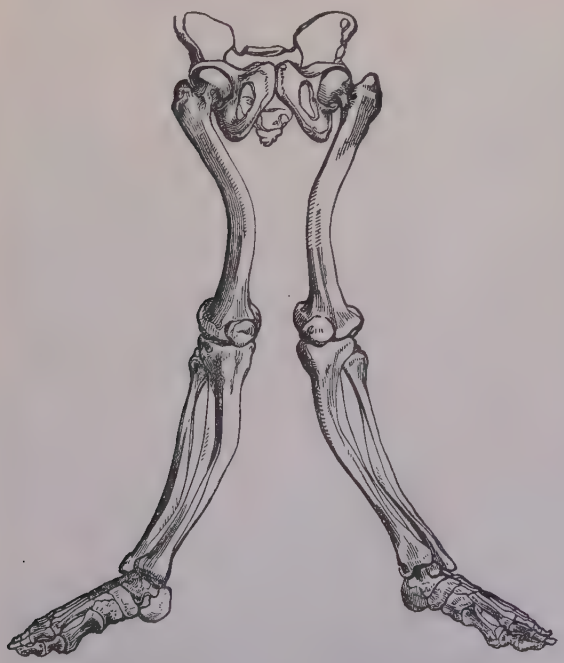
responds to the upper margin of the liver, spleen, and stomach, and is produced (as the longitudinal thoracic furrow is) by the atmospheric pressure: the parts of the parietes below being prevented from receding by these solid organs, whose influence in preventing recession of the chest-walls is illustrated by the *apparent* bulging of the precordial region in every case of well-marked rickety thorax. The chest-walls covering the heart do not recede so much as those on the opposite side; and as the left side is thus much fuller than the right, it might be supposed that there is abnormal fulness of the precordial region, which there is not.

Another point fully noticed by Sir W. Jenner is the influence of attrition in producing "white spots" or "white patches" on the surface of the heart, in children whose chests are deformed by Rickets, and in such children the chosen seat of the "white patch" is on the *left* ventricle, a little above its apex, just at the spot which impinges against the fifth rib where it projects or knuckles inwards; for the sternum of the rickety thorax being forced forwards, the relative positions of the chest-walls and of the heart are no longer normal, and the apex of the heart strikes *outside* the nipple. "White patches," similar in nature and origin, may also be found on the spleen. They are to be distinguished from those due to embolism by the fact that they do not extend below the fibrous tunic of the organ.

In connection with the morbid anatomy of the skeleton, the results of Mr. Shaw's observations must not be overlooked. He has shown that arrest of the growth of the bones, and of the parts in relation with them, is a very important consequence of Rickets; and the arrest of growth commences during the progress of Rickets, and persists after the general disease has ceased. Hence children are not only stunted in growth, when the subjects of Rickets, but they never grow into ordinary-sized adults. Arrest of growth is most strongly marked in the legs and thighs, imparting dwarfishness to the frame as well as distortion. While the bones of the thighs and legs are often bent in a variety of fantastic forms, they are at the same time flattened, generally so that the great diameter of the bone is antero-posterior in relation to the curve; consequently, when they ossify and harden in after life, as the disease subsides, the limbs are not so weak as they otherwise would be. The heads and necks of the thigh bones bend downwards, and may ultimately come to be on a lower level than the trochanters.

The articulating ligaments fail, causing deformity of the knee and ankle-joints. The pelvis is small, its front wall is flattened, and forced back upon the sacrum. A characteristic hollowness is thus imparted to the loins, by the sacrum being thrust downwards; its promontory becoming usually salient, and its posterior surface forming the bottom of a hollow on the back part of the pelvis. At the same time both ilia are displaced backwards, so as to overlap the sacrum and approach each other, sometimes having scarce an inch of space between their posterior borders. (Miller.)

Fig. 22.



Example of limbs deformed by Rickets. (Miller.)

According to Mr. Shaw's researches, all the bones of the adult whose skeleton has suffered from Rickets are diminished in length, but the lower limbs are disproportionately diminished in size, and the face is small in proportion to the skull. This arrest of growth of the bones of the face and the sinuses causes the forehead to appear to project more than it really does. Mr. Shaw's observations were directed to the examination of the relative proportions which the different parts of the skeleton bore to each other. The relative dimensions of rickety skeletons show that they have a configuration quite different from that which belongs to the natural skeleton. (Med.-Chir. Trans., vols. xvii. xxvi.)

Such alterations in important visceral cavities as have been described, inevitably lead to secondary lesions in the contained organs. Two lesions of the lungs are constantly present when the thorax is deformed by Rickets, namely, collapse of lung tissue and that form of emphysema which has been termed *insufflation*, due to over-distension with air of the vesicular tissue of the lung. In the lungs of rickety children its site is constant. It invariably occupies the whole length of the anterior border of both lungs, extending backwards for about three-quarters of an inch from the free margins.

The emphysematous portion is separated from the healthy portion of lung by a groove formed by the collapsed air-cells of the lung; and this groove of collapsed tissue corresponds to those projections inwards of the ribs where they unite with their cartilages.

The mechanism of the production of these lesions is thus described by Sir W. Jenner: "The softened ribs, instead of being drawn outwards at each inspiration, are forced inwards by atmospheric pressure; the consequence is, that not only are the lobules of the lung beneath not expanded, but they are compressed; and the compression of the lung, aided by its elasticity, causes the collapse."

The emphysema of the anterior border is produced thus: "The lateral diameter of the thorax is diminished at the part corresponding to the line formed by the junction of the ribs and cartilages. But at each inspiration the ribs recede; and in proportion as the ribs at this part are forced inwards, the sternum must be thrust forwards; and just as less air or no air enters into the tissue under the receding ends of the ribs, so an excess of air is drawn (as we commonly call it) into the lung tissue subjacent to the abnormally advancing sternum and cartilages of the ribs.

"The collapse is thus directly consequent on the recession of the ends of the ribs during inspiration; the emphysema is directly consequent on the thrusting forward during inspiration of the sternum."

These lesions stand in direct relation to the anatomical sign of Rickets, namely, the affection of the bones; but there are other lesions which, equally with the bone lesion, are anatomical signs of the constitutional affection, and which as such have been described by Sir W. Jenner. The emaciation of Rickets is mainly associated with the lesions about to be noticed, and which consist in a form of albuminoid infiltration of lymphatic glands, spleen, liver, brain, kidney, heart, and thymus gland. Sir W. Jenner is of opinion that these lesions are never limited to one of these organs in Rickets: but that every one of them is more or less affected, perhaps all the tissues. The cut surface of such organs is singularly pale and transparent, compact, smooth, tolerably moist, and to the unaided eye uniform in appearance. The substance is tough and the organ heavy in proportion to its size; and although Sir W. Jenner is opposed to the belief of such lesions being the same as those described by Virchow under the name of amyloid degeneration, yet with the exception of his failing to get a *blue* reaction with iodine, their characteristics appear to be similar.

Prognosis.— The sooner the disease becomes established after birth the more certain is the result likely to prove fatal; but those in whom the disease appears later in life may recover before the fifth or sixth year. The general health begins gradually to improve, the tumefaction of the abdomen begins to subside, and the bones to acquire firmness. Thus becoming *set*, as it were, in their abnormal shapes, the figure always retains a certain degree of deformity. When the disease ceases, the mind, like the body, recovers, and regains all its powers. The muscles of those who were once rickety, in after-life are often marvellously powerful, their bones singularly strong, and their intellect certainly not below the average. (Jenner.) Recovery sometimes takes place after an acute febrile attack, occasionally accompanied by the appearance of a cutaneous eruption. (Cumin.)

At the approach of convalescence, an extraordinary liking for particular articles of food may be exhibited; and Dr. Cumin relates how one patient had so strong a desire for salt, that she devoured it like sweetmeats. The quantity taken was very great, and the parents believed it had proved the means of curing the child.

In severe cases, where deformity of the thorax is extreme, the great danger of death is from catarrh and bronchitis. The danger is not only in proportion to the severity of the inflammation of the air-passages, but to the degree of softening of the ribs. In estimating the danger of bronchitis in a sickly child, it is by no means sufficient to listen to the auscultatory signs, or to note the lividity of the lips, or action of the nares, or the frequency or severity of the cough, or evidence of febrile disturbance; but the child must be stripped, so as to note the degree to which the ribs are softened, how much they recede during inspiration, and to what extent they are forced outwards during expiration. (Jenner.)

If the complaint occur in children about the second year of age, or later, although it may be of considerable duration, yet amendment is generally rapid when it commences; but, even when the growth

is stunted and the deformity considerable, still the period of puberty may remarkably develop growth and diminish deformity, especially when a change of air and out-door exercise can be enjoyed.

Generally—if the disease be not far advanced, if the child be not greatly debilitated, if the deformity have not greatly affected the spine, chest, or pelvis—a favorable result may be anticipated; but when the reverse of these conditions prevails, then complete recovery may not be expected.

THERAPEUTICS.—Looking to the nature and causes of Rickets, it is obvious that no specific remedy will cure the disease, nor will any detailed and fixed line of treatment be applicable to every case. An outline of the principles on which cases of Rickets are to be managed may be given; but the selection of particular remedies must be made for each case, according to the pathological conditions of the patient.

Improvement of the general health is the first object to be sought after. Such hygienic influences must be obtained as to secure for the patient healthy nursing, a warm and dry atmosphere, due ventilation, and pure air. The food must be selected as suitable to the respective periods of infancy or childhood, as becoming in the stages of lactation, weaning, or dentition. The diet, therefore, and the state of the digestive organs, are to be carefully seen to by the physician. If the child be under eight months old, and "brought up by hand" wholly or in part, milk diluted with about one-fourth part of lime-water, and with a teaspoonful of cream added to the half-pint of fluid, will be found the best food. It is better not to add sugar to the milk; and if it is used, it is better to be sugar of milk, and not cane-sugar. (Jenner.) Gruel, plain biscuit, aërated bread-crumbs, or baked flour, may be added to the milk; and as the child gets older, beef-tea and bread, eggs and farinaceous pudding, may be added to the diet. If the child be still sucking, the milk of the mother or nurse should be examined as to its quality and quantity; and, if deficient in either, the nurse ought to be exchanged for one more suitable, or the child entirely weaned, and "brought up by hand." Ass's milk has been recommended at this period; or farinaceous articles of diet should be given with mutton-tea, veal-tea, or beef-tea; or the yolk of an egg with milk may be given once or twice a day, if the child is old enough. If the child is still to be suckled by the mother or nurse, it ought to be permitted to take the breast only at stated periods; and it may have at regular intervals, in addition to the breast milk, two or three meals of milk and lime-water.

The child should be kept as much in the open air as the weather and temperature will permit. Its clothing should be warm. Its body should be washed all over, at least once in the twenty-four hours, with warm water and soap; and daily tepid or cold sponging, according to the state of the weather and condition of the child, are also beneficial. Warm salt-water bathing or sponging, or a tepid salt-water douche on the back, loins, and limbs, preceded and followed by active friction on the surface of the child, may be of service if the strength is sufficient. The child should sleep alone, and the utmost care should be taken to preserve the bed and bed-clothes clean, fresh, and perfectly dry. The sleeping room must be well ventilated; and a lamp may be burned in the chimney, to facilitate the passage of air through the room and its exit by the chimney. (Jenner, Copland.)

With regard to the administration of medicines, the physician must be guided mainly by the presence or absence of pyrexia, the state of the urine, the state of the bowels, and the progress of the cachexia generally. The irritative fever and quickness of the pulse must never induce the prescription of such lowering remedies as antimonials, nor of blood-letting. Active depletion in any form is not to be thought of; and antimony is to held "as a poison to the subject of progressive Rickets." Mercury, unless as an aperient, in conjunction with some other drug, is equally objectionable. The fever which attends Rickets being generally characterized by asthenia, by copious or colliquative perspiration, by pale phosphatic urine, and general pallor of the skin and softness of pulse, remedies must be restorative and calmative. Alkaline remedies are considered serviceable by Dr. Copland under these circumstances, especially *carbonate of potash*, *liquor potassæ*, *magnesia*, with *infusion* or *decoction of cascarilla*, or of *cinchona*, with aromatics. Lime-water charged with fixed air is useful as a drink.

If carbonates are prescribed, small doses of dilute *hydrocyanic acid*, or of the *extract* or *tincture of conium*, will be of use. If the urine be only slightly acid in reaction, or if it soon becomes alkaline, the mineral acids, especially *hydrochloric* and *nitric*, or *nitro-hydrochloric*, may be given with aromatics, or with small doses of *hyoscyamus*, *conium*, or of *opium*. Baths, acidulated with *hydrochloric acid*, are also very worthy of use, especially if the stools are devoid of bile.

If the bowels be confined or fecal accumulations exist, they must be relieved and evacuated by stomachic aperients, such as *compound decoction of aloes*, or equal parts of *infusion of gentian*, *senna*,

or of *rhubarb*, with aromatics. If the stools are offensive, even if the bowels are relaxed and the stools yeasty, a teaspoonful of *castor-oil*, or of *gray powder* combined with *jalap*, may be given, and once or twice a week a small dose of *rhubarb* and *soda;* but equal parts of *rhubarb*, *soda*, and *calumba* powder are better than gray powder, and if continued, as they may be, with safety, the acidity of the alimentary canal will be held in check. *Prepared chalk* and *soda*, once or twice a day, may also correct superfluous acidity. When the stools are yeasty and the bowels griped, alkaline remedies should be given in lime-water and milk, with minute doses of *tincture of opium*, or of *compound camphor mixture;* and an enema containing similar alkaline ingredients may be administered. (Copland.)

When febrile disturbance has been subdued, the child must be taken as much as possible into the open air, and in the sunshine. It should as much as possible live in the open air, due regard being had to the weather, and care being taken that it be warmly clad, and not exposed to cold and damp winds. If the child has lived in town, it ought to be removed to a dry bracing sea-air, such as that of Scarborough, Lowestoft, and the east coast of England generally, in the hot months, or Brighton in the colder or foggy months. The waters of Tonbridge are well spoken of by Copland and Jenner; and although the place is inland, it has special advantages, inasmuch as while the air of the place is dry and bracing, the water from its chalybeate spa is a powerful curative agent in Rickets, on account of the iron it contains—about a quarter of a grain of the *oxide of iron* in a pint, with just sufficient *carbonic acid* to hold the iron in solution. The water of the spring has a temperature of 50° Fahr., and children will drink it readily. *Steel wine* may be added to the water, if it is desirable to give more iron. It is an extremely useful remedy, and, according to Sir W. Jenner, one of the best forms for administering iron to rickety children. A teaspoonful or two of *steel wine*, with half a grain of *quinine*, and a drop or two of *dilute sulphuric acid*, constitutes one of the best mixtures for such cases. It is especially useful when the skin is flabby, covered with perspiration, and anæmia well marked. *Tincture of perchloride of iron* in the *infusion of calumba*, or in *quassia*, or the *iodide of iron* in the *syrup of sarza*, is the best preparation of *iodine* in Rickets (Copland); and the *iodide of potassium*, in a tonic decoction or infusion, is sometimes also of service. A preparation new to the pharmacopœia—the *syrup of the phosphate of iron*—possesses the general properties of ferruginous compounds, and invigorates and increases the powers of digestion. The *citrate of iron* and *ammonia* will agree with the stomach sometimes, when it will not bear more astringent preparations of iron. It is best taken during effervescence in solution of *citric acid*, flavored with tincture of orange-peel—the salt being first dissolved in the water. (Squire.)

When the use of iron must be continued for a long time, as it is desirable it should be in cases of Rickets, *magnetic oxide of iron* is a good preparation to give twice or thrice a day. The *citrate of iron* and *quinine* is also a new and useful preparation.

The influence of *nux vomica*, *iron*, and *quinine* in combination ought not to be lost sight of; it is one of the most valuable of tonic remedies. The formula for its composition, as devised by the late Professor Easton of Glasgow, has been given by my friend Dr. Maclean, in his article on Dysentery; and therefore it need not be repeated here.

Any of the tonic remedies containing iron should be taken just before dinner.

Cod-liver oil is a remedy often remarkably beneficial. (Copland, Bouchut, Jenner.) It is best given immediately after meals, in orange juice or in orange wine, or as an emulsion, with milk and solution of *compound gum tragacanth*.

The condition of the intestinal discharges should be examined when cod-liver oil is given. I have often seen that much too large a dose of the oil is being given, by seeing the oil pass by the stools. If such should happen, a much less dose is sufficient; and generally, too large doses of cod-liver oil are prescribed. Superfluous acidity must be corrected by aperients and alkaline remedies, as already indicated.

The food should be carefully masticated; and if the teeth are deficient, Sir W. Jenner recommends that the food should be pounded in a mortar, and potatoes especially should be most carefully mashed. Children of twenty months or two years of age, suffering from Rickets, require a small quantity of meat every day, in addition to beef-tea; and milk should form for all children the evening and morning meals. Sir W. Jenner recommends that when the stools are reported white they ought to be examined, so as to determine whether or not the whiteness is due to the quantity of undigested curd contained in them. If such should be the case, alkaline remedies are to be given, and a little lime-water added to the milk; or beef-tea may be substituted for part of the milk. The liver is not to be stimulated by purgative remedies, nor alteratives given in the belief that bile is deficient.

In the inflammatory, bronchial, and lung affections of progressive Rickets, *ammonia*, with or without *ipecacuanha* and

citrate of potash, is the great remedy to be relied on; while for the cure of *laryngismus stridulus*, the purely tonic treatment comprehended in the combination of air, exercise by friction or otherwise, and diet, with iron and cod-liver oil, as already indicated, constitute the course of treatment to be followed up with perseverance.

Mechanical appliances are not to be recommended for straightening deformities; but Sir W. Jenner has sometimes applied splints in such a way as to project below the feet for the purpose of preventing walking.

[This prohibition of mechanical appliances is, perhaps, rather strongly expressed; although such means of treatment are undoubtedly often misused or abused. Unless very skilfully contrived and carefully employed, they had better be let alone altogether.—H.]

[SCROFULA.

By Henry Hartshorne, M.D.

Synonyms.—Scrofulosis, Struma, Tuberculosis, Chœras, Écrouelles, King's Evil, The Evil.

History.—The ancients were well acquainted with glandular swellings; the χοιράδες of Hippocrates, *strumæ* of Celsus, and *scrophulæ* of later Latin authors. It does not appear that the idea of a relation between these and a constitutional disease, or even with pulmonary consumption, was thought of until the seventeenth century. Silvius (Le Boë) theorized in regard to supposed glandular enlargements and suppurations in the lungs in phthisis; and other medical writers for a time accepted this view. Cullen, in the eighteenth century, most distinctly recognized the characters of the scrofulous constitution. In the same century Percival studied the pathology of phthisis with new views concerning tuberculous deposits; and then Portal, Bayle, Baillie, and Laennec (1819) brought in the pathological doctrines concerning Tuberculosis which have remained until within a few years undisturbed.

Popularly, Scrofula has been the subject of a curious but quite ancient superstition. Serenus Samonicus, in his "Carmen de Medicina," written about the beginning of the third century of our era, alludes to the cure of the "regius morbus" by the imperial hand. In England and France, many monarchs were supposed to have the power to restore, by the touch, those affected by it.

Edward the Confessor in the eleventh century, Louis XI. and Charles VIII. in the fifteenth, Francis I. in the sixteenth, and Charles II. in the seventeenth centuries, were famous in this respect. Queen Anne in England and Louis XVI. in France were the last who touched for the King's evil; Samuel Johnson, when a boy, having been one of those so favored by the British queen. A special formula in connection with this royal office remained in the liturgy of the Church of England as late as 1719.

The essential identity of Tuberculosis and Scrofulosis has been recognized by most pathologists since tubercle has been definitely studied. Several eminent authorities, however, have denied it; while the exact nature of their relation continues to be much discussed. That either tubercle or the cheese-like deposit or degeneration of scrofulous glands is an affair of merely local disorder, is now maintained by very few. A constitutional tendency, "diathesis" or "cachexia," is generally understood to lie beneath all such local manifestations of disease. The importance of this subject is well expressed thus by Aitken:[1] "There is perhaps no subject in the whole range of medical science which the student ought to study more carefully than the cachexia associated with the occurrence of tubercle. As a practitioner he will find that he becomes often painfully concerned in the deepest interests of families and society, through the threatened or actual ravages of tuberculous diseases." In England, for example, in one year, 1862,[2] there were reported as dying from—

[1 Science and Practice of Medicine, Phila. edition, vol. ii. p. 227.]

[2 Tanner, Practice of Medicine, Philada. edition, p. 104.]

Scrofula	3,416
Tabes mesenterica	5,203
Phthisis	50,962
Hydrocephalus	7,031
	66,612

In France, about 1 per cent. of the conscripts have been declared unfit for military duty on account of Scrofula.[1]

To the same effect as that above quoted is the following language of Birch-Hirschfeld:[2] "Practitioners have at all times opposed the attempt to get rid of this nosological conception, and, indeed, whoever has had some experience in the diseases of childhood, must acknowledge that the manner in which certain individuals react against noxious influences leads to the assumption of a definite constitutional habit."

The general agreement of clinical observers and pathologists in regard to the existence of such a "constitutional habit," furnishes sufficient reason for the retention of the old term, Scrofula, or Scrofulosis.

SYMPTOMATOLOGY.—Postulating at the start the essential identity of the tubercular with the scrofulous constitution, it is proper to mention the traditional account of the strumous *physiognomy*.

Miller[3] gives the following delineation of its characteristics: "The complexion is fair, and frequently beautiful, as well as the features. The form, though delicate, is often graceful. The skin is thin, of fine texture; and subcutaneous blue veins are numerous, shining through the otherwise pearly-white integument. The pupils are usually spacious; and the eyeballs are not only large, but prominent, the sclerotic showing a lustrous whiteness. The eyelashes are long and graceful, unless *ophthalmia tarsi* exist, as not unfrequently is the case; then the eyelashes are wanting, and their place is occupied by the swollen, red, unseemly margin of the lid.

"In the phlegmatic form the complexion is dark, the features disagreeable, the countenance and aspect altogether forbidding, the joints large, the general frame stinted in growth, or otherwise deformed from its fair proportions. The skin is thick and sallow; the eyes are dull, though usually both large and prominent; the general expression is heavy and listless; yet not unfrequently the intellectual powers are remarkably acute, as well as capable of much and sustained exertion. The upper lip is usually tumid, so are the *columnæ* and *alæ* of the nose, and the general character of the face is flabby; the belly inclines to protuberance; and the extremities of the fingers are flatly clubbed, instead of presenting the ordinary tapering form."

Dr. Aitken, in the article in the present volume upon Rickets, has contrasted (in a Table) the appearances and tendencies of Tuberculosis and Scrofulosis, along with those of Rickets. To this comparison we may refer the reader for instructive suggestions. Not very dissimilar is the description given by Birch-Hirschfeld[1] of the two types, of *erethic* and *torpid* Scrofulosis. It may be remarked, however, that the necessary connection of each of these physiognomies with scrofular or tubercular disease can hardly be sustained. What is true seems to be, that one (erethic) of these habits of constitution affords a delicacy, which lapses readily into irritability; while the other (torpid) manifests a relaxation or atony, easily depressed into degeneration. A predisposition only, to Scrofulosis, may be inferred from such signs; and this predisposition may not unfrequently be escaped from, under favorable hygienic circumstances, without the occurrence of actual disease.

Scrofulous disorders affect often a quite early period of infancy, although seldom congenital—the *tendency* to them, as above indicated, being all that is usually transmitted by inheritance.

Earliest, are apt to occur morbid affections of the skin and mucous membranes; after these, those of the serous membranes and lymphatic glands: then of the bones; and, commonly, though far from always, last, of the lungs. This is, it is true, a clinical rather than a pathological account of the most frequent order of succession. Many times, tubercular disease of the lungs and serous membranes is latent or obscure; and then the superficial or obvious disorder attracts, for a time, an attention out of proportion to its relative pathogenetic importance.

Willigk, in 1317 cases of Tuberculosis examined, found the order of frequency of invasion of different parts of the body as follows: lungs, intestines, mesenteric glands, larynx, lymphatic glands, peritoneum, spleen, kidneys, pleura, liver, air-passages, bones, genital organs, brain, cerebral membranes, urinary passages, pericardium, stomach, bowels, skin, muscles, tongue, pharynx, œsophagus, heart. Rokitansky has given the following order from his enormous experience: lungs, intestines, lymph glands, larynx, serous membranes, brain, spleen, kidneys, liver, bones, uterus, testicles.

Young scrofulous subjects are much in-

[1 According to Marc d'Espine, 1·6 per cent. of all deaths in Geneva result from scrofulous diseases.]

[2 On Scrofulosis; in Ziemssen's Cyclopædia of Practical Medicine, vol. xvi.]

[3 Principles of Surgery, p. 55.]

[1 Loc. cit.]

clined to disease of the *skin*, especially upon the head and face. Yet it can hardly be said that any one form of cutaneous eruption is exclusively peculiar to scrofula. *Scrofuloderma* is a term applied to those affections, somewhat various, which present characters presumed to be especially dependent on the strumous diathesis.

Lichen scrofulosorum is named by Hebra as a papular disease, consisting of papules of the size of millet seeds, reddish or brownish-red, in groups, generally on the trunk, least often on the limbs. Being painless, and itching but little, they may exist without being noticed; but are liable to remain for a long time unchanged; or successive eruptions may come out, even for years. Males between ten and twenty-five years of age are most often affected with this disorder. The hair-follicles and sebaceous glands are chiefly involved in it.

More common, however, is scrofulous *eczema*. Beginning not unfrequently with small papules, this becomes vesicular, or pustulating (impetigo), or scabbing, with an oozing serum, or drying into crusts (*crusta lactea* of children). If it differs in scrofulous patients from the same affection in others, it is in its being, with them, less inflammatory, and more chronic in duration.

Sometimes we meet with cases in which, along with enlargement or destruction and cicatrization of lymphatic glands in the neck or axilla, there are ulcers or small cutaneous abscesses, more or less numerous, whose history is almost pathognomonic of Scrofulosis. The ulcers, in this affection, are apt to be rather deep, with smooth, undermined edges. The discharge from them is thin, and scarcely purulent; and the area around them is bluish-red or violaceous—not copper-colored, as in syphilitic cases. After these, which are very slow to heal, are left cicatrices of an irregular, radiated, or funnel-like appearance, sometimes knotty, but more often depressed, or with both knots and depressions. Such cicatrices, along with enlarged cervical glands, or scars from their previous suppuration, always point to a scrofulous constitutional origin.

Children, even young infants, are often affected with strumous disorders of the *mucous membranes*. That of the nose is the most common seat of this trouble; with redness, moderate swelling, and soreness, and a discharge, prone to harden in the nostrils, but flowing out sufficiently to irritate the skin at their edges, and to inflame and thicken the upper lip. The external meatus of the ear is frequently likewise affected. Otorrhœa then occurs; at first mucous, afterwards purulent; it may be very tedious in duration. From the external ear sometimes, but more often from the nares, inflammation may extend to the middle ear, with danger of perforation of the membrana tympani; or, it may even pass more deeply, and affect the brain, with meningitis, thrombosis of the cavernous sinuses, or cerebral abscess. Such complications or sequelæ are rare, however.

Chronic *conjunctivitis* (scrofulous ophthalmia) is a very common attendant of the strumous constitution; although it cannot be inferred that every case of obstinate chronic ophthalmia is scrofulous. Occurrence in childhood, without observed causes of local irritation, with a particular disposition to affect the eyelids, and with extreme *photophobia*, out of proportion to the other inflammatory symptoms,—these are traits which only need to be found coincident with disease of the lymph-glands or of the bones, to establish the diagnosis of scrofulous ophthalmia.

A minute herpetic eruption may exist and involve the conjunctiva, and even the cornea. Sometimes the iris and deeper structures of the eye may suffer serious injury. While, however, blindness may thus occasionally result, in a large proportion of cases scrofulous ophthalmia does not reach the retina, and leaves the sight unimpaired.

The mucous membrane of the mouth is, no doubt often affected with chronic inflammation in scrofulous children, though less frequently than the nostrils and ears; unless we ascribe to the same constitution numerous cases of sore throat, to which infants are subject. More consistently, although still far from invariably, we may assert the same origin for chronic enlargement, without active inflammatory symptoms, of one or both tonsils.

The *lymphatic glands* are, as has been observed since the time of Hippocrates,[1] affected chiefly after the time of infancy. From six to sixteen years may be approximately named as the time of their especial liability to strumous enlargement; but it is far from rare both earlier and later.

The glands of the neck are most frequently affected. Balman[2] found these involved in 81 per cent. of all cases; the axillary glands in 6 per cent. only; the inguinal in 7; those of the knee about 5; and those of the popliteal space in less than 1 per cent.

Commonly, the swelling of the glands is moderate, or increases gradually, without pain or much soreness. In some cases, however, inflammatory symptoms are more active; even bringing on febrile reaction of the system. S. Ringer's observation of the coincidence of elevation

[1 Aphorism iii. 26.]

[2 Researches and Observations on Scrofulous Disease: London, 1852.]

of temperature with the deposit of tubercle, while often confirmed in regard to pulmonary tuberculization, has not been so extensively noted in connection with glandular strumæ; in which, nevertheless, Schüppel and others have discovered well marked tuberculosis. Fever is most apt to occur either when several glands commence to enlarge at once, or when cheesy degeneration is followed by softening, preliminary to opening and discharge.

Very often "kernels" may form and remain with little change for months or years; or they may grow slowly, until they become as large as a goose's egg, or larger; or, after a time, slow absorption may follow a partial softening, and they may disappear altogether. Whether really more frequent or not, however, the attention of the practitioner is most often called to those which advance from the condition of apparent hypertrophy (hyperplasia of Virchow), through caseous metamorphosis, to softening, discharge, ulceration, and cicatrization. Scrofulous ulcers may be indolent and slow to heal; but they are not invariably so; and it is remarkable how fair a share of general health may co-exist with them. Quite often a succession of single or multiple enlargements and degenerations may appear in the course of a few years. It is when febrile irritation attends them that we have the greatest reason to apprehend the later sequence of pulmonary consumption.

Scrofula does not often affect the salivary glands. Now and then, however, the sub-maxillary, or the parotid, or both, may be involved along with the cervical glands. Sometimes mumps, occurring in strumous subjects, may be followed by chronic enlargement of the parotid, or even by its degeneration and suppuration.

The *bronchial* glands are not unfrequently affected, and may undergo the same succession of changes as those of the neck, axilla, or groin. The symptoms are then modified by the locality of the adenoid disease.

Hooping-cough or measles may be followed by obstinate chronic bronchitis. If other signs of the scrofulous predisposition exist, we should always in such a case suspect bronchial glandular enlargement. But it may occur without any such exciting cause; being then as "idiopathic" as any other glandular struma.

Signs of this affection are a dry, irritative cough; dyspnœa, rather gradually increasing; and, sometimes, tenderness on pressure in the intercostal spaces near the spine. Percussion resonance will be dull from the fourth to the eighth dorsal vertebra. Auscultation will give, in the same region, a bronchial respiration much louder than normal; the breathing sound being conducted from the air-tubes by the enlarged glands.

It is much less common with the bronchial than with the cervical glands for softening and discharge to take place; unless in cases in which, tuberculization of the lungs occurring, the more important and destructive lesion masks the earlier and lesser one. Cases are observed, however, in which caseous or suppurating bronchial glands have discharged their contents through the bronchi or trachea, in the absence of pulmonary disease.

Pressure of much enlarged bronchial glands upon the vena cava may cause interference with the circulation; shown by distension of the jugular veins, blueness of the lips, and a tendency to puffiness of the cheeks.

The *mesenteric* glands are liable to scrofulous enlargement, mostly slower in progress than that of the bronchial glands, and equally obscure in its symptomatic manifestations. In cases attended by much wasting, the glands may be felt by careful abdominal palpation. Tumefaction of the belly, however, is quite common; and, even in its absence, the tumors cannot very often be felt. Disorder of digestion, diarrhœa, debility, and emaciation (marasmus) make up the usual *ensemble* of *tabes mesenterica*. Softening, with discharge of the contents of the glands, is not common. Occasionally it has been traced by the passage of caseous matter through the bowels; and, more rarely, discharge into the cavity of the peritoneum has caused peritonitis with a fatal result.

Tubercular peritonitis may occur, however, without any such cause. It is attended by pain and tenderness on pressure over nearly the whole abdomen; with tumefaction, and dulness on percussion, fever and debility. Fluctuation also marks the occurrence of serous effusion.

Periglandular abscesses form, sometimes, when the glands themselves do not soften and suppurate. *Cold abscesses* is an old name given to accumulations of pus in various places, chiefly of scrofulous origin. One locality in the lower abdomen and pelvis has long given occasion for the name of psoas abscess. These purulent collections appear to form sometimes irrespective of glands, in the connective, and, perhaps, the muscular tissue. The presence or absence of acute febrile symptoms in connection with them seems to depend mainly upon the rapidity of their formation. Hectic fever, however, attends a late stage of them in many cases. The result may be favorable, if a discharge of the accumulated matter takes place externally, or by the bowels. If we are to suppose that a considerable amount of pus cannot be safely removed by absorption, it is to be inferred that such tumefaction, in the abdomen or elsewhere, may occur, with only a non-purulent exudation; since undoubtedly they do in some instances

disappear, after persisting for several weeks or months. Dulness on percussion, clearly circumscribed, without fluctuation, and with local signs excluding enlargement of the spleen, kidney, liver, &c., give diagnostic evidence upon these points.

An important part of the history of Scrofulosis, especially in the period of adolescence, is the frequent occurrence of disorder of the digestive and assimilative apparatus, sometimes called *strumous dyspepsia*. This has been especially studied and described by Wilson Philip, Tweedy Todd, J. Hughes Bennett, and Jonathan Hutchinson, as an important preliminary condition to phthisis, in a great number of cases. Difficulty in the digestion of fatty substances is a prominent feature of it; and Hughes Bennett has emphasized this as bearing upon the pathogeny of tuberculization.

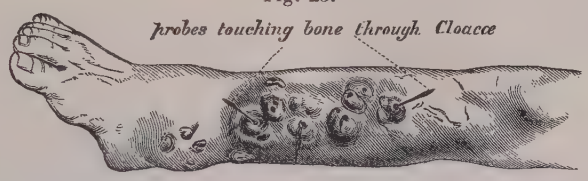
Scrofulous disease of the *bones* includes ostitis, periostitis, caries, necrosis, and spina ventosa; with inflammatory affections also of the joints,—as fungous arthritis (white swelling), kyphosis (Pott's disease of the spine) and coxalgia or hip-joint disease.

Of the bones, the tibia is most often involved; next in frequency, the femur; then the radius, ulna, humerus, and vertebræ. Among the joints, next after the coxo-femoral articulation, the knee is the most liable; then the ankle and elbow; least so of all, the shoulder and wrist.

Commencing periostitis, as, *e. g.*, of the tibia, is shown by a smooth, slightly reddish swelling of the bone, tender to the touch, and suffering an aching pain. As the bone itself becomes affected, at last perforation occurs, with a semi-purulent discharge, ulceration, exfoliation of bone in fragments, &c.

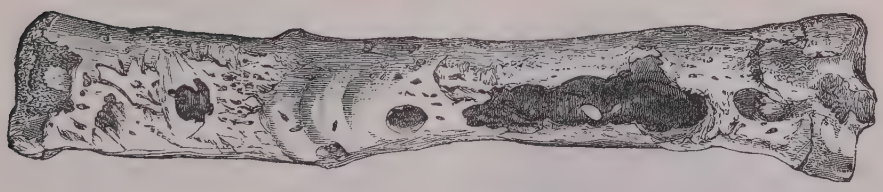
Caries of the spine is attended by deformity, from the weight of the body crushing the softened vertebræ. Considerable pain is apt to result, from pressure upon the spinal nerves, and irritation near their roots; also, disorders of innervation in the parts supplied by the nerves entering and leaving the cord near the seat of disease. Indigestion, palpation, dyspnœa, neuralgia, and paralysis are among the troubles thus produced. Recovery, however, is not very rare.

Fig. 23.



External appearance of limb, the seat of Necrosis of the shaft of the Tibia with cloacæ leading down to dead bone.

Fig. 24.



Necrosis of the whole shaft of the Tibia.

Coxalgia, as well as the other affections just mentioned (Pott's disease, caries of the long bones, &c.), is by general consent regarded as a surgical subject, not appropriate for full consideration in a work of this kind. It will be suitable here, therefore, only to remind the reader of one or two points of interest to the medical practitioner.

Every hospital student knows that the earliest signs of hip-disease are, pain in the knee without other indications of disease in that joint, and a limping gait, bending the knee of the affected limb, so as to touch the toe only to the ground. Pressure upon the great trochanter on that side, or upward upon the knee or heel, will give pain, because of the concussion of the head of the femur against the acetabulum. Atrophy of the muscles surrounding the joint follows; the whole limb seeming to become smaller. Destructive caries of the head of the femur may occur, with suppuration, spontaneous opening of the joint and discharge, burrowing of pus under the muscles and fasciæ, hectic fever, emaciation, and exhaustion. If the disease gives way to treatment at an early stage, no deformity may be left; if later, the best attainable result may be permanent anchylosis of the joint.

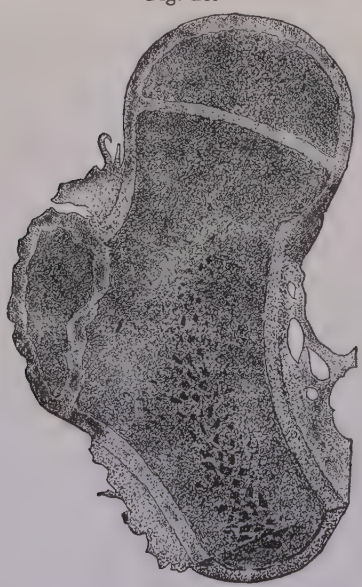
White swelling (fungous arthritis, arthrocace) is the designation formerly applied especially to scrofulous inflammation of the knee-joint. This may begin with

Fig. 25.

Section of head of Coxalgic Femur.

some violence, particularly if it have had an injury as an exciting cause. More

Fig. 26.



Healthy Femur, section.

often, however, its approach and progress are gradual. In this case febrile reaction belongs to a rather advanced stage of the disease. Pain is not apt to be very severe. Lameness of course results. Ulceration and suppuration occur in severe cases; from which, however, restoration may take place, if the constitutional health be invigorated. Too often it proves to be the case, that the obstinacy of the arthritic affection is but one of the manifestations of imperfect nutritive power; to be followed, in later adolescence or early maturity, by fatal phthisis.

Scrofula (according to the view adopted in this article) affects the brain, in children, quite often, with what is denominated tubercular meningitis; more rarely with cerebral or cerebellar Tuberculosis. Several eminent pathologists have recorded the observation, to whose correctness I can bear testimony on the basis of my own autopsies, that cases having all the typical clinical characters of tubercular meningitis may prove, after death, to have reached their end without any tubercular deposits. Bouchut, Rilliet, Hahn, Hughes Wiltshire, and Litten may be named as having given evidence to a similar effect upon this point. The term "scrofulous meningitis" has been preferred for such reasons, by some of these writers, to the ordinary designation of the disorder.

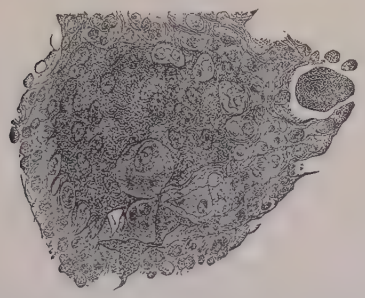
Broncho-pneumonia and "catarrhal" pneumonia, in persons of the strumous constitution, are attended in many instances by caseous deposition or degeneration in the lungs, discriminated by some recent pathologists from tuberculization. Symptomatically, these cases of pulmonary caseation present nothing distinctive, for clinical description, from phthisis of recognized tubercular character. Louis is credited with having established the general law, that "whenever tubercle occurs in the body it also exists in the lungs, and whenever it occurs in the lungs it appears first at the apex." But the opinion has also been sustained by such authorities as Addison, Gairdner, Jenner, Reinhardt, Virchow, and Niemeyer, that pulmonary consumption may occur, and proceed even to a fatal termination, without tubercular deposit in the lungs. Such cases, like those of fatal meningitis above alluded to, may be safely designated as instances of scrofulous phthisis; this term referring to the *diathesis* or cachexia, whether tubercular deposition result from it or not.

For the study of the history of all varieties of consumption, we may refer the reader to the article in this work on Phthisis (Vol. II.) by Prof. J. Hughes Bennett. On Tubercular Meningitis, full information is given in this volume, in the article by Dr. Samuel Jones Gee. Our present subject, Scrofula, requires now the further consideration of some general questions, which may be regarded as still *sub judice*.

ANATOMY AND PATHOLOGY. — Few subjects in the domain of medical inquiry have been more closely investigated, or more variously discussed, in modern times, than that of tubercle. When we remember how general, although not universal, has been the inclusion together of Tuberculosis and Scrofulosis as practical synonyms, we may perceive how relevant and necessary is a view of at least the salient points of this discussion to the study of Scrofula.

Two stages may be traced in the ordinary course of Scrofulosis in *glands*. First, a moderate and not rapidly increasing enlargement, with an elastic and tolerably firm consistence. In the few instances of examination of glands at this stage with the microscope, abundant cell-proliferation has been observed and reported. Cornil asserts, also, that there occurs a prodigious development of the connective tissue of

Fig. 27.



Tuberculosis of a Lymphatic Gland. The earliest stage of the process. Showing the giant-cell. ×200. (From Green.)

the gland. Secondly, comes the stage of caseous metamorphosis. The gland becomes more enlarged and harder. Gradually it is altered to a dry yellow mass, consisting, as seen under the microscope, of irregular corpuscles, shriveled cells and nuclei, and (Birch-Hirschfeld) "flaky elements which may be claimed as giant-cells." Schüppel and others assert the distinctly tubercular character of the yellowish-gray nodules found in many such glands. Such, it is declared, may exist largely in glands without caseation; also, cheesy degeneration may occur without being preceded or accompanied by gray tubercular deposit, though this is rare in the lymphatic glands.

Subsequently, a strumous gland frequently softens, and its contents liquefy into a material resembling pus to the naked eye, but without pus-corpuscles, consisting only of granular débris, mingled with the shriveled and altered elements of the affected tissue.

Sometimes, after seeming to have become caseous, such glandular enlargements may slowly disappear. Virchow considers that their substance must in such cases be absorbed. More often, without sign of softening, there is a persistent enlargement, whose contents, if opened, will be found to be a yellowish-white, cheese-like substance, with more or less normal gland-structure, crowded with altered cells; in some cases having undergone calcareous degeneration. Always, in the early stage of scrofulous deposition, animal material, chiefly albuminous, predominates; later, fatty elements become more numerous; last, if not entirely liquefied, phosphate and carbonate of calcium abound.

Far more frequently, however, gradual softening and liquefaction occur, resulting in spontaneous opening through the skin, with discharge, ulceration, and final granulation, leaving the characteristic cicatrization before described.

Through all the recent discussions as to what constitutes tubercle, it has been generally admitted that, while all caseation is not tuberculous, true tubercles are often found in scrofulous lymphatic glands. Bazin, Köster, Friedländer, Nélaton, Roux, Lannelongue, and Brissaud have observed them in scrofulous diseases of the joints and long bones; Schüppel, in those of the skin; Wagner, Rindfleisch, and others assert their presence in nearly all cases of caseous pneumonia; Charcot declares that, in all instances of the latter affection, the caseous deposit is a truly tuberculous mass. Friedländer insists that tubercles are also found in old non-scrofulous ulcers, as of the cervix uteri, in syphilitic sores, &c. Virchow considers that local tuberculosis may occur irrespective of any general diathesis, although the scrofulous constitution undoubtedly predisposes to it.

To the question, *what is tubercle?* leading pathologists do not, to-day, give a united answer. Formerly, the account of it was tolerably simple;[1] describing it as of two *kinds*, gray, semi-transparent, granular—and yellow, opaque, and caseous; distributed also in two *forms*, miliary and infiltrated. In both of these varieties, however distributed, the most characteristic elements under microscopic inspection were considered to be the tubercle-corpuscles; irregular, round or oval, from $\frac{1}{200}$ to $\frac{1}{100}$ of an inch in largest diameter, regarded by some (Virchow) as shriveled nuclei. With these have always been seen elements of disintegrated tissue; epithelial cells, oil globules, calcareous particles; also, results of inflammation or hemorrhage, as exudation corpuscles, blood-corpuscles, &c.

Now, under the leadership of Virchow, caseation is held by many to be a process

[1 See "Phthisis Pulmonalis," by Prof. J. Hughes Bennett, in Vol. II. of this work.]

of degeneration not peculiar to tuberculous or scrofulous deposits, although most frequently occurring in them. Virchow, Herard, and Cornil, with many others, attach the name of tubercle only to miliary, and not to infiltrated, deposits. Rindfleisch asserts[1] that the cells found in exudations from scrofulous inflammation are larger than those of other inflammatory exudations; and Oscar Schüppel's "giant-cells" are commonly regarded as the most characteristic elements of tubercle in any organ.

Allusion was made above to the adhesion of Charcot (so recently as 1878)[2] to the old doctrine of Laennec, that caseous infiltration of the lung in phthisis is truly tuberculous. While so much conflict of opinion exists, the subject being evidently in a transitional state, it would be out of place here to dogmatize upon it. Yet an opinion, if it be but provisional, must be hazarded upon each of the most important disputed questions.

Does only tubercle undergo caseation?

It seems necessary to accept the view of several pathologists, that caseation is but a modification of fatty degeneration; which may affect morbid products of different kinds, as, for example, cancer or other tumors. Yet, in far the greatest number of instances, caseous metamorphosis results from the scrofulous constitution, predisposing all the organs of the body to abnormal *vulnerability under slight irritation*, and *deficient restorative power* when diseased.

Scrofulous inflammations, therefore, are characteristically attended by slowly forming exudations, crowded with cells of feeble vitality, whose accumulations are, more than any other deposits, liable to caseous degeneration.

Is tubercle ever purely *local*, not constitutional?

An affirmative answer must be given to this question also; but simply as a matter of fact, so far as shown by the absence, in certain cases,[3] of scrofulous symptoms or changes in any but a single organ or part of the body. This does not at all contravene the judgment that a *predisposition* to similar disease in many organs existed in the same individuals; as predispositions are commonly latent for a time, being made known only by disease under exciting causation. Escape from the effects of such predisposition may take place, when locally exciting causes continue to be absent.

Is tubercle always inflammatory in origin?

Notwithstanding the high authority of those who have urged that it is invariably so, the facts appear to warrant the maintenance of Laennec's doctrine upon this point; to the effect that tubercle may be deposited, as a local result of a constitutional fault, without inflammation preceding it. Litten, of Berlin, in some recent elaborate investigations of acute Tuberculosis,[1] has sustained what may be believed to be a true proposition, viz., that "tubercles may develop extensively in the membranes of the brain, without a trace of exudation." Yet much the larger part of the clinical history of Scrofulosis undoubtedly consists in that of scrofulous inflammations of various organs; sometimes the one process (inflammation) and sometimes the other (tuberculosis) being primary; and, not unfrequently, the peculiarity of the case being, the *modification* of the local inflammation by the diathesis, making it less violent but more prolonged and unfavorable in result, even without any tubercular *deposit* at all.

Does gray tubercle always precede the yellow?

If in the same part both occur, the older doctrine here seems to be best defended; notwithstanding the quite opposite views of Buhl, Waldenburg, Niemeyer, and others. Gray tubercle always occurs as a primary form. Yellow nodules or infiltrations result from degeneration either of gray tubercle or of some other material; which may be inflammatory exudation, or (Schroeder van der Kölk, Radclyffe Hall) the epithelium of the organ involved.

Is "caseous infiltration" tuberculous in nature?

Charcot's authority may be (as above cited) used in defence of the conviction, that, however morphologically different from miliary tubercle, the essential nature of the process and of the product is identical in both. To a contrary effect are some expressions of Cornil and Ranvier; for instance:[2] that scrofulous and tuberculous arthritis are "*bien distinctes.*"

Is tubercle *infective* in origin?

Following Dittrich, Buhl, in 1857, introduced the hypothesis of the origin of pulmonary tubercle in the "resorption" of a virus from scrofulous lymphatic glands, or from some other locality of primary disease. Villemin (1865), Colin, Lebert, and others succeded in producing tuberculosis in animals by inoculation. These experiments and reasonings are well considered and sufficiently disposed of by Prof. J. Hughes Bennett, in his article in this work (Vol. II.) on Phthisis

[1 See Rindfleisch's article on "Chronic and Acute Tuberculosis," in Ziemssen's Cyclopædia of Practical Medicine, vol. v.]

[2 British Medical Journal, May 25, 1878.]

[3 Birch-Hirschfeld, loc. cit.]

[1 Volkmann's Sammlung Klinischer Vorträge, No. 119, 1878.]

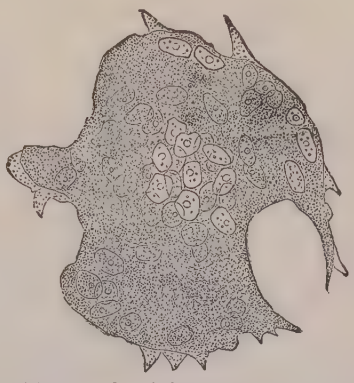
[2 Manuel d'Histologie Patholog. p. 433.]

Pulmonalis. It has been shown that various morbid materials, introduced by inoculation into the veins or lymphatics, may bring on local tuberculization, by their obstructive and irritative action. There is no demonstration whatever either of the existence of a specific scrofulous "virus," or of the origin of tubercle by infection, in any true sense. There may be added here to Dr. Bennett's denial of the theory of infection, the cogent argument derived from the observations and statistics of Prof. Austin Flint, of New York,[1] against the recently often asserted origin of phthisis in catarrhal pneumonia. In 670 cases of phthisis, carefully analyzed, Prof. Flint obtained ample clinical evidence of the small relative share taken by inflammation of the lungs in the pathogeny of consumption. Prof. A. Clark,[2] of New York, also, has adduced serious objections against the theory of resorption or injection. A very important obstacle to its acceptance is the frequency of the occurrence of Scrofulosis of the lymphatic glands, bones, &c., especially in children, without phthisis following it.

What is the nature of the "giant cells" of tubercle?

Schroeder van der Kölk seems to have

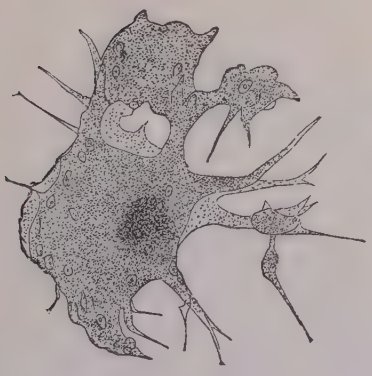
Fig. 28.



A multinucleated cell from the Lung in a case of Chronic Phthisis. Showing the large number of nuclei with bright nucleoli. × 400. (From Green.)

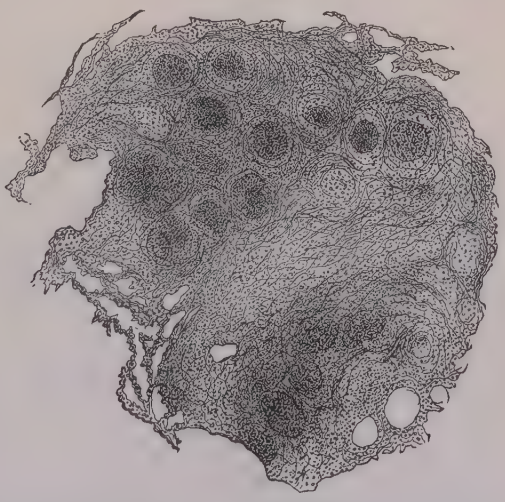
described these many years ago, as "compound cells, consisting of epithelium charged with the nuclei which become

Fig. 29.



A multinucleated cell from the Lung in a case of Chronic Phthisis. Showing the long-branched processes, which are continuous with the reticulum of the surrounding indurated growth. Some of the processes are in connection with smaller nucleated elements. × 200. (From Green.)

Fig. 30.



Multinucleated and branched cells from a firm gray miliary tubercle of the Lung in a case of acute Tuberculosis. Wide meshes are seen in the immediate vicinity of the cells inclosing a few lymphoid elements. The branched processes are directly continuous with the adenoid reticulum of the tubercle. × 200. (From Green.)

the ordinary tubercle-corpuscles." Virchow referred to them in his Cellular Pathology[3] as "larger cells with a manifold division of the nuclei, so that twelve, twenty-four, or thirty are contained in one cell." Prof. O. Schüppel, of Tübingen, however, has especially insisted on the importance of these elements of tubercle as characteristic. They somewhat resemble, but are clearly not identical, even in appearance, with the "myeloid cells" of some sarcomatous tumors, found especially in connection with the beads of the long bones. They are irregular in form, without any cell-wall, the largest ones

[1 New York Medical Record, Feb. 1, 1873.]

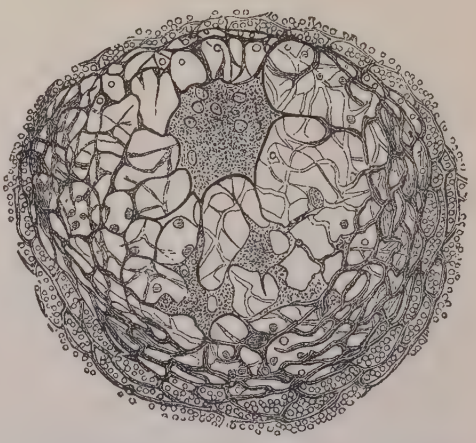
[2 New York Medical Record, Nov. 15, 1870.]

[3 Lecture XX.]

having a diameter of about $\frac{1}{250}$ of an inch. Each contains from three or four up to thirty or forty nuclei, which are conspicuously nucleolated. Branching processes extend from many of them, making a reticulated arrangement, occupied, according to Schüppel, with epithelial cells. These last, however, are not always to be seen.

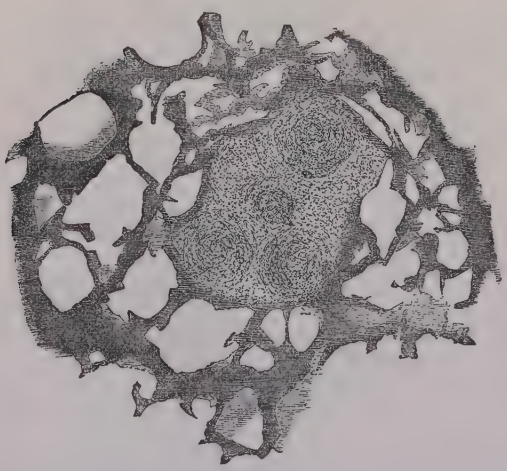
The origin of these forms has not been satisfactorily traced. Klein suggests their derivation, in the lungs, from the alveolar epithelium. If this occurs, it is most probably by the fusion of several epithelial cells. Certainly the giant-cells are not found to undergo any further development. Quite probably they are not entitled to the name of *cell* at all; but are mere masses of epithelioidal protoplasm, shaped altogether by their surroundings. In a discussion in 1878, Cornil[1] expressed an opinion concerning them which has great intrinsic probability. This was to the effect that the origin of giant-cells is *intra-vascular;* resulting from the coagulation of plasma in capillaries in the course of obliteration, whose walls soften and disappear; leucocytes, moreover, being entangled in the coagula, and their nuclei undergoing enlargement and proliferation. Deichler some years since reported an observation of "knots or buckles" of minute arterial branches in masses of tubercle; their walls softening as the cells increase in number. Aneurismal distension of the walls of the pulmonary capillaries has been described also by Buhl,[1] Lenker and Aitken.[2] By such an explanation as this we may best reconcile the recent accounts of tubercle with some of those of equally competent observers, made but a few years since.

Fig. 32.



A firm Gray Tubercle from the Lung in a case of acute Tuberculosis. Showing the grouping of the elements around separate centres, the nodule consisting of several giant-cell systems. × 33. (From Green.)

Fig. 31.



A small soft Gray Tubercle from the Lung in a case of acute Tuberculosis. The whole of the tubercle is shown in the drawing, and it is obviously constituted largely of intra-alveolar products. × 100, reduced to ½. (From Green.)

To resume: we may classify the distinctive opinions concerning the nature of tubercle as follows: 1, that it is an *exudation* (Rokitansky, Ancell, Bennett); 2, an organized *neoplasm* (Wedl, Vogel, Virchow); 3, a *degeneration* of tissue elements (Henle, Reinhardt, Van der Kölk, Radclyffe Hall); 4, a *specific infection* (Villemin, Colin, Schüppel, Green[3]); 5, an *abortive formation* of tissue, followed by degeneration, from defect of vital energy (Williams, Ziegler,[4] Sir J. Clarke, J. Hutchinson, Aitken, G. B. Wood.) Although Prof. J. Hughes Bennett has expressly designated tubercle as an exudation, he has contributed much towards the establishment of the dependence of its origin upon a *defect or perversion of nutrition;* which corresponds very nearly with the last named of the above views concerning its nature.

That tubercle often does occur in coincidence with local inflammation, and thus immediately in, and, in a sense, from

[1 London Medical Record, June 15, 1878.]

[1 Virchow's Archiv, 1862, p. 183.]

[2 Science and Practice of Medicine, vol. ii. Phila. edition, p. 238.]

[3 Infective, at least, according to Green, though not specific. See his "Pathology and Path. Anatomy," Phila. edition, p. 184.]

[4 Deutsche Zeitschr. f. pract. Med., 1874, No. 5.]

inflammatory exudation, is certain. But we have already seen reason for dissenting from the views of Niemeyer, Oppolzer, and others, that Tuberculosis *always* follows and depends upon inflammation. It may be deposited, as Laennec taught, in the lungs or elsewhere, prior to all inflammatory changes.

Against the "neoplastic" view, not much need be said at this time. Tubercle is the pathological opposite of cancer. While, in the latter, there is a perverted, misdirected excess of formative action, in Tuberculosis all the phenomena exhibit deficiency of histogenetic energy. Nothing is more marked than the *absence of vascularity* in every tuberculous mass. The reverse is true of cancer.

That a degeneration of already formed tissue-elements takes place as a part of the tubercular process, is clearly true. But it would appear that this is not the central, characteristic fact concerning it; and only a portion of the appearances presented by its morbid anatomy can be thus accounted for.

The hypothesis of a *specific virus* of infection has been overthrown by the careful investigations of Lebert, Frānkel, Burdon Sanderson, and Wilson Fox. Buhl's "resorptive" theory is well disposed of by Birch-Hirschfeld,[1] as "anatomically improbable, and never demonstrated." It is certainly not impossible that, when the lymph-glands are in an unhealthy state, corpuscles may pass from them into the circulation, then accumulating in the lungs or elsewhere, and producing obstructive disease. But it is at least equally probable, when a constitutional predisposition has been shown to lie behind the one local mal-nutrition, that it should also account for the other, especially as the order of succession of the two is variable, and many instances occur of each taking place without the other.

It remains, then, for me to express the conviction, that the essence of Scrofulosis or Tuberculosis consists in a *defect of tissue-forming power;* showing itself most conspicuously in the organs of assimilation (digestive organs and lymph-glands), in those of most active chemical change (the lungs, skin, and mucous membranes), and in those of least vital resistance (bones and joints) on account of their minimum of vascular supply. Even in the absence of inflammatory irritation, these organs may be subject to tissue-abortion, constituting tubercular deposit. But, in the scrofulous subject, a special *vulnerability* is present, so that slight causes induce inflammation, which is low in activity and prolonged in duration, "strumous inflammation." Degeneration of the products of this morbid process, with that also of the tissue-elements affected, causes that conglomerate of pathological deformations, whose description and explanation have been the theme of so much debate.

[1 Ziemssen's Cyclopædia, loc. cit.]

CAUSATION.—As an account of the etiology of phthisis is given in the article by Dr. J. Hughes Bennett upon that disease (Vol. II.), a brief summary only is here needful concerning that of general Scrofulosis.

Hereditary predisposition is universally believed to be a principal factor in the production of all varieties of Scrofula. Every physician must know instances of its occurring in several members of the same family, in successive generations. Yet statistics show that very many cases, especially of pulmonary Tuberculosis, are non-hereditary. Dr. Pollock,[1] amongst 1200 cases of phthisis, ascertained it to be hereditary in 30·16 per cent. Notwithstanding our acceptance of the view that Scrofula, affecting the skin, glands, mucous membranes, and bones, is, constitutionally, identical with phthisis pulmonalis, it cannot be denied or ignored that many children suffering with the former grow up and live long lives without lapsing into the latter; while many persons die in adult life with phthisis who never had glandular enlargement or other signs of Scrofula during childhood or youth.

In promoting the tendency to hereditary Scrofulosis, impairment of the health of parents, from any cause, is undoubtedly important. Syphilis has long been believed to bear, in this way, a close relation to Scrofula. Some have even asserted the latter to be, so to speak, a descendant, with modification, of the former. Kortum, near the end of the eighteenth century, vigorously opposed this doctrine; which is now nowhere maintained. The relation between the two is, clearly, only that belonging to the enfeeblement of vital energy brought on by Syphilis, in common with all other protracted constitutional diseases.

Smallpox has been charged with a similar promotive power. Before the time of Jenner, this was, there is reason to believe, largely shown. Dr. Greenhow reports that "during the middle of the last century, before vaccination was known, the scrofulous death-rate was more than five times as great as our present one." Of course other sanitary improvements may have contributed to this result.

Intemperance must be supposed to act unfavorably in preparing the way for every vice of constitution. But it has not been shown to promote Scrofula, in the children of drunkards, so much as it does disorders of the brain and nervous system.

[1 Second Report of Hospital for Consumption, Brompton, 1863.]

Consanguineous marriages are commonly thought to favor Scrofulosis in the offspring of parents nearly related. Whether this is really the case when no family taint had previously existed, has been much disputed. At least it is true that such a taint is aggravated, and is more likely to be manifested, in those who issue from parents of the same stock. If two scrofulous persons marry, whether blood-relations or not, the probability of an inheritance of the predisposition by their children is four times as great as if only one of the two were scrofulous and the other of a faultless constitution. At least this follows from the assumption, apparently well grounded, that the mingling of "pure blood" in marriage with that which is tainted, tends to subtract from, or antagonize, the morbid procreative influence.

Climate is more important, apparently, in regard to the prevalence of pulmonary than of glandular Scrofulosis. The statistics of the United States show that the Northern States have more deaths from consumption than the Southern; but there is no evidence that so great a difference exists in the comparative prevalence of general Scrofula. Probably, however, while no race nor climate is free from it, the artificial conditions of life induced by the need of protection from cold, in the higher latitudes, make all forms of Scrofula more common, especially in cities, in the temperate and cold than in tropical regions.

Change of climate, from a warmer to a colder one, has undoubtedly an effect, in many cases, in promoting Tuberculosis. This is seen in the Negro race, especially, when transported to a northern country. Monkeys, taken to England, not unfrequently die of consumption. All domestic animals, however, are liable, much more than the same in the wild state, to tubercular disease. This has been abundantly shown to be the case with the cow, when confined in the stable; also with the sheep and the rabbit. Animals naturally wild and active in their habits, as the lion, tiger, elephant, &c., often die of tuberculous disease when confined in menageries. This appears to be irrespective of climate, depending upon the unnatural change in their mode of life.

Dampness of locality has been shown, most fully by Dr. Bowditch in this country, and Dr. Buchanan in Great Britain, to favor Tuberculosis. A. Keith Johnson recorded the statistical fact that, in the cities of Europe, the mortality from consumption is in direct proportion to the nearness of a locality to the level of the sea. This general law has been approximately confirmed in America.

Habitually breathing foul air, made so by confinement of persons together in ill-ventilated factories, workshops, and dwellings, is a potent agency in generating Scrofulosis. This cause is aided, no doubt, by the exclusion of sunlight; as among those who work and live in mines. Baudelocque, Carmichael, Arnott, MacCormac, and Parkes are among those who have especially forwarded the demonstration of this practically momentous fact. Nothing could be more cogent in this respect than Dr. Parkes's exposition of the excessive mortality from consumption of British soldiers, living in barracks, in various parts of the world.

Poverty promotes Scrofula, by almost compelling close habits of living, in cold regions. Also, it involves another very serious cause of vital depression, namely, *deficiency of food*. Prof. Bennett has emphasized this as perhaps the most influential of all the causes of Tuberculosis. *Impropriety* of food for children, amongst ignorant people, whether poor or not, acts, evidently, in a similar manner. Dr. Tanner states[1] that during the cotton famine in Lancashire, in 1862-63, the mortality of infants among the manufacturing population diminished greatly. The mothers, who, when at work, neglected their children, being then unemployed, attended to feeding them in the natural manner; and could less afford, moreover, to stuff them with unsuitable food. Birch-Hirschfeld[2] cites the statement of Huss, that Scrofulosis is very frequent in Schonen, the richest province of Sweden, where infants, almost as soon as they are born, are fed on coffee, sour bread, and potatoes. The same author asserts that a similar condition of things occurs in thrifty districts of Bavaria, Saxony, &c.

An inquiry is alluded to by Birch-Hirschfeld, in regard to the possibility of the generation of tubercle in human subjects by their being fed with milk from cows having the pearl disease. Schüppel is considered to have shown the identity of this affection with Tuberculosis.

Gerlach, Klebs, Chauveau, and others,[3] have found it possible to produce tuberculization in animals by feeding them on such milk. The pearl disease is not uncommon amongst cattle in many places. It is, therefore, a not unimportant question, whether there is any proportion, in such localities, between the prevalence of pearl disease in milk cows and Tuberculosis in human beings. At present, there seems to be no actual proof of the occurrence of such a coincidence or result.

Insufficiency of clothing is another of the

[1 Practice of Medicine, Phila. ed., p. 109.]
[2 Ziemssen's Cyclop., loc. cit.]
[3 Bollinger, on Tuberculosis by Inoculation and Feeding. Arch. f. exp. Pharm. und Pathol., vol. i. Nos. 4 and 5, 1873.]

means by which the poor are often, in the colder climates, made to undergo depression of vital power, promotive of Scrofula. Prevention of the healthy action of the skin must impede the removal of impurities from the blood. The action of cold, too, interferes with the regularity and balance of the circulation, not only of the blood, but also of the lymph and chyle. Thus both the glands and the lungs are made vulnerable, and the whole system suffers damage to its recuperative capacity.

TREATMENT.—This must be adapted, first, to the constitutional cachexia; and, secondly, to the local disorders present.

A tuberculous mother is not likely to furnish perfect nutriment to her infant from her own breast. Many authors advise, therefore, that such should never suckle their offspring.

This counsel is judicious, if an altogether healthy and reliable wet-nurse can be obtained. If not, it may be best for the mother, when her milk is abundant, to nurse the child through the earlier months of infancy, and then gradually to wean it. Much experience shows that even very feeble women often give the best of their substance to their babes, which thrive upon it while the mothers waste.

When it is weaned, cow's or goat's milk, little if at all diluted, is the nourishment to be preferred for the infant. Care is needful that solid food be not given too soon, and unsuitable, indigestible articles never. If the child's appetite be poor, and especially if it rejects milk, concentrated animal food, as beef-tea, should be afforded to it. The indication for a highly nitrogenized diet is never more clear at any time of life, or in any condition, than in a growing infant whose powers of assimilation are low. Fatty food, moreover, as we have already seen, is especially pointed out as desirable for the scrofulous in early life. Cream and good sound butter are its most available examples; but beef and (more doubtfully) mutton gravy are sometimes also acceptable and useful.

Fresh air is a cardinal requirement in the hygiene of those having the tuberculous predisposition. The country will be, for them, better than the town, and active out-of-door interests and pursuits better than those which are sedentary. Too long confinement in the nursery or school, or, worst of all, the workshop, factory, or mine, may entail the doom of early death or lingering disease upon those whom the life of the harvest-field or the mountain-side might have made robust and of good longevity.

Bathing is another hygienic measure of much prophylactic value. During infancy, tepid bathing is usually the best; reaction against cold being then uncertain or imperfect. Later, as resistance is shown to increase, moderately cold or cool water, especially by the shower-bath, or salt water, as of the ocean surf, may be used. But the notion of hardening delicate children by severe exposure of any kind, is a mistaken one, fraught with danger.

It is only such degrees of cold as can be fully overcome by the natural calorific processes of the body, that are ever salutary. Clothing, for scrofulous children, or persons of any age, ought to be sufficient to protect the body, and, most of all, the chest and the feet, against ever being chilled. In a climate of extreme and sudden changes, like that of the Northern United States, such persons should wear flannel through the whole year; lighter in summer, and heavier in the winter, spring, and autumn. The protection afforded by silk next to the skin is probably quite as great, against vicissitudes; but the stimulus of flannel to the circulation of the skin, and its absorbent porosity, are advantages. Careful avoidance of dampness of dwellings and localities, is, of course, of great consequence.

Of *medicines* believed to be anti-scrofulous, iodine was, thirty or forty years ago, the most trusted. Lugol's essays on the effects of iodine in Scrofula were translated from the French and published in London in 1831. "Lugol's Solution" became the standard remedy, for a long time, for Scrofula as well as for goitre. Its external use for tumors of all kinds was much relied upon. Like other overrated medicines, it has now fallen into undeserved neglect. Iodide of potassium, however, holds its place in the treatment of many disorders, especially constitutional syphilis; but it is seldom mentioned by authors at the present day in connection with Scrofula or Tuberculosis.

Chloride of calcium and chloride of barium have both been advocated by a few practitioners, as having anti-strumous power. In consumption, particularly, the phosphate and hypophosphite of calcium have been much lauded and largely used. Dr. Churchill, of Paris, attracted general attention to this medication about twenty years ago. Dr. W. Minor Logan, of Cincinnati, has emphasized the significance of the well-known pathological fact, that phosphate of calcium is always present in tubercular deposits in large amount. The result of the trial of the phosphates and hypophosphites by the profession has been their failure to sustain general confidence in their value in Tuberculosis. My own use of them in the treatment of consumptive patients in the Episcopal Hospital of Philadelphia, some years ago, as well as in private practice, has convinced me that

they cannot, at all events, compare with cod-liver oil in analeptic power.

Dr. W. M. Logan reports a number of cures of phthisis by the use of nitric acid internally (30 or 40 drop doses) after meals, with tincture of chloride of iron; along with suitable hygienic measures.

Cod-liver oil, which had been for a long time a common remedy for chronic rheumatism, in the coast-regions of Germany, Holland, and Great Britain, came into use for consumption first in Germany, between 1830 and 1840. The introduction of it to the knowledge of the profession in Germany is credited especially to Schuette; in Great Britain to Percival, Donovan, and Hughes Bennett; in America to G. B. Wood. Its value in the treatment of phthisis has been placed beyond doubt; and no one has so well expounded the rationale of its analeptic action as Prof. J. Hughes Bennett.[1]

Numerous other remedies have had a temporary reputation in the general treatment of different forms of Tuberculosis. One more may be mentioned: *koumiss*, a fermented drink made from mare's or cow's milk, used of late years by the physicians of Russia. Referring to the article by Dr. Bennett on Phthisis (Vol. II.) for the special therapeutics of that malady, our attention may be given now, in part retrospectively, to the treatment of other forms of Scrofulous disease.

Iodine has certainly disappointed the hopes of many of those who have used it, both internally and externally, with the hope of its dissipating glandular or other tumors. Yet it would seem reasonable, from the evidence accumulated, to employ it in moderate doses, especially the iodide of iron, in the early treatment of scrofulous glands. Externally, in tincture or ointment, it appears to act simply as a stimulant to the circulation of the part, without other specific effect. Indeed, the expectation of finding any *specific* against Scrofula has now very much passed away.

If scrofulous glands are inflamed, they may be treated like other local inflammations; seldom with the prolonged application of cold, as the "phlogosis" is not apt to be intense; but with soothing poultices of bread, slippery-elm bark, or flaxseed meal. In all strumous inflammations, of the glands, conjunctiva, and bones, I have become convinced that the local application of carbonate of lead is more serviceable than it is in other inflammatory affections. It is best applied in the form of an unguent, made by adding two drachms of the carbonate to an ounce of simple cerate.

Cod-liver oil has not shown any remarkable power in removing glandular tumors. Its recuperative influence, however, in all states of defective nutrition, gives reason for employing it in general Scrofulosis. It certainly does good in cases of caries, &c., of the bones. Children dislike it less, proportionally, than adults. Beginning with small doses, they may be increased, until a child of ten years of age may take two tablespoonfuls or more in a day.

Iron is indicated, particularly in the feebler cases of Scrofulosis. The syrup of the iodide, already mentioned; or Blancard's pills; or the citrate, lactate, pyrophosphate, or other chalybeate preparations may be used. It should be remembered (as mentioned in the article on Chlorosis) that iron will not always agree well, even with anæmic patients. Headache and indigestion, and sometimes feverish symptoms, will, in such cases, show that the dose is too large, or that it had better be withdrawn for a time.

The measures of local treatment of scrofulous otitis and otorrhœa, as well as of ophthalmia, ostitis, and periostitis, fungous arthritis of the knee, coxalgia, and Pott's disease of the spine, are, in detail, most appropriately set forth in works on Surgery, general and special. A few suggestions only, from the standpoint of the medical practitioner, will be in place here.

Scrofulous inflammation, in any part, undoubtedly requires and bears less active depletory treatment than that of any other type. Leeching inflamed strumous glands, for instance, is rarely to be thought of; and local abstraction of blood is only likely to do good in a few cases of scrofulous inflammation of the ears, eyes, bones, or joints. Emollient applications are, in the early stages, usually the most suitable. For continued discharge from the ear, lime-water (poured into the ear, gently, rather than injected with a syringe) is an excellent application. Solution of Castile soap; glycerin and rose-water (one part in five); and, if the discharge be copious and obstinate, solution of acetate of lead (one or two grains in the ounce of water), are among the lotions available in this complaint. For an attack of earache, a drop or two of laudanum, with two or three drops of olive or almond oil, or glycerin, may be poured into the ear.

Leaving strumous ophthalmia to the surgeon and oculist for particulars of treatment, mention may be made of my own experience of the benefit obtained from the nightly application of cerate of carbonate of lead on the outside of the eyelids, upper and lower; this treatment being persevered with for a considerable time. White precipitate ointment is an old application, and a favorite with many physicians, in similar cases.

In scrofulous periostitis of the tibia, I have been in several cases surprised with

[1 Vol. II. loc. cit.]

the improvement following the free application of the cerate of carbonate of lead, attended in more than one instance by the subsidence of the swelling, tenderness, and pain, which, in the same patients, had on previous occasions heralded prolonged troubles, with caries and necrosis.

In one case of "cold abscess" of the hypogastric region, attended with febrile symptoms, the application of the lead cerate freely over the abdomen was followed by recovery in about three weeks from the commencement of the treatment. Surgical management of these abscesses need not be here discussed; but reference may be made to Lister's assertion of the safety and advantage of opening them freely in many cases, using the antiseptic method of operation and subsequent dressing.[1]

For coxalgia, the treatment by prolonged rest, by aid of a carved splint, is associated in American practice with the name of Dr. Physick, of Philadelphia. Dr. Henry G. Davis, of New York, claims, apparently with reason, to have been the first[2] to add an important principle to this, in the management of all chronic inflammations of joints, namely, the removal of pressure through separation of the ends of the bones, by *extension* of the limb, or other part affected. Elastic extending bands serve an excellent purpose here; and not only great relief of suffering, but cures, otherwise improbable, have been obtained by skilfully constructed and watchfully used apparatus.

Dr. L. A. Sayre, of New York, has made known to the profession, within a few years, his successful application of this principle in the treatment of Pott's disease of the spine, by a suspending apparatus and "plaster jacket." Dr. B. Lee, of Philadelphia, and others, use a porous felt jacket instead.

For chronic arthritis of the knee, wrist, &c., Trousseau and Dieulafoy have insisted on the great value of the prolonged application of cataplasms. Trousseau's cataplasm is made by preparing a poultice of soaked and steamed bread of a "plum pudding" consistence, over which is spread a liquid mixture, composed of seven parts of camphor, five parts each of extract of opium and extract of belladonna, and alcohol q. s. This is applied to the joint, covered with oiled silk, and secured by a flannel bandage over the whole joint, over which is bound a calico bandage of the same length. It is intended to be left on for eight or ten days at a time.

Returning to Scrofulosis of the glands, mucous membranes, and bones, the advantageous effect of soft soap upon glandular tumors has been asserted by Kapesser;[1] in two cases their disappearance resulting, followed by rapid recovery also from strumous ophthalmia.

Dr. J. Moleschott, of Turin,[2] after an experience with it of many years, has reported that iodoform exhibits remarkable power in promoting the removal of scrofulous glandular enlargements, as well as of other accumulations of "formative elements and exuded fluid." He prefers the application with a brush at night, or night and morning, of a combination of one part of iodoform with fifteen of elastic collodion, or an ointment of the same strength.[3]

Lastly, allusion must be made to the benefit often obtained from massage, with inunction, in the management of general Scrofulosis. Massage alone, while no doubt sometimes beneficial in various conditions of debility, has been, in some quarters, overrated and overdone. But systematic inunction of the whole body, in like states of torpor and debility, has not received full justice on the part of the profession. The wisdom of the ancients has, in this, been too little esteemed. While it is true that no large amount of oleaginous material is absorbed through the skin, experience shows that the disposition to excessive waste is thereby lessened; and this is no small part of the pathogeny of advanced or advancing scrofulous disease. Diminution of general irritability of the system is also thus promoted. Simpson, Inman, E. Wilson, W. Taylor, and other British writers have favored this practice; as well as W. R. Fisher, W. H. Thompson, E. C. Angell, and others in this country.[4] Dr. S. Weir Mitchell, who strenuously advocates massage in cases of nervous debility, encourages inunction with cocoa oil or vaseline.[5] For scrofulous patients, as well as others, olive or cocoa oil may be recommended; although, but for its odor and comparative cost, the preference might be awarded to cod-liver oil.]

[1 Med. Times and Gazette, Nov. 1878.]

[2 Boston Med. and Surgical Journal, August, 1852.]

[1 Berliner Klinische Wochenschrift, Feb. 11, 1878.]

[2 Giornale della Reale Accademia di Torino; cited in London Medical Record, Nov. 15, 1878.]

[3 The odor of iodoform is somewhat unpleasant; but, if used only at night, it can be removed by soap and water in the morning. Its preparations should be protected from the light, which causes their decomposition.]

[4 See the *Sanitarian*, New York, November, 1878.]

[5 Fat and Blood, and How to Make Them. Phila., 1877, p. 54.]

GOUT.

By Alfred Baring Garrod, M.D., F.R.S.

Definition.—1. *Regular Gout.*—A specific form of articular inflammation invariably accompanied with uric acid in the blood, and the deposition of urate of soda in the affected tissues.

2. *Irregular Gout.*—(*a*) The same specific inflammation of non-articular tissues, or (*b*) disturbance of the functions of various organs, accompanied with the same abnormal state of the blood.

Synonyms.—Of *Regular Gout.*—Podagra (πούς, the foot, and ἄγρα, a seizure); Chiragra (χείρ, the hand); Gonagra (γόνυ, the knee): the first only of these synonyms has been much employed. Arthritis (ἄρθρον, a joint), a term used for general gout by the ancients, has been applied also to other joint affections by both ancient and modern writers: Goutte, *French;* Gutta, *Latin;* Gôta, *Spanish;* Gicht, *German;* terms probably derived from the idea of the dropping (Gutta, a drop) of a morbid fluid into the joints—first used in the thirteenth century by Radulphus.

Of *different forms of Irregular Gout.*—Non-articular Gout, Anomalous Gout; Podagra larvata, Goutte larvée, Goutte vague; Misplaced Gout, Retrocedent Gout.

History.—Gout was well known to Hippocrates, and his account of the disease shows that he was well acquainted with many of its salient phenomena; his remarks upon the seasons of the year at which it is most likely to occur, the subjects which it more commonly attacks, the alterations in structures it induces, the probable nature of the malady, and the difficulties experienced in effecting its cure, are well worthy of careful study.

From the time of Hippocrates almost every ancient writer on medicine has made reference to the subject of Gout. Galen speaks of the difference between the times in which he lived and those of Hippocrates, in respect to the character of the subjects afflicted with Gout. Seneca also alludes to the same topic; and accounts of the disease, more or less complete, may be found in the writings of Celsus, Aretæus, Cœlius Aurelianus, Alexander Trallianus, Aëtius, Paulus Ægineta, Demetrius Pepagomenos, and others. Nearly all these authors were humoralists, and of opinion that the disease depended upon the retention of certain matters in the blood (as bile, phlegm, &c.), caused by imperfect digestion or deficient excretion; and that these humors, or even the diseased blood itself, were thrown upon the textures of the joints, and thus gave rise to the production of inflammation, and the frequent formation of tophi, or chalkstones.

Divisions and Classification of Gout.—It is stated in the definition that Gout may manifest itself, simply, in the form of inflammation of one or more joints, or as inflammation of some non-articular structure; or by causing an alteration in the functions of certain organs; and these latter manifestations may either be independent of, or accompany the articular affection.

When the joints are solely or principally involved, the disease may be conveniently designated regular or articular Gout, which may be either acute or chronic; but when severe affections of internal organs ensue, or when inflammation of tissues, other than those pertaining to the joints, arises from the presence of Gout, these affections are known by the name of irregular or non-articular Gout. The whole phenomena of the disease can be conveniently discussed under these two heads.

Description of an Attack of Acute Gout, and of the Progress of the Disease.—Under this heading will be included first a sketch of an early and uncomplicated attack of Gout, as ordinarily met with in practice; next, an account of the progress of the disease when unchecked by hygienic or medicinal means; and this task being accomplished an analysis will be made of the different symptoms exhibited during the paroxysm, and of any structural alteration caused by it. In many instances the first attack of articular Gout comes on without previous warning; or, if there be premonitory symptoms, they are so slight as to pass unnoticed by the patient. This absence of warning, however, is by no means so common as is usually supposed, and I have met with several cases in which the premonitory symptoms have been very distressing; although before the seizure they were not suspected of being the precursors of any joint affection. Under ordinary circumstances, an individual retires to rest in his usual health, but early in the morning, usually from

two to five, awakes with an uneasy feeling, probably confined to one of his great toes; on attempting to place his foot on the ground, he finds himself unable to support the weight of his body, or, if capable of so doing, the act is accompanied with great pain.

If the painful part, generally the ball of the toe, be examined, it is found to be swollen, red, hot, and exquisitely tender, and sometimes to such an extent that the mere weight of the bed-clothes is intolerable, and even the vibration of the room causes discomfort. The veins proceeding from the toe are turgid with blood, and the joint stiff. Although occasionally no constitutional disturbance is present, yet more frequently there is evidence of slight fever; the patient has a feeling of chilliness, followed by heat of skin and perspiration, some thirst and loss of appetite, a white tongue, and confined bowels, with great restlessness, and is unable to find an easy position. The urine is usually small in quantity, high-colored, and deposits, on cooling, a sediment varying in color from pale buff to brick-dust red; occasionally when febrile disturbance runs high the fur which encrusts the vessel is of intense pink color; cramps of the legs are often present during an attack, and add much to the sufferings of the patient.

If moderate precautions are taken, and the foot kept in a horizontal position, the inflammation usually subsides in the early part of the day; but at evening an exacerbation takes place, and for the greater part of the night the patient is kept awake by the pain, which again subsides as morning advances.

After a day, or as soon as the swelling increases, considerable relief is experienced, and in a few more days the tension becomes diminished, as well as the heat and livid redness, and slight sustained pressure will then cause distinct pitting. Subsequently, as the cause disappears, desquamation of the cuticle takes place, and occasionally the skin peels off in flakes of considerable size. Not all cases, even of first attacks, assume this sthenic form; in weakly subjects, and especially in women, the fit may have an asthenic character; the pain and heat may be slight, the redness and swelling by no means well marked, yet as far as ultimate mischief is concerned, this variety is often much worse than the other.

The duration of the joint inflammation varies considerably in different cases, and is much influenced by the diet and regimen adopted, and likewise by the medicines administered. If no material change is made in the diet, and no remedies taken, the inflammatory action seldom subsides under a week or ten days, and occasionally it lasts two or three weeks: but, under more favorable circumstances, the duration of the fit is usually limited to four or five days. After the complete subsidence of the joint affection, the patient not infrequently expresses himself as feeling lighter and altogether better than before its occurrence. It will be seen that the ball of the great toe has been mentioned as the joint especially chosen as the seat of the gouty seizure, and it is a remarkable fact, that a patient may experience repeated attacks of Gout in this one joint, without either the tarso-metatarsal or the phalangeal articulations being in the slightest degree implicated.

It is not an uncommon occurrence for both great toes to be attacked, even in a first fit of Gout, sometimes simultaneously, but more frequently alternately, the inflammation rapidly subsiding in one toe, and as quickly appearing in the other. Sometimes other joints, as the ankle, are affected at the same time as the toes, and occasionally the knees, or more rarely some joints of the upper extremities.

In many instances, some two or three years elapse before the occurrence of the second attack, but in the majority of cases not more than twelve months; and then either the same joint as in the first seizure, or the corresponding joint in the other foot, is usually affected. Similar intervals elapse between the next few paroxysms, and again the same joints are implicated, or the inflammation extends along the foot, involving the articulations of the arch and the ankles.

As time goes on, the disease becomes more general, and almost every joint of the extremities suffer, those of the lower usually taking precedence of those of the upper limbs. The hips and shoulders are perhaps less liable to be attacked than the rest, although they do not necessarily escape. In exceptional cases, other articulations, as the spine and jaw, become the seat of gouty inflammation.

In the course of years, the intervals between the attacks diminish still more—the yearly visitations become half-yearly; afterwards the attacks recur every few months, until at length the patient can scarcely calculate upon being free, so numerous and uncertain are the visitations of his malady.

Phenomena occurring during an Acute Gouty Attack.—It will be interesting to examine a little in detail the phenomena which present themselves during an acute paroxysm of Gout, for our diagnosis must be founded in part upon the peculiarities exhibited at such a time.

1. *Febrile Disturbance.*—The febrile disturbance, indicated by heat of skin, temperature of axilla, thirst, loss of appetite, and rapid pulse, is almost invariably in close relation to the number of implicated joints, and the intensity of the inflammatory action; in other words, the fever is

secondary, and dependent on the joint affection. It is important to remember this fact, because it will be found that in some other diseases — for example, in acute rheumatism—a patient may exhibit all the symptoms of intense febrile excitement, at a time when the joint affection is scarcely appreciable.

2. *Local Appearances.*—The appearance of the inflamed joint is usually characteristic; there is much swelling present, and enlargement of the veins proceeding from the joint, also great tension of the skin. As the inflammation subsides, pressure produces distinct pitting, indicating the presence of œdema. After a further interval, desquamation of the skin almost invariably occurs, usually in a marked degree.

There is a point connected with gouty inflammation which is not without interest—namely, the fact, that however acute in character, it never leads to the formation of pus. An inflamed joint may be intensely red, even scarlet, the skin shining from the distension, and it may altogether exhibit the appearance of suppuration; yet all these symptoms quickly subside, and by resolution merely. When it is stated that a part affected by gouty inflammation never suppurates, it should be added, unless previously the seat of chalk-like deposits; in which case it is not uncommon to find matter formed around such concretions, this formation of pus being probably due not to gouty, but to common inflammation set up around previously existing deposits, which have by their presence acted as foreign and irritating bodies. In enfeebled conditions of the system, such an occurrence is frequently met with.

The pain which attends the joint affection must not be overlooked. It is a common opinion that gouty pain is very intense, a degree more so than that arising from other articular inflammation. Doubtless this is often the case, but it sometimes happens that an acute attack of Gout may be nearly painless, the amount of suffering depending much on the rigidity of the structure of the affected articulation as well as the peculiarities of the patient. The first attack of gouty inflammation in a joint—for example, in the wrist—may be attended with exquisite pain, but in subsequent seizures this symptom may be comparatively slight.

Œdema of the affected part has been specially mentioned among the peculiar symptoms of gouty inflammation; and although it now and then occurs in other forms of inflammation, yet is it scarcely ever absent in Gout. It is owing to the presence of the œdema that so great tension usually accompanies the swelling, and probably the subsequent desquamation of the cuticle is also partly due to it. Not only does effusion occur in the texture of the skin, but, when a synovial membrane is inflamed, a large amount of fluid is generally poured into the joints, or, when bursæ are implicated, they become rapidly filled; this copious effusion frequently causes considerable alteration of shape in the joints.

Joints affected in Gout. — In giving a sketch of the progress of Gout, allusion has been made to the order in which different joints are affected as the attacks become multiplied, and it was then stated that the great toe is commonly selected as the first seat of the disease. The extreme frequency of the selection not only of the great toe itself, but even of a particular joint of this toe, is a fact so peculiar as to make it desirable that a few lines should be devoted to the consideration of the subject.

The joint of the great toe, so commonly the early seat of gouty inflammation, is, as before stated, the metatarso-phalangeal joint, ordinarily termed the ball of the great toe: and from a table collected by the late Sir C. Scudamore, it would seem that in 512 cases of Gout, at its first seizure, the great toe was implicated in 373 cases; and in 341 out of 512 cases, one or other, or both, great toes were affected, to the exclusion of other parts. My own experience fully confirms the general accuracy of these numbers; but it should not be forgotten that this joint occasionally escapes altogether, and cases of severe Gout, accompanied with excessive deposits, have come under my care — cases of at least twenty years' duration— in which the great toes have throughout remained free from disease. The occurrence of inflammation confined to the metatarso-phalangeal joint of the great toe always conveys suspicion of the existence of a gouty habit: but it requires caution before making a diagnosis from this symptom alone, as the joint may be for a time exclusively inflamed in other and more serious conditions of the system. It is not, however, the great toe which is always first attacked; for it often happens that an injury to the knee, caused by a fall from a horse, will induce the first development of Gout in that joint, although after a short time the great toe may be likewise affected. Even an old injury will, as it were, attract Gout to the damaged part, and cause it to linger there longer than in other localities.

It is a very common remark that Gout differs from rheumatism in implicating the smaller articulations of the body. This is doubtless true, if it has reference solely to the earlier attacks, but after a time the larger and smaller joints appear to be indiscriminately affected. It is not uncommon to hear a patient calling his disease Gout as long as it is confined to

the feet, but rheumatism or rheumatic Gout when the upper extremities become attacked, although the same condition of the system which causes the one gives rise to the other also.

There are certain joints of the extremities which appear to be less liable to suffer from Gout than others. Of these the hips and shoulders are the chief: still, it must not be thought that even these joints are unassailable by the disease, for they are sometimes severely affected.

After-effects of Acute Gout.—It has been stated above that, after an attack of acute Gout, especially if an early one, the patient not unfrequently expresses himself as feeling even better than before the seizure, the affected joint recovers, to all appearance, its natural size, the tenderness entirely subsides, and its power of movement is not perceptibly interfered with. To explain this improvement is not difficult. During the occurrence of the inflammation, the blood, as we shall find, loses to a great extent, if not entirely, the morbid condition which previously existed, and hence the disappearance of the malaise; and the joint, although, as will be proved, decidedly altered by the attack, may yet be not sufficiently changed to interfere with its normal functions. This favorable termination is by no means constant, for as the disease continues to make progress, and the joints have been more frequently attacked, some little remaining stiffness is commonly experienced, due in part to mischief which is irremovable, in part to thickening of the tissues and enlargement of the vessels, which are long in recovering their natural condition.

At times, even an early attack of Gout may lead to much mischief; when, for example, the feet are allowed to remain inflamed for any lengthened period, either from want of treatment, or from treatment injudiciously applied, considerable œdema may remain long after all pain and heat have subsided—a state often requiring special treatment for its removal. This result I have several times witnessed in patients who have allowed the disease to run its own course, and also after homœopathic treatment. Occasionally anchylosis of a joint occurs even after a few attacks; in some of these cases it is probable that disease of the joint previously existed, although not in itself sufficient to produce appreciable inconvenience; in others, active and injudicious treatment, as the application of leeches to the joint, has been the cause of the mischief.

Number of Joints affected.—In our description of an attack of acute Gout, we have taken as an illustration a case in which only the metatarso-phalangeal joint, or the ball of a great toe, has been affected. This often happens in the first seizure, and may even occur for several years in succession; but sooner or later, if the disease continues to make progress, not only are other joints implicated, but several are affected either at the same time or in the course of the same attack; and it is not uncommon to find many joints, both large and small, of the upper and lower extremities, simultaneously in a state of acute suffering. When such is the case, as the accompanying fever is in proportion to the joint affection, the patient's malady may easily be mistaken for one of acute rheumatism; and unless the history is carefully inquired into, the diagnosis is somewhat difficult.

Premonitory Symptoms of Gout.—Some of these are referable to an altered condition of the digestive organs; a form of dyspepsia is induced, and the patient experiences flatulence, often to a very uncomfortable degree, accompanied with heartburn and acidity. Many gouty subjects, from the unusual prevalence of these symptoms, can predict the advent of the acute seizure. In some the function of the lower bowel becomes altered, and either constipation or diarrhœa ensues. The character of the alvine evacuations may also be changed.

A crampy state of the muscles is another very common forerunner of a gouty paroxysm, usually in the lower extremities, and more especially in the calves of the legs.

Palpitation of the heart is experienced by some patients on the eve of a gouty seizure, and this may or may not be accompanied with dyspeptic symptoms.

In some individuals the respiratory function is implicated under like circumstances, and a species of asthma produced. At times the urinary secretion undergoes a very visible change in character; from being copious and clear it may become scanty and turbid. On the other hand, the urine sometimes becomes unusually abundant and limpid a few hours before the establishment of the articular inflammation.

In other persons, derangements of some portions of the nervous system are produced, the temper becomes very irritable, unusual drowsiness, or headache, grinding of the teeth during sleep, startings of limbs, and various other phenomena, may be experienced.

Lastly, it now and then happens that a feeling of unusually good health, with apparent increase of both mental and bodily power, is a prelude to a gouty attack.

All these phenomena are probably dependent on the altered state of blood which always exists previously to the development of articular Gout, and the cause of the diversity of the symptoms in different persons must be sought for rather in individual peculiarities, and the proneness of certain functions to be disturbed, than in

any variation in the proximate cause of such symptoms. When any organ or function is implicated in a very marked degree, a form of irregular Gout is established, which we will afterwards describe.

Phenomena of Articular Gout when it assumes a Chronic Form.—In Gout, as in other inflammatory diseases, it is difficult to draw an accurate line between the acute and chronic stages; but it is not difficult to establish a boundary sufficiently well marked for all practical purposes. It has already been remarked, that acute attacks may come on so frequently, and apparently from such slight causes, that the patient can never calculate upon being free; but when, in addition to the frequency of the attacks, their duration is prolonged, and a notable change has taken place in the structures of the articulations, the case has assumed the characters to which the name of Chronic Gout is commonly applied.

It is a somewhat remarkable fact, that although the feet and the joints of the lower extremities are usually the seat of gouty inflammation, often for many years before the hands; still the latter are frequently seriously injured when the feet have as yet escaped appreciable damage. The explanation of this tendency of Gout to cause greater mischief in one part than another, will be attempted when the pathology of the disease is investigated.

Chalk-stones, or Tophi.—The principal changes which take place in parts affected with gouty inflammation, are due to the deposition of a peculiar chalk-like matter in the different structures; and as such deposits are not only peculiar to Gout, but when capable of being seen become a pathognomonic sign of the disease, it will be interesting to describe somewat fully their nature and origin.

White spots often appear upon the helix of the ear, and an opportunity is occasionally afforded of observing the whole train of phenomena exhibited from the commencement to the full development of the little chalk-stone. The earliest appearance presented is that of a small vesicle under the skin of the helix, as if situated between it and the fibro-cartilage; the contents of the vesicle are at first opalescent, or milky, but afterwards become white and opaque, and acquire the consistence of cream. After some months, the vesicle assumes the appearance of a small, hard, and white bead, closely resembling a pearl, and it may remain as such for years; but occasionally the thin skin is worn off, and the bead itself becomes detached from the cartilage, leaving only a slight indication of its presence. If the vesicle is punctured in the early stage, a milky fluid exudes, which presents under the microscope the appearance of a transparent liquid, in which are floating a large number of very fine crystalline needles; if the contents are examined at a later stage, the crystals are found aggregated into small bundles; if the bead is solid, it is difficult to separate them, as they adhere strongly together, and form a closely-interlaced crystalline mass; if, instead of the little chalk-white bodies in the ear, the formation of deposits in other situations is observed, very similar phenomena are exhibited.

It will be found as we proceed that although chalk-stones, or white deposits visible upon the surface of the body, are far from being constant in cases even of long-continued Gout, yet deposition of urate of soda invariably occurs within some of the structures in every paroxysm; and thus stiffening and deformity are often induced. When the deposition is confined to the cartilages, unless very extreme, the injury to the mobility of the joint is comparatively slight; but when the ligaments are infiltrated, they are made rigid, and the play of the parts is consequently seriously interfered with. Much distortion is caused when the bursæ become infiltrated; this infiltration, at times, takes place to an enormous extent; but it may be well to state, that the amount of secretion of urate of soda has no necessary relation to the acuteness of the inflammation.

Without the aid of drawings, it is not an easy matter to give even an idea of the extent of crippling and deformity which occurs in some subjects who have suffered from severe chronic Gout. The hands become greatly altered in appearance: sometimes, when the deposits are chiefly located in the ligaments and tendons, extreme stiffening takes place, without any important amount of bulging; many of the phalangeal joints become rigid and flexed, others equally rigid but extended beyond the straight line or curved backwards: thus the metacarpo-phalangeal of a finger may be flexed, the first phalangeal joint curved backwards, and the second phalangeal articulation sharply flexed; one or several fingers may be thus affected. At other times, not only is there anchylosis of several joints, but likewise great deposition of matter, which causes bulging at different points, from the formation of concretions more or less hardened. In extreme cases, an appearance is presented by the hand very closely resembling a bundle of French carots with their heads forward, the nails appearing to take the place of the stalks; sometimes the toes are affected, though usually in a less degree. The bursæ over the elbows are often distended till they attain the size of small oranges; the bursæ over the patellæ may likewise become enlarged. Deposits of different sizes are found along the shafts of bones, apparently originating in

the periosteum; also on the tendinous sheaths of some of the muscles; in fact, every bursa may be affected, as likewise every tendon and membranous structure, and thus no limit can be set to the deformity which chronic Gout can produce.

When external deposits are visible in any patient, no possible doubt can exist as to the nature of the case, for, as the deposition of urate of soda in the tissues occurs only in Gout, its presence constitutes a pathognomonic sign; but, as before observed, the occurrence of visible chalk-stones is not constant, and it was asserted by Sir C. Scudamore that not one gouty case in ten exhibited them. This statement, however, is not correct, for the small concretions we have described in the ear are extremely frequent, and may constitute for many years, or even during life, the only visible deposition of urate of soda throughout the body; and as they produce no amount of inconvenience, they may altogether escape notice unless specially sought for. Some few years since, having been able in some difficult cases to make correct diagnosis from the presence of these aural deposits, the writer was induced to investigate the matter, and, in thirty-seven cases examined within a short period, they were found to be present in sixteen cases; in seven no other concretions could be seen; in nine there existed deposits around the joints; and in one case only were chalk-stones visible elsewhere without being present in the ears likewise.

Small chalk-like deposits are found in other situations than those above mentioned; sometimes they can be felt under the skin along the tendinous aponeuroses of certain muscles, especially those of the leg and thigh, varying in size from a flattened pea to a small bean; they have been observed on the sclerotic coat, likewise on the tarsal cartilage at the angles of the eyes. It is questionable if they ever originate in a very vascular tissue such as the skin, although it may subsequently become pressed upon and involved.

It has already been stated, that the original condition of these deposits is that of a liquid, rendered more or less milky or opalescent from the presence of acicular crystals; that as the fluid part is absorbed, the consistence becomes creamy, and at last a solid concretion is produced. If the effusion has taken place in a bursa, the resulting chalk-stone is free and of uniform composition; but, if it has been infiltrated in a tissue, the structure of the part becomes mixed up with it when solidification occurs; hence the discrepancies which have arisen in different statements regarding the composition of chalk-stones.

Several analyses have been made of chalk-stones which have either been removed during life, or obtained from the body after death, and from these it will be seen that, omitting the animal matter and the soluble salts derived from the structures in which the concretions have formed, urate of soda is practically the only salt which they contain. Possibly in some instances, as in a concretion analyzed by L'Heretier, the phosphate of lime found in large amounts was derived from the tissue; but it is not improbable that it may be occasionally secreted as the result of common inflammation set up by the presence of the urate of soda, which latter salt has acted as a foreign body, in the same manner as tubercular matter often becomes infiltrated with bone earth.

Gouty Abscesses.—When gouty deposits increase in size and approach the surface, the skin over them becomes gradually thinner, and often gives way; a discharge takes place, either of a white solid substance, should the concretion have been of long standing, or of liquid matter, if the deposit is more recent; but not unfrequently, however, a mixture of both solid and liquid chalk occurs, and thus a gouty abscess is established. Such abscesses are usually difficult to heal, and may remain open for months, and even years; and this is especially the case when the morbid matter penetrates to any considerable depth, as, for example, in the neighborhood of joints; but when in a bursa, as that over the olecranon process of the elbow, this difficulty is not experienced, in fact the healing takes place with as much facility as in the case of an ordinary abscess. The discharge from these abscesses may be unaccompanied with pus, and consist simply of urate of soda; but in exceptional cases, when concretions have become very solid, and the patient is reduced to a very weak state of health, inflammation and suppuration may arise, and pus mixed with white fragments is then freely discharged.

It is not uncommon to find that patients, in whom chalk-stones have been freely formed, have a great number of abscesses discharging at one time, and it is astonishing to see how little disturbance of the system is produced by them. Occasionally five or six such abscesses will be open on each hand, and nearly as many on the feet; the free outlet thus given to the matter appears, in fact, to give relief to the system.

Constitutional Symptoms in Chronic Forms of Gout.—It remains, before completing the description of chronic Gout, to speak of the symptoms which accompany the changes of structure above described. It may be here assumed (a fact which will be afterwards proved) that the blood in chronic Gout is always in an impure state, and we should expect, therefore, that symptoms indicating its irritating action upon various organs would be pres-

ent. This is often the case, and chronic dyspepsia, accompanied with acidity, heartburn, flatulence, pyrosis, and pain after food, are common accompaniments; the bowels are apt to be disordered, and the function of the liver impaired, palpitation and irregular action of the heart may be present, and occasionally symptoms referable to the nervous and muscular systems, as cramps, twitchings of limbs, nervous depression, and so forth. Although the patient is apt to be afflicted with some or even several of the above-named miseries, yet it is not always the case, for it would seem that in many such subjects the system gets gradually accustomed to the impurity of the circulating fluid, and it is only when there is an excess of such impurity that they become sensible of its presence.

The urine of patients suffering from chronic Gout, with extensive deposit of chalk-stones, is generally pale, of light weight, and often contains a little albumen; the occurrence of deposits is rare, except about the time of a paroxysm of a more acute character.

Irregular Gout.—The subject of irregular Gout is one of no small difficulty, and requires careful handling in order to avoid falling into error. Some pathologists appear to assume that all the ailments which happen to patients subject to Gout necessarily owe their origin to that diathesis, and hence the descriptions given of gouty pneumonia, gouty hepatitis, and many other inflammatory affections; but a closer investigation of several so-called gouty complications has led me to believe that they are nothing more than ordinary forms of inflammatory disease, modified, indeed, to some extent by the diathesis of the patient. A man with a gouty diathesis may be exposed to cold, and have pneumonia developed from such exposure, and yet the lung inflammation may not differ in its essential character from what occurs in a previously healthy person. Such a subject is, in fact, as liable to be attacked with inflammatory disease as any other individual; it is necessary, therefore, that good proofs should be shown that a malady is truly gouty in its nature, before we are justified in coming to the conclusion that it is so. On the other hand, it is equally important that, the possibility of a disease being dependent on a gouty state, the habit should not be overlooked, for upon a correct diagnosis of such cases success in treatment mainly depends.

As yet we have only described Gout as implicating the structure of the joints, or at least tissues similar to those of the articulations, and situated near the surface; but it must be remembered that like tissues exist in the deeper-seated parts of the body, and it is a question of interest to know if they often become affected; in the case of acute rheumatism, in which inflammation of the membranes of the heart so frequently happens, such occurrences are looked upon as ordinary phenomena of the disease.

Retrocedent Gout.—There is a popular belief that a patient when laboring under gouty inflammation of any joint, if exposed to cold, is liable to have the local malady suddenly checked, and to be attacked with some acute affection of an internal organ, as the stomach, heart, or brain; and when this takes place, the term Retrocedent Gout is applied.

Gout affecting the Nervous System.—When the brain or its membranes become implicated, the symptoms may be exhibited in the form of intense pain of the head, epilepsy, &c., or the intellect may be impaired and delirium ensue.

Apoplexy has been said to have been caused by retrocedent Gout. If the patient is suffering from any chronic brain disease liable to induce apoplexy it can easily be imagined that the suppression of articular Gout might cause its rapid development. Serous or congestive apoplexy may possibly result from gouty inflammation; but these cases are rare: the author has not yet witnessed one which could be fairly classed under such a head; those he has seen have always been accompanied with albuminuria.

A severe form of headache is not very uncommon in Gout; sometimes it occurs prior to the development of the joint inflammation, and then it usually vanishes at once on the occurrence of the latter, and now and then the alternation between the headache and toe affection is characteristically and unmistakably marked. At times when a patient is suffering from Gout in some joint a cessation of the articular pain suddenly ensues; but this, far from being the termination of the disease, may be followed by delirium, attended with more or less febrile disturbance. This condition may last for hours, days, or even weeks; it may be relieved by the reappearance of the joint disease, or it may gradually disappear without such redevelopment.

When these symptoms arise in the course of articular Gout, to what pathological condition must they be ascribed? It would seem probable that they may be ascribed to the sudden occurrence of gouty inflammation about the membranes of the brain, or the lining of the skull; and, although I am unaware of any trustworthy post-mortem examination proving the existence of deposits of urate of soda in these structures, still such may arise, and the proof of their existence or non-existence is a point of much interest and importance. Deposits of this kind can readily escape notice, unless specially sought for; and even those which occur

in the joints in early attacks of Gout were not observed till within the last few years.

Epilepsy is not uncommon in gouty subjects, and appears to be closely dependent on the diathesis which gives rise to the articular affection; it sometimes distinctly alternates with the joint affection; at others, the two may occur simultaneously.

Mania is frequently the result of retrocedent Gout. I have seen numerous cases: gout often leaves a joint suddenly from exposure to cold or an intense mental shock and after a short time distinct mania is developed; this may last for days or weeks, but generally terminates favorably.

Spinal affections, probably with inflammation of the meninges, with startings of limbs, hyperæsthesia and other characteristic symptoms, occasionally result from the sudden suppression of articular Gout, or occur along with the joint inflammation.

Acute neuralgia of different nerves is at times closely connected with Gout. Sciatica of this character often occurs, and facial neuralgia occasionally. These affections probably depend on gouty inflammation affecting the sheath of the nerves. Forms of local paralysis have also been observed apparently due to the same cause; and cramp sometimes becomes so excessively developed, and so permanent, as to justify its being looked upon and classified as a form of irregular Gout.

Gout affecting the Digestive Organs.—When the stomach is affected by gouty metastasis, which sometimes occurs from the application of cold to the extremities, there is usually sudden intense pain and spasm in the epigastrium, oppression, and vomiting of bilious matter; at times the heart's action is involved, and a feeling of great anxiety, with palpitation, produced. Many of the cases reported as examples of retrocedent Gout will not bear a close investigation; still there is no reason to doubt the possibility of such an occurrence. True retrocedent Gout affecting the stomach is probably of an inflammatory nature, though no direct proof has been afforded in support of this statement.

Sometimes the upper extremity of the digestive tube, as the pharynx and œsophagus, becomes distinctly affected in Gout, producing difficulty of swallowing; at other times the rectum is implicated, and the patient is troubled with piles or tenesmus. Constipation is very common, but, on the other hand, diarrhœa may prevail, apparently of a conservative character, and cases now and then are seen in which articular Gout appears to be warded off by the discharge from the intestinal canal.

Gout affecting the Circulating Organs.—When articular Gout suddenly recedes and the heart becomes affected, the symptoms experienced by the patient are a sensation of extreme anxiety, difficulty of breathing, constriction of the chest, much palpitation, often accompanied with intermission, weak thready pulse, and syncope. The heart's action occasionally becomes exceedingly slow, or it may be unusually rapid.

The subject of heart affection in relation to Gout is one of considerable interest, more so from the fact that the organ is so frequently affected in acute rheumatism. No conclusive evidence has yet been advanced proving the existence of true gouty inflammation of the heart; the examination of the surface, the lining of the organ, and the valves in gouty subjects, has not shown the presence of deposits; but it must at the same time be allowed, that in cases where the heart has been implicated, they have not been specially sought for. It has been asserted that the post-mortem examination of gouty subjects has frequently revealed the presence of white patches upon the surface of the heart, and Dr. Begbie fancies that these are often the results of gouty inflammation; he is also inclined to think that the endocardial vegetations and puckerings are due to the same cause; no proof, however, has been brought forward in confirmation of these views, and the writer of the present article can state positively that his examinations have failed to detect urate of soda, either in the white patches, the endocardial deposits, or the atheromatous spots. It must also be remembered that the causes which lead to the production of Gout are such as are likely to induce chronic valvular diseases of the heart.

Gout affecting the Respiratory System.—Acute inflammation of the lungs or pleuræ, if it ever occurs from retrocedent Gout, is certainly very rare indeed, although some authors have described these forms of disease; but functional affections of the respiratory organs are of very frequent occurrence in gouty habits, and so probably is a form of bronchitis; the most common manifestations of such disturbance being shown by asthmatic breathing and cough. Occasionally these symptoms are unaffected by ordinary remedies, and yet yield at once to the development of articular Gout, or to the administration of medicines calculated to give relief in ordinary Gout.

Gouty Conditions of the Urinary Organs.—The different parts of the urinary tract appear peculiarly prone to be affected by Gout. The kidneys are undoubtedly im-

plicated in very many instances, and structural alterations are frequently produced, which will be described under the "Morbid Anatomy" of the disease.

From many observations and post-mortem examinations, I am of opinion that gouty inflammation of the structure of the kidney is by no means rare, and that a true deposition of urate of soda takes place as its result. That this may occur early in the gouty life of a patient is certain; possibly it may at times even precede the joint affection. It must be borne in mind, when investigating these cases, that renal calculi are somewhat common in gouty subjects, and the symptoms must therefore be carefully analyzed; in irritation of the kidney from a calculus, the pain is more likely to be confined to one side of the loins; albumen is not so frequently present, and if so, it is probably due to a little blood; and, lastly, there is the absence of febrile disturbance.

The bladder and urethra may also become affected with Gout, and a species of chronic cystitis and urethritis induced, especially in old people. Where the irritation of these surfaces is simply the result of this diathesis, the symptoms are greatly relieved or altogether removed when the joints are attacked; but in many instances some organic urinary mischief exists, the symptoms of which are aggravated by the gouty habit.

Gout affecting the Eye and Ear.—Ophthalmia appears to be occasionally of a gouty character, although many of the cases recorded have been the result of purulent urethral affection.

I have recently seen two cases of gouty sclerotitis accompanied with the white deposits of urate of soda on the surface of the tissue.

The occurrence of the little bead-like chalk-stone on the helix of the ear has already been fully described; it has been supposed that the surfaces of the drum and of the ear-bones are at times the seats of like deposits; those I have examined have consisted simply of bone earth, and have not occurred in gouty subjects.

The external ear is at times painfully affected by acute Gout, even to the extent of preventing the patient from resting on it when in bed.

Gouty Affections of the Skin.—If inquiry is made it will be found that skin eruptions are very common in those who are the subjects of Gout, and if they are not absolutely produced by the state of the system which leads to the articular affection, still they are evidently kept up by it.

Psoriasis is, perhaps, the most frequent form in which the cutaneous disease manifests itself, and there are records of many cases in which the skin and joint-affection are alternated.

Eczema is likewise not an infrequent accompaniment of the gouty diathesis; sometimes it assumes an acute, sometimes a chronic character.

Prurigo is also met with in connection with Gout, either in the limited form of prurigo ani, or as a more general affection.

Acne, in the face and other parts, is sometimes found to be closely dependent on the gouty diathesis, and I have known one case in which the patient could predict the advent of a gouty paroxysm from the appearance of these spots.

Diseases occurring in Gouty States of the System.—There are certain diseases to which gouty subjects are especially liable, and amongst these gravel and calculus may be particularly mentioned.

When the pathology of Gout is considered, the occurrence of uric acid gravel cannot be a matter of surprise; from a very early stage deposits of urate of soda take place in the tubules, and Dr. Prout has remarked that occasionally patients void this salt in considerable quantities. Some patients suffer in early life from calculus, in after periods from Gout; in other subjects calculus and Gout alternate, and occasionally the two affections are present at the same time.

Oxalic acid, which is so readily produced from uric acid, not infrequently occurs in the urine of gouty subjects, and may lead to the formation of calculi.

The existence of a gouty diathesis has been regarded as antagonistic to the development of phthisis; I have, however, seen instances in which phthisis and Gout have run a simultaneous course.

Gout and diabetes occasionally occur in the same individual, but the development of the latter is usually followed by the cessation of the former disease.

Condition of the Blood in Acute and Chronic Gout.—The blood undergoes important alterations in Gout, changes which are almost in themselves pathognomonic and which require to be carefully studied and clearly understood. In the writings of the ancients, nothing definite is found on this subject; and even until the past few years our knowledge of the state of this fluid in Gout may be said to have been of little value.

The blood-corpuscles, as far as yet known, undergo no necessary change either in number or quality; they become lessened in chronic forms of disease, when the general nutrition of the body is impaired, but not more so than in other maladies. Many of the poorer subjects of chronic Gout, it is true, are pale and anæmic, and among painters and other gouty patients who work in lead, this is often due more to the influence of the metal which has been imbibed than to any other cause.

It is in the serum of the blood that the chief deviation from the healthy standard is discovered; and in this portion it is not so much that the normal constituents are affected, as that excretory substances which should have been eliminated are retained—an effect due to the imperfect action of certain of the excreting organs, more especially the kidneys.

In healthy blood, it is impossible by ordinary tests to discover the presence of uric acid, the quantity being so extremely small—in fact almost inappreciable; but in Gout one can easily not only show its presence, but even obtain it in a crystalline form. It was first proved to be present by the author in 1847.[1] The following process can be adopted for its detection:—The serum of the blood is first dried over a water-bath, then reduced to coarse powder, and treated with hot alcohol; the spirit being removed, the residue is afterwards to be digested for some minutes in distilled water, and raised to the boiling point; the watery solution is then filtered and evaporated to a thin syrupy consistence. A drop or two of the solution, when heated on a piece of porcelain, with nitric acid and ammonia afterwards added, exhibits at once the murexide test. A small portion of the same solution, if acidulated strongly with acetic acid, and allowed to evaporate spontaneously, gives rise to the crystallization of uric acid, the crystals exhibiting its characteristic form; and lastly, the syrupy solution, if merely allowed to evaporate without the addition of any acid, exhibits upon its surface, after a few hours, small white tufts of acicular crystals of urate of soda; the nature of the base being determined by the examination of the white alkaline ash left after incineration; the acid by the murexide and other tests.

In the clinical examination of the blood, this process would be too elaborate and tedious; but another method,[2] which answers admirably for practical purposes, is to put about two drachms of the serum in a flat glass dish, somewhat larger than a watch-glass, acidulate slightly with acetic acid, and having placed in the fluid an ultimate fibre from a piece of linen cloth (unwashed huckaback answers well), set it aside in a safe place until the evaporation has proceeded sufficiently far to cause it to become of a gelatinous consistence. If there is uric acid in any abnormal quantity in the serum, the fibre becomes studded with crystals of uric acid, which can be at once recognized by placing the glass under the microscope with a low power, or by the use of a small pocket magnifying-glass. I have never yet, after very numerous trials, failed to discover uric acid in the blood of gouty patients by this method, and the test has an especial advantage in only requiring the abstraction of a very small quantity of so important a fluid. It may certainly be stated as a fact, absolutely proved, that the blood in Gout always contains an abnormal amount of uric acid, and that this acid exists as urate of soda. Besides uric acid, urea is frequently found in varying quantities in the blood in this disease, especially when the affection is of long standing, and the kidneys have become much involved and their excreting powers impaired.

In 1849[1] the writer discovered the presence of oxalic acid in the blood, and since that time has, in several instances, detected it in gouty subjects; its presence appears due to the decomposition of the retained uric acid.

The other alterations in the serum of the blood occasionally met with in cases of chronic Gout, are, the lessening of its specific gravity, due to the loss of albumen, as well as to the impaired nutrition of the body; and the diminution of its alkaline reaction. With the exception of collapsed cholera, and perhaps certain cases of albuminuria, the reaction of the blood is found to be nearer the neutral point in severe forms of chronic Gout than in any other disease; this lessening of the alkaline condition probably depends on the deficient action of the kidneys and the retention of acid products, as it is only when the kidneys are much affected that such a state of the blood is observed. The amount of fibrin is always increased when active inflammation of the joints is present, and the augmentation appears to be in the ratio of the inflammatory action; in fact, the fibrin follows the same laws as in other forms of inflammation; hence, in acute Gout, if blood is abstracted, the surface of the clot exhibits a buffed and often a cupped appearance.

Condition of the Blood in the intervals between the Attacks of Gout.—It is a matter of some importance to ascertain the state of the blood in gouty subjects, when no inflammatory action is present; that is, during the complete intervals of the attacks. Although a somewhat difficult task, from the unwillingness of patients to be bled when not suffering from pain or fever, I have been enabled, in a few instances, to collect some facts of importance on this point.

1. In the intervals between the early attacks of Gout no appreciable amount of uric acid was found in the blood.

2. A very marked decrease of uric acid was observed in the blood of patients partially recovered from an acute attack.

[1] Medico-Chirurgical Transactions, 1848.

[2] Medico-Chirurgical Transactions, vol. xxxvii. 1854.

[1] Ibid. vol. xxxii. 1849.

3. In chronic Gout the blood, even in the intervals between the exacerbations, was always rich in uric acid.

4. In some cases when symptoms of irregular Gout were manifested, without any accompanying joint disease, uric acid was present in the blood.

State of the Urine in different forms of Gout.—Much error prevails in regard to the alterations which the urine of gouty subjects exhibits during the progress of the disease, and such misunderstandings have partly arisen from the prevalent idea, that the appearance and non-appearance of certain principles in the urine necessarily indicate their presence or absence in the blood; and partly from a want of accurate knowledge of the reaction of healthy urine.

It must be remembered that healthy urine exhibits a strongly-marked acid reaction, and that this is, in all probability, due, not to the presence of a free acid, but to the existence of an acid phosphate, probably the phosphate of soda, a salt containing two equivalents of water and one of soda to each equivalent of tribasic phosphoric acid ($2HO, NaO, PO_5$) [$Na_2HPO_4.12H_2O$]. The acidity of urine varies much at different times of the day, and is in close relation to the state of the digestive functions. The fact of the acidity of healthy urine has been specially insisted upon, because the application of litmus papers is not infrequently made both by medical men and patients, and very wrong deductions drawn from the indications thus obtained.

It must be remembered that a strong acid reaction is no proof of the presence of any abnormal state of the urine; and that urate of soda is one of its constituents, a salt which can exist in a solution of the *acid* phosphate without decomposition, and hence the possibility of the coexistence of the urate of soda with an acid condition of the urine.

The amount of uric acid passed by a healthy subject during twenty-four hours is from eight to ten grains—a quantity capable of existing in solution when the urine is in a healthy state.

Having premised thus much, we are better prepared to speak of certain changes which are observed in the urinary secretions in Gout, changes the import of which has been often misunderstood. It has already been proved that the blood in all stages of Gout is invariably rich in uric acid, and, as the kidneys are the only organs which can be shown to eliminate this body, it follows that these organs must be inefficient for their required task when such a condition of the blood ensues. It becomes, then, a matter of some interest to discover whether these organs in gouty cases lose any of their normal power of excreting uric acid, or whether the formation of this acid in the system is increased.

In acute gouty seizures of a sthenic character the urine usually becomes scanty; it is then high-colored, and not infrequently gives rise, in cooling, to an amorphous deposit, varying in tint from pale yellowish red to dark red, or at times an intense pink. The color varies under different circumstances, such as the amount of febrile disturbance which is present, the state of the portal system, and that of the biliary secretion also. If a judgment is formed from the inspection of a small specimen, without taking into consideration the limited amount passed in the twenty-four hours, it may readily be supposed that a great excess of uric acid is secreted; and such, in fact, has been the prevalent idea. However, a more careful examination of the urine in these cases leads to a different conclusion; for example: in a set of observations, taking the averages in seven acute cases, the daily secretion of uric acid was found to be under four grains, an amount far below the normal amount, which is from eight to ten grains. The cause of the prevalent idea that in Gout the uric acid exists in the urine in excess has probably arisen from the following circumstances: During the febrile stage there is usually a deficient flow of urine, the acidity of the fluid is augmented, thus causing the precipitation of the whole of the urates; at the same time the increase of coloring matter in the deposit gives the appearance of a larger amount than actually exists.

It must not be concluded from the above that in any given amount of urine passed in a gouty paroxysm there may not be a larger amount of uric acid than that which is found in health—often it is so; but our remarks apply not to the relative, but to the absolute weight eliminated in the twenty-four hours.

The question whether the deficient excretion of uric acid is due to a loss of renal function, or to a diminished formation of the acid in the system, is at once determined; for it can be demonstrated that at the time that the urine is deficient in this principle, it exists in the blood in abnormal quantities. It is also known that in some other diseases, in which uric acid is formed in augmented quantities, the kidneys are found equal to the task of eliminating such excess, and the blood is thus kept free from any contamination.

The excretion of the urea also is probably somewhat diminished during the febrile excitement of acute Gout, but by no means in the same ratio as the uric acid.

As Gout assumes more and more a chronic character, so it will be observed that the excretion of uric acid becomes gradually lessened, even in the intervals between the attacks; showing that the kidneys are permanently injured, so far

as their uric-acid excreting power is concerned; and in the advanced stages of the disease, when the chalk-like deposits are thrown out in various parts of the body, and when the blood is permanently loaded with the acid, the renal organs often lose entirely, or all but entirely, their eliminating property for this principle. The results obtained from a large number of examinations of urine, and in numerous cases of chronic Gout, may be thus summed up:—

The urine is generally paler than in health, lighter in specific gravity, and often passed in augmented quantities.

There is, for the most part, an absence of any deposits on cooling; at times, however, such may occur, especially when an exacerbation of the disease is passing off. The quantity of uric acid eliminated in the twenty-four hours is notably diminished, and not infrequently it is reduced to a mere trace.

The kidneys sometimes throw out uric acid in an intermittent manner; for example, for several days the uric acid gradually decreases, until scarcely a trace is present, then suddenly a large elimination takes place; this has been clearly made out in several cases.

Even in the intervals between the fits, the urine of patients suffering from chronic Gout is deficient in uric acid.

The urea also, in many cases, is slightly diminished, but not in any remarkable degree, if the diet of the patient at the time of examination is taken into account.

A small amount of albumen is very frequently met with, also granular casts; and, in some cases, waxy or fibrinous casts are likewise found.

Secretion from the Skin in Gout.—It is a common opinion that the production of excessive perspiration is the means of getting rid of gouty matter from the system, and the value of hot-air and vapor baths, and other modes of increasing the cutaneous secretion, have been thus accounted for. There are also to be found, in different works, statements to the effect that uric acid is capable of being eliminated by the skin, and that a white, powdery matter is occasionally seen upon the surface of patients suffering from a paroxysm of Gout. I have made many observations upon this subject, which have led me to doubt the accuracy of such statements, and within the last two years have had an opportunity of putting the question to a severe test. A gentleman, suffering from a very decided attack of Gout, went into a Turkish bath, and took precautions to enable him to save a considerable amount of the perspiration which flowed from his body during the operation. To this fluid rectified spirit was at once added, in order to prevent decomposition, and it was afterwards carefully examined by the following process: It was first evaporated to dryness, then heated with absolute alcohol, and the residue afterwards treated with hot water. The alcoholic solution yielded a notable amount of urea, which was obtained as the crystallized nitrate; but no trace of uric acid could be discovered in the watery solution by the most careful search. This observation, coupled with many others above referred to, leads me to believe that the healthy skin does not possess the power of eliminating uric acid, even when it exists in normal quantities in the circulating fluid. It must, however, be remembered that the liquid thrown out from blistered surfaces in like cases is rich in uric acid, and it is not improbable that in some cutaneous eruptions, as in the secretion in eczema when occurring in gouty habits, it might be detected. Allusion has been made to a white deposit seen on the surface of patients suffering from Gout; on one or two occasions, when I have had the opportunity of examining such matter, it has been found to consist of epidermic scales, together with dry salts, but to be entirely devoid of uric acid.

Other Secretions in Gout.—It has not been determined whether uric acid is contained in the secretions from the mucous membranes of the bowels in subjects laboring under Gout, and, when evidence of its presence in the blood is beyond doubt, it would be worth seeing if it is contained in the watery excretions produced by the action of saline or other purgatives. In morbid secretions uric acid is often present: thus it is found in the fluid effused in cases of pericarditis, also in ascites, when these diseases occur in subjects in which the blood is contaminated with this principle. It is also found under like circumstances in the fluid effused by the action of blisters, and we can sometimes make use of this fact in diagnosis, employing the blister fluid in lieu of the blood serum. There is, however, one precaution necessary to be observed, which is, to avoid taking the fluid from a part affected with gouty inflammation, as it would appear that inflammation has a tendency to destroy the uric acid in the blood of the part. This fact, if well established, would be of great value in elucidating the pathology of the disease.

The thread experiment may be employed for the detection of uric acid in the blister fluid, as in blood itself.

MORBID ANATOMY OF GOUT.—Gout is seldom fatal, yet opportunities for investigating the morbid appearances produced by the occurrence of the malady, even in its slightest forms, are not difficult to obtain, provided that the gouty history of

cases be carefully taken for a considerable period of time in any great public medical institution. It is only by this method that the writer has been enabled to collect the materials of which he now gives a short summary and which he hopes will go far to elucidate many of the phenomena of the disease.

In one case there had been only two very slight attacks of Gout, affecting the metatarso-phalangeal joint of the right great toe, the first attack occurring two years, the second only one year, before death. The condition of the joint was as follows: On the head of the metatarsal bone was a white patch made up of minute aggregations of a white deposit, occupying altogether about a tenth of the articulating surface; on the cup-shaped surface of the phalanx, the same sprinkling of white matter was observed upon the cartilage, occupying a greater extent of surface than on the metatarsal bone; on the inner surface of the ligaments the same substance was here and there observed; the surfaces of the sessamoid bones were free from any deposits, and, although the same joint on the left side, as well as many other small and some large joints were examined, no deviation from the normal state was observed in them. The gouty attacks, it will be remembered, had been confined to one articulation, and this joint only was found to be altered after death.

In another case, only one attack of Gout had occurred, affecting the right great toe very severely, the left but slightly; and similar appearances in the cartilages and ligaments were discovered, but exceedingly slight in the left toe-joint. In this case, also, several other joints were examined, but found to be free from any morbid alteration.

We thus see that even a single attack of Gout leaves marks behind, which appear to be very nearly, if not altogether, indelible; for, in the second case, at least thirteen years had elapsed from the time of the gouty seizure to the death of the patient.

Before proceeding to speak further of the morbid appearances produced by gouty inflammation it will be necessary to point out the nature of the alteration we have already noticed—that is, the nature and situation of the white deposit. When the alteration in the joint is slight, no appreciable elevation of the surface can be discovered; and if we pass the finger over it, nothing abnormal is detected, nor can we remove the deposit by moderate friction, nor, in fact, by any means short of removing the surface of the cartilage itself. On the other hand, if we immerse the bone for some hours in water at the temperature of the body, or keep it in a cold and weak solution of carbonate of potash, the white matter is slowly dissolved out, and on afterwards drying the bone, the articulating surface appears to be restored to its healthy condition. We can thus show that the deposit is not on the surface of the cartilage, but within it, and that it consists of a material soluble both by warm water and in a weak alkaline solution. A further insight into its nature may be obtained from a microscopical and chemical examination; if, for example, we make a vertical section of the cartilage over the seat of the white deposit, we at once see, either with the naked eye or a simple lens, that the infiltrated matter is most dense near the free surface, and gradually diminishes as it approaches the bone, seldom, indeed, extending half-way into the substance of the cartilage.

If we place the thin vertical section under the microscope, using a quarter-inch objective and low eye-piece, a very beautiful appearance is exhibited; the opaque white matter is observed to consist of very fine crystalline needles or prisms closely interlaced, and according to the density of the network, so is the amount of opacity produced. As the deposit becomes more sparse, many separate crystals are seen, which appear to project into the substance of the healthy cartilage. If we examine horizontal, in lieu of vertical, sections of cartilage, we find that, after we have removed a few slices, the deposit becomes sufficiently thin to allow light to pass freely through it, and the crystals are for the most part seen to be arranged in little clusters radiating from centres, the interspaces being nearly free, or with only scattered crystals. If, instead of using ordinary light, we employ the polariscope, the appearances above described are much intensified, the crystals become strongly illuminated, and more or less colored; at the same time, the healthy portion of the cartilage, if sufficiently thin sections be made use of, gives a black background.

The chemical nature of the deposit can be readily demonstrated. If slices of the altered cartilage be first washed with a little cold distilled water, to remove any soluble matter, and afterwards digested for some hours in hot water under 200° Fahr., the infiltrated matter is dissolved, and a solution is obtained which, treated with a little nitric acid and afterwards evaporated in a porcelain capsule, and when nearly dry exposed to the vapor of ammonia, exhibits an intense purple color from the formation of murexide; on the other hand, if some acid, as acetic, be added to the solution so as strongly to acidulate it, and the whole allowed to evaporate spontaneously to a thin syrupy consistence, crystals of uric acid, more or less colored, are slowly deposited, and the

forms they exhibit under the microscope at once distinguish them; again, if the watery solution, without the addition of any acid, be slowly evaporated to a syrupy consistence, and then allowed to cool, urate of soda, in bundles of crystalline needles, will form on the surface, which can be readily collected, and, if necessary, chemically examined.

If a thin vertical section be obtained from any articular surface which has not been much worn—that is to say, from a joint which has not been often attacked, or is not liable to injury from friction—a distinct organic layer devoid of crystals can be seen by the aid of the microscope, superficial to the deposit, and probably consisting of flattened cartilaginous cells.

As yet we have only spoken of very early cases of Gout, in which merely the ball of the great toe has been attacked; if we take more advanced cases, where the disease has lasted for several years, and in which many of the larger as well as the smaller joints have been implicated, opportunity is afforded of investigating still further the changes effected by the disease.

If a knee has been but slightly attacked, only a few spots or small patches can be detected upon the articulating surfaces of the femur, tibia, and patella; but if it has been severely affected, a large portion of the surfaces may exhibit the peculiar alteration; the condyles of the femur are often completely incrusted, except at their margin, in the situation of the synovial fringes, the vascularity of which appears to protect this part from the deposit;[1] the concave surface of the tibia is usually less covered; the patella is often extensively coated; and in many instances the fibro-cartilages and crucial ligaments are more or less implicated. In such a knee-joint the synovial fluid is thickened, and in extreme cases contains tufts of urate of soda; the synovial membrane is also seen to be speckled with little white points looking like amorphous granules. When these are placed under the microscope, they are found to consist of acicular crystals radiating from a centre, and forming with polarized light a very beautiful object. In cases of Gout in which the disease has so far advanced as to cause considerable stiffening or complete anchylosis, the ligaments are found to be much infiltrated and thereby rendered rigid; and, as a rule, the movement of the joint is impeded more by the ligamentous than the cartilaginous alteration. Instances are not uncommon in which the movements of the great toe-ball are but partially interfered with, although the ends of the bones are completely covered; but the ligaments in such cases have escaped.

The shoulders and hip-joints often remain free from disease when most of the other articulations of the body are implicated, but occasionally they present similar appearances to those above described. I have a specimen in which the head of the femur is almost completely incrusted; the ligamentum teres, however, is free, and the movements of the joint are consequently preserved.

The carpus and tarsus are often severely attacked by gouty inflammation, and it is not unusual to find every articulating surface of these bones completely covered with the deposit; also the surfaces of the metacarpal and metatarsal bones, and frequently of several of the phalanges. There is, however, one point of interest in relation to the joints of the great toe, worthy of being borne in mind; it is the fact that, although the metatarso-phalangeal articulation is so constantly affected, the tarso-metatarsal and the phalangeal joints on either side are for the most part free from morbid alteration.

Sometimes urate deposits are found in other articulations. I have met with them even in the arytenoid cartilages, but it requires a special search, not usually made in post-mortem examinations, to discover their presence.

It has been asserted that urate deposit has been seen in bone itself; Cruveilhier found it in the astragalus, os calcis, and patella. Although I have carefully searched for it in bone, I have not yet succeeded in finding evidence of the deposit having originated in this tissue. It is true that deposits lie in contact with bone, as they often originate in the periosteum, and sometimes acquire sufficient size to press on the osseous tissue and cause its absorption. Dr. Charcot, in conjunction with M. Cornil, has recently (1864) published an account of the post-mortem appearances in the case of a female, aged 84, who had suffered from Gout for many years, and had been long crippled and deformed; a full description of the appearances presented to the naked eye is given, as well as those seen in the microscopic examination; in every important respect the results are the same as, and confirmatory of, those above described.

Condition of the Kidneys in Gout.—From the earliest time it would appear that an idea prevailed that Gout and renal diseases were in some way allied; Aretæus held that opinion, as also Sydenham, Morgagni, and others. The idea was derived from the fact that gravel and calculi are so frequently met with in gouty subjects.

In the early stages of Gout, should an opportunity be afforded of examining the

[1] Dr. W. Budd, Medico-Chirurgical Transactions, 1855.

kidneys, little or perhaps no alteration will be observed, but occasionally an appearance indicating the action of the disease may be presented to the eye, and the first change usually noticed is found to be due to the occurrence of some crystalline deposits in the organ. In 1849, the writer, when examining the kidneys of a man who had suffered from Gout, but who had died of another disease, found small white streaks which appeared to follow the direction of the tubes of the pyramidal portion of the organ; he also discovered that at the extremity or mamilla of each cone there were certain white points;—Dr. Todd and Mr. Ceeley had previously noticed this fact.

On placing a little piece of the substance of the kidney under the microscope, the above-mentioned white streaks and points were found to be caused by the presence of numerous crystals, prismatic in shape, and consisting of urate of soda, that is, identical with those which form ordinary chalk-stones, and which are found in cartilaginous and ligamentous tissues, but the crystals are usually larger. Shortly afterwards, the same condition of kidney was discovered in numerous other subjects, some of whom had been but slightly affected with Gout, and in one only eight seizures had occurred, and no external deposition or deformity had been produced. At first it was thought that the white streaks were due to the blocking up of the uriniferous tubes, but afterwards the conclusion was arrived at that many of the crystals were imbedded in the structure of the tubes themselves. M. Charcot, in an excellent paper, entitled "Contributions à l'Étude des Altérations anatomiques de la Goutte, et specialement du Rein et des Articulations chez les Goutteux," has investigated this subject very thoroughly, and has given good drawings: he comes to the conclusion that, as far as the kidneys of the subject examined by himself were concerned, the deposit, which at first sight appeared to be situated in the intervals or between the tubes, was in reality in part contained within them, and in a great measure amorphous; there were, however, crystals not within the tubes which appeared to radiate from the above matter into the intertubular structure.

It would appear from the observations which have been made both in this country and abroad, that even in the early stages of Gout the kidneys become implicated, probably in some cases much sooner than in others: that deposits of urate of soda take place in the tubes, which afterwards extend into the renal tissues.

Causes of Gout.—The causes of Gout may be conveniently treated of under two heads; first, those depending on the age, sex, hereditary and other peculiarities of the individual; secondly, those independent of the affected subject.

Causes dependent on the Individual.

Hereditary Influence.—Some individuals are undoubtedly more disposed to Gout than others, and in such the disease is liable to be induced by agencies which would be comparatively harmless if applied to other people. There is, in short, a proclivity to Gout which may be inherited; and it is certainly true as regards this malady, that the sins of the fathers are visited upon the children to the third and fourth generation. Instances illustrating the hereditary predisposition to Gout are so frequently met with, that no one who has had the least acquaintance with this disease can have failed to have observed it. My own experience would show that more than half the gouty subjects can distinctly trace their ailment to hereditary taint; and if patients in the upper class of society are exclusively selected, the percentage is found to be considerably greater. There exists a popular idea that Gout frequently skips over a generation, and that it has a peculiar tendency to attack the grandchildren rather than the children: this idea I believe to be erroneous, but at the same time acknowledge that there are often apparent grounds for it. Occasionally the child of a very gouty patient, having the fear of suffering before his eyes, will live such a life as to keep the disease at bay; his children, however, may be fully under the hereditary influence of their grandparent and liable to a development of the malady from the ordinary exciting causes. When true Gout is met with at an early age, the existence of hereditary taint may be suspected, and in the case of children this cause is certain to be powerfully operative. However, it must not be forgotten that Gout may be acquired at a comparatively early age, by the influence of causes other than that of hereditary predisposition.

[Modification of the gouty diathesis often occurs in the course of hereditary transmission. The children or grandchildren of a person subject to regular Gout, may have it in its "flying" or irregular forms; or they may have gouty neuralgia; or a proclivity to attacks of indigestion, which may be called gouty dyspepsia. Another modification not sufficiently appreciated by systematic writers, is *gouty rheumatism:* an affection which may involve the digestive apparatus as well as the larger joints, muscles, and fibrous tissues, in attacks which differ more or less from typical rheumatic fever; which are

attended usually by symptoms of indigestion; and which are especially more responsive to the remedial action of colchicum, than is the case with ordinary articular rheumatism.—H.]

Influence of Sex.—Men are much more frequently the subjects of articular gout than women, and the causes of the comparative exemption of females is not difficult to understand, when we are acquainted with the pathology of the disease. As far as hereditary causes are concerned, women are similarly circumstanced with men; but there are many reasons explaining the immunity enjoyed by women. Some of these are intimately connected with the functional peculiarities of the female sex; others depend upon the habits of life more commonly adopted by women. The occurrence of the catamenia during a long period of female life is doubtless a great safeguard against the disease, and, as a rule, whenever Gout does occur in the female, it is only after the cessation of this function. There are, however, striking exceptions now and then met with—instances of the most severe Gout attended with great crippling and deformity, in comparatively young women; these cases are extremely rare, and most of them capable of explanation.

Influence of Age.—Children are usually free from Gout; and, although I have been assured of some being attacked when very young, yet, in every case where there has been an opportunity of strict investigation into the nature of the affection, there has been good cause to distrust the accuracy of the diagnosis which had been made. Many patients have informed me of their having had Gout when at public schools, and I have no reason to doubt the correctness of their statements, more especially as they have afterwards become the subjects of Gout in its severe forms; I have myself seen the disease fully developed in the great toe at the age of sixteen, but always in youths who have strongly inherited the affection, and who at the same time had not been altogether abstemious. As a rule, the stronger the hereditary predisposition, the earlier Gout develops itself in any individual, and it is rare to find it before the age of thirty unless some well-marked hereditary predisposition exists.

From a table made by the late Sir C. Scudamore from a large collection of cases it appears that, dividing the period of life from twenty to sixty-five into intervals of five years, by far the greater number of first attacks occur from thirty to thirty-five years of age; below twenty they are exceedingly rare; and after sixty-five they are likewise very few in number. As there appear to be exceptional cases in early life, so also are there in old age, and I have known Gout make its first appearance after the age of eighty, and in one instance when the patient was in her ninetieth year.

Influence of Temperament.—Little that is positive can be asserted in regard to the influence of what is called the temperament of the individual upon the development of Gout. The more acute varieties of Gout are usually found in those of a sanguine temperament and full habit of body; whereas the asthenic and irregular forms occur chiefly in spare subjects of a nervous temperament.

Causes independent of the Individual.

Alcoholic Beverages.—No one who has paid attention to the clinical study of Gout can doubt the influence of alcoholic drinks, both in laying the foundation of the gouty diathesis, and likewise in exciting attacks of the disease; and, moreover, no one who has carefully analyzed the causes of this malady can fail to perceive that different kinds of spirituous liquors differ greatly in their power in this respect. This subject is of so much importance that we shall not hesitate to dwell shortly upon it. Distilled spirits have certainly less tendency to induce Gout than either wine or malt liquor: the truth of this remark can be proved by investigating the cases which occur in the large cities of England, and, more clearly, by noting the prevalence of the disease in other countries where little of any other spirit is taken. Among the laboring classes of London, Gout is very frequently met with; whereas, among the same class in Edinburgh and Glasgow, it is scarcely ever seen: the former partake largely of porter and beer, the latter almost entirely of whisky, and in no inconsiderable quantities. The same fact is illustrated in the almost entire absence of Gout in many cities on the Continent, especially in Poland and Russia, where a distilled spirit is almost exclusively made use of.

As predisposing to Gout, I cannot from experience say whether all distilled spirits are equally innocuous: brandy, whisky, and gin have certainly little predisposing power: rum has been asserted to cause Gout in the West Indies, but the statement is old, and there is no good clinical evidence in support of it.

In countries where the lighter kinds of wine form the chief or sole alcoholic beverage of large classes of the people, the occurrence of Gout is comparatively rare: as, for example, among the working population of France, most parts of Germany, and, I believe, also of Italy; but it is asserted that in certain parts of Germany, as in Berlin and Munich, where malt liquor is largely consumed, the disease is much more prevalent.

It must not be thought, however, that these light wines can be taken with impunity, for although their gout-producing tendency is small, compared with some of the stronger wines and malt liquors, still it is very decided. Light claret, hock, and moselle are probably the best of light wines.

The stronger wines, as port, madeira, and sherry, probably also marsala, are much more potent as gout-producers, and a free indulgence in their use for several years will very often bring on the disease in those not known to have derived any taint from their ancestors. Port enjoys a very marked reputation in this respect; it is doubtless as we receive it from Portugal, a wine very likely to cause Gout—perhaps more so than any other. Sherry, however dry and pure, is by no means the innocent beverage, as far as Gout is concerned, that many people imagine; I have met with several cases of severe Gout brought on solely by this wine, and have also known attacks of the disease kept up for an almost indefinite period by the patient continuing the use of it, even in small quantities. Madeira is fully as injurious as sherry to the gouty subject, and doubtless, if freely partaken of for any length of time, would be capable of inducing the malady in those not previously disposed to it. The same remark applies to several other wines, possessing characters closely allied to those of the abovementioned class.

That malt liquors predispose strongly to the production of Gout is made evident by the frequency of this disease among the laboring classes and artisans of the large cities of England, where porter is so freely indulged in: thus Gout is very common with brewers' men, ballasters, and many others. Even the pale bitter ales, though to many so grateful and useful, will, when too freely partaken of, give rise to the development of Gout, and several cases in which such ale was the sole cause have occurred in my practice.

It only remains for us to investigate the influence of one other alcoholic drink—that is, cider—which forms so favorite a beverage in many of the counties in England, more especially in Herefordshire and Devonshire; also, of the United States of America. Concerning its gout-producing powers many and diverse opinions have been held. Dr. Wood of Philadelphia informed me that, as a predisposing cause of Gout, cider cannot be very potent, else the disease would have been more prevalent among the people of New England and the Middle Atlantic States, where it is very commonly drunk. Having taken some pains to investigate the question in this country, the results I have arrived at are as follows:—

1st. That fully fermented cider—that in which the whole of the saccharine matter has been got rid of, and which constitutes genuine rough cider—has but little power in inducing the gouty diathesis.

2d. That sweet and partially fermented cider, when taken in large quantities, predisposes to Gout.

3d. That the latter variety, and even the former, when taken by gouty individuals not much accustomed to its use, is apt to excite an attack of the disease.

Having enumerated the relative powers of different alcoholic liquors in common use in this and other countries, in inducing Gout, it becomes a question of interest to endeavor, if possible, to get some clue as to the cause of such differences; or if unable to accomplish this, at any rate to point out the known peculiarities of each class of such beverages.

The distilled liquors consist of alcohol, more or less diluted, and combined with very small amounts of volatile oils or ethers: thus, brandy contains œnanthic ether, the peculiar principle of all wines; gin, a little oil of juniper; and so on. They should not hold in solution any non-volatile substances, and should be free from saccharine matter and acidity. The percentage of real or anhydrous alcohol in the different distilled spirits varies greatly, ranging from 70 per cent. in undiluted rum, to about 30 per cent. in gin.

Wines consist of diluted alcohol, combined with certain soluble compounds, as œnanthic and other ethers, free acids and salts, and, besides these, coloring, astringent, and saccharine matters.

The amount of alcohol varies, from about 18 to 22 per cent. in ports, sherries, and madeiras, to 7 to 9 per cent. in clarets and hocks.

The acids and salts consist chiefly of tartaric acid and the acid tartrate of potash: there are also small amounts of other salts, as phosphates of lime, magnesia, and iron.

The amount of alcohol in port wines varies from 17 to 21 per cent.; in madeira it is about the same; in sauterne it varies from 12 to 15 per cent.; in red French wines from 9 to 14 per cent.; in champagne 10 to 11 per cent.; in Rhine wines 6 to 12 per cent., usually from 9 to 10 per cent.

Measured by the amount of acidity, Dr. Bence Jones has arranged wines and spirits as follows:—Sherry, port, madeira, champagne, burgundy, hock, and moselle. The least acid of all alcoholic spirits are geneva and whisky, then rum and brandy, afterwards ale, porter, and stout; all wines are found to be more acid than malt liquors.

Measured by the amount of contained saccharine matter, commencing with the least sweet, spirits, wines, and malt liquors may be thus arranged: geneva,

rum, whisky, claret, burgundy, Rhine wines, and moselle have no sugar; then brandy, sherry, madeira, champagne, port wine, cider, porter, stout, malmsey, ale, tokay, samos, paxarete, and cyprus.

The knowledge of the composition of the different kinds of alcoholic fluids has not thrown much light upon their varying powers of inducing Gout, and the following summary includes nearly all that can be clearly made out on the subject:—[1]

1. Diluted alcohol, in the form of distilled spirits, has little power in causing Gout, at least in those who are not predisposed to it.

2. Alcohol, when in combination with other substances, as in wines and malt liquors, becomes a potent cause of Gout, and the greater the amount of contained spirits in such beverages, the more powerful their influence in producing the disease.

3. Neither the acid, sugar, nor any known principle contained in these liquors, can as yet be proved to impart to the alcohol its predisposing influence; for wines the least acid, and liquors the least sweet, are among the most baneful.

4. Alcoholic drinks which have little tendency to cause dyspepsia, and those which more especially act as diuretics, can, as far as Gout is concerned, be taken with greater impunity than beverages of an opposite character.

Influence of the Solid Food in causing Gout.—It is matter of much difficulty to assign the share that different articles of solid food have in the production of Gout, and likewise to separate the effect of indigestion caused by any article of diet from the secondary influence of the same food after its absorption into the system. Cullen remarked that Gout seldom attacked persons who lived much on vegetable diet, but he added, or persons who were employed in constant bodily labor; and doubtless the disease is rare amongst those who live in the country, working hard, and living abstemiously. Experience, moreover, has pretty well established this fact—namely, that an excess of food, more especially animal food, favors the production of Gout, and it probably does so by causing an increased formation of nitrogenized compounds, more especially uric acid. Vegetable substances have comparatively little direct influence, unless they cause dyspepsia from their mechanical structure, or other peculiarities. As far as composition is concerned, it is probable that articles of food containing a considerable amount of saline principles, as for instance the salts of potash, are useful in keeping up the activity of the secreting organs, especially the function of the kidneys, and many such substances, even if acid to the stomach, yet tend to alkalinize the blood and urine from the decomposition of the vegetable acid, and the formation of a carbonate of the alkali.

Pie-crust and sweet substances probably are injurious rather from the indigestion they induce than from containing any noxious principles.

Made dishes, and those which are very rich and complex, are liable to upset the stomach, and thus act rather as exciting than predisposing causes of Gout.

Indigestion as a Cause of Gout.—In investigating this subject, great difficulty is experienced in discriminating between the dyspepsia leading to Gout and that which arises from an already formed gouty diathesis. Many persons suffer from dyspepsia all their lives, and yet never exhibit a gouty symptom; on the other hand, some of those most severely afflicted with Gout have scarcely ever felt the sensation of indigestion.

Indigestion, if it causes the production of an increased amount of acidity in the system, can easily be supposed to aid the development of Gout, by causing a less alkaline state of the blood, and hence favoring the insolubility and deposition of the urate of soda in the tissues; it may also act as a predisposing cause by promoting the formation of uric acid itself. The form of dyspepsia which seems most injurious is that which is connected with congestion of the portal system, and increased vascularity of the mucous membranes of the stomach.

Although observations have not shown that exercise has any marked influence upon the amount of uric acid excreted in a given time by a healthy person, still it is certain that want of exercise soon leads to a sluggish performance of the more important functions of the body, and the production of dyspepsia indirectly favors the development of Gout.

Influence of Nervous Depression.—Any circumstance which lowers the tone of the nervous system tends very greatly to the development of the gouty paroxysm, although it is questionable if this cause alone can produce the gouty diathesis. The effect of the mind upon the function of the kidneys, is well known and easily appreciated, and upon the other secreting organs, although less readily observed, it is doubtless equally potent. Any severe mental labor is often followed by an attack, and so is prolonged sorrow; and venereal excesses probably act by lowering the tone of the nervous system.

Influence of Climate and Season.—There is no doubt that Gout is much less prevalent in hot than in cold or temperate countries, and it would appear from the reports of travellers that the disease is unknown among the natives of the interior of Africa; it is rare, if not unknown, in

[1] Nature and Treatment of Gout. Second edition.

China, Japan, the East Indies, and Turkey, and much less frequent in the south of Europe than in this country. Great stress, however, must not be laid on these facts, for Gout is scarcely seen among the laboring populations in Poland, Russia, and other cold climates. The character of the beverages used by these different peoples has probably more influence upon the production of Gout than the climate under which they live.

As far as regards the development of an attack, climate and season have a very decided influence: for example, a gouty man may often escape his accustomed winter attacks by removing from England to Malta or Egypt during the cold season in this country; and the histories of gouty patients afford abundant proof of the comparative frequency of gouty seizures in the winter months.

In the early stage of Gout the attacks are most frequent in spring, and are often confined to that season; after a time an autumnal seizure is added; but when the disease has become further ingrafted into the system, the fits may occur at any season, and at most irregular intervals.

There are occasionally found exceptions to the above rules; for some patients, owing probably to individual peculiarities, suffer far more in summer than in winter.

In warm climates and during hot seasons the function of the skin becomes more active, and this fact affords an explanation of any influence dependent on these circumstances.

Influence of Lead Impregnation in causing Gout.—About fifteen years since I was struck with the fact that a large percentage of the gouty patients who had come under my care in hospital practice consisted of painters, plumbers, or other workers in lead, and a more careful investigation of the subject forced the conclusion on my mind, that the influence of this form of metallic impregnation in inducing a gouty condition of the system was very considerable.[1] Since that period I have made many observations on the point, and the conclusions may be summed up in a few words:—

1. Among the patients in London hospitals, a very large proportion of the gouty (about thirty per cent. in my hospital practice) have been subjected to the influence of lead: many of these have had painters' (lead) colic; some have suffered from wrist-drop or more severe forms of lead paralysis; and all have exhibited the peculiarly characteristic blue line on the gums.

On careful inquiry into the habits of these men, nothing remarkable has been elicited; they have been about as temperate as other men employed in different occupations. It is not painters alone who form this high percentage, but plumbers, composition doll-makers, workers in lead mills, and others whose trades have caused them to be exposed to the use of lead. It may be considered as established, that the metal lead acts as a powerful predisposing cause of Gout.

2. Many cases have occurred which have induced me to believe that individuals suffering from the gouty diathesis are more susceptible to the influence of lead than the majority of other people. It is a well-known fact, that when the drinking water in any house is slightly impregnated with this metal from the cistern or pipes with which it has come in contact, some of the residents in that house may experience the symptoms of saturnine poisoning, while the rest may be unaffected, although drinking an equally large quantity of such water; thus proving that some people are peculiarly susceptible to its influence: some remarkable examples of this fact have come under my notice. In the medicinal administration of lead preparations, as in cases of hemorrhage, or excessive mucous discharges, it will be observed, if patients be closely watched, that the blue line appears on the gums in some cases with extreme rapidity, and that even colic pains are soon experienced; but that in others the use of the lead salts can be continued for a lengthened period, without the production of any such phenomena. In several instances where patients have proved to be very easily affected, it has been found that they were of a gouty habit, and many of them had repeatedly suffered from severe attacks of the disease.

3. In some instances, severe attacks of Gout have been induced in gouty patients by the medicinal administration of lead salts, on account of the occurrence of epistaxis or other forms of hemorrhage; and the frequent recurrence of the seizures, whenever the medicine has been repeated, has satisfactorily shown that the phenomena were to each other in the relation of cause and effect.

4. On investigating the physiological action of the administration of lead salts, it is discovered that a very decided effect is produced by that metal upon the secreting power of the kidney, as far as uric acid is concerned; the function is notably diminished.

5. The blood of individuals suffering from lead paralysis always contains an abnormal amount of uric acid; and the same, probably, holds good in all cases of lead colic.

6. There are many facts which seem to show that the influence of lead, when uncombined with that of fermented liquor, is scarcely able to produce gout; for al-

[1] Transactions of Medico-Chirurgical Society, vol. xxxvi., 1854.

though in England Gout is so common among those who work with lead preparations, yet neither in France nor Scotland has the connection been noticed. In Edinburgh, however, even lead poisoning appears to be much less frequent among painters than in England.

Before concluding this subject, it should be observed that it has long been a matter of medical observation, that lead poisoning often gives rise to pains in the limbs which have generally been regarded as rheumatic or neuralgic in character. These pains, however, which appear to be likewise produced by some other metals, must not be confounded with Gout, with which they probably have no relation.

PATHOLOGY OF GOUT.—Our space is insufficient to allow of even a brief summary of the different views which have been held from time to time, and by different writers, of the real nature of Gout. As the disease has been known from remote antiquity, and as it is one which has always appeared to interest mankind, it is not to be wondered at that very numerous and diverse views have been advocated. The existence of an altered condition of the blood has been always a popular belief among the ancient as well as modern pathologists; but various opinions as to the nature of the alterations have been held. The ancients, as was their wont, ascribed Gout to the superabundance of phlegm, bile, and other natural secretions in the system, and they attributed chalk-stones to the concretions of such matters in the affected parts; and such ideas were held by some physicians even up to a comparatively late date. There have not, however, been wanting supporters of a totally different doctrine—men who, discarding all ideas of a morbid state of the fluids of the body, have looked upon the disease as dependent upon an alteration in the structure or functions of the nervous, vascular, and other systems.

Cullen was the great supporter of the anti-humoral doctrine, and brought forward many cogent reasons for not regarding gout as due to an altered condition of the blood or the secretions therefrom. Cullen was a physician whose views are always worthy of consideration, and although many of his statements can now be shown to be erroneous, still a short summary of his objections may not be disadvantageously given, if only for the purpose of being combated. Cullen thought that there was no evidence of the presence of any morbid matter in gouty persons; that neither the blood nor secretions from it had been proved to be altered in this disease, and that previous to an attack there was no symptom indicating such a change; but that the balance of evidence was against this idea, seeing that many individuals before a seizure were apparently in unusually good health. From what has been already stated in the present article, it will not be difficult to show the error of the above opinion; for it has been demonstrated beyond doubt, by clinical evidence, that the blood is invariably altered in Gout, from the presence of uric acid in the form of urate of soda; that the secretions from the blood are likewise influenced; that symptoms are usually present before the development of the articular inflammation; and, lastly, that the inflamed parts are always altered by the secretion into their structures of the same urate of soda—a phenomenon which has never been shown to occur except in this disease, and which may therefore be regarded as pathognomonic.

Cullen, who was necessarily aware of the occasional presence of chalk-stones in gouty subjects, overcame the difficulty by stating that such deposits only occurred now and then, and after the disease had been present a long time; and, in fact, regarded chalk-stones as accidental phenomena, and in no way essentially connected with the disease. Cullen considered Gout to depend upon a peculiar conformation of some portion of the animal economy, more especially the nervous system; he regarded the chief exciting causes, such as intemperance, indigestion, cold, and other depressing influences, as acting upon the nervous centres, and looked upon most of the symptoms of retrocedent Gout as affections of the same kind.

Cullen's theory of Gout is best expressed in his own words: "In some persons there is a certain vigorous and plethoric state of the system, which, at a certain point of life, is liable to a loss of tone in the extremities. This is, in some measure, communicated to the whole system, but appears more especially in the functions of the stomach. When this loss of tone occurs, while the energy of the brain still retains its vigor, the *vis medicatrix naturæ* is excited to restore the tone of the parts, and accomplishes it by exciting an inflammatory affection in some parts of the extremities. When this has subsisted for some days, the tone of the extremities and of the whole system is restored, and the patient returns to his ordinary state of health. This is the course of things in the ordinary form of the disease, which we name *regular* Gout; but there are circumstances of the body, in which this course is interrupted or varied. Thus, when the atony has taken place, if the reaction do not succeed, the atony continues in the stomach, or perhaps in other internal parts, and pro-

duces that state which we have, for reasons now obvious, named *atonic* Gout.

"A second case of variation in the course of the Gout is when, to the atony, the reaction and inflammation have, to a certain degree, succeeded; but, from causes either internal or external, the tone of the extremities, and perhaps of the whole system, is weakened; so that the inflammatory state, before it had either proceeded to the degree, or continued for the time, requisite for restoring the tone of the system, suddenly and entirely ceases. Hence the stomach and other internal parts relapse into the state of atony, and perhaps have thus increased by the atony communicated from the extremities; all which appears in what we have termed *retrocedent* Gout.

"A third case of variation from the ordinary course of Gout is when, to the atony usually preceding, an inflammatory reaction fully succeeds, but its usual determination to the joints is by some circumstances prevented; and is, therefore, directed to an internal part, where it produces an inflammatory affection, and that state of things which we have named the *misplaced* Gout."

It is as easy to show the fallacies in this theory of Cullen concerning the nature of Gout, as to refute the many dogmas which he puts forward; and, therefore, the mere enumeration of it must be considered sufficient. Since Cullen's time there have been writers on this disease, who, even with the knowledge of some at least of the facts recently discovered, still cannot bring themselves to admit the truth of the humoral doctrine of Gout. The late Sir C. Scudamore, although confessing many difficulties, was, up to a few years before his death, of opinion that there existed a species of plethora. The subject of chalk-stones was already a difficulty with him, and the discovery of the constant presence of uric acid in the blood of gouty patients shook his confidence in his old ideas upon the subject. A recent writer, Dr. Gairdner, believes in the existence of a state of plethora of the chylopoietic organs as a constant accompaniment of Gout, and thinks likewise that the veins of the part are in a varicose state, that the heart is oppressed with a flood of returning venous blood, made impure from the non-elimination of urea, uric acid, and biliary matters; and he thus views the phenomena of a paroxysm of Gout: "Venous congestion I consider the first condition essential to the formation of the gouty diathesis. It is no new observation; it is found interspersed through the writings of all former authors. Even those who adopt explanations inconsistent with such a state of things, notwithstanding admit it. This state of the blood was first clearly announced as the great cause of Gout by Galen, whose opinions have continued to influence the minds of succeeding physicians in a greater or less degree to the present day. The truth of the fact being, I imagine, unquestionable, it will always continue to embarrass the doctrines of those who advocate opinions with which it is incompatible. But the great venous canals of the body, as well as the larger arterial vessels, are endowed with a resiliency which enables them to struggle well against the flood of returning blood. This fluid, then, is compressed between two opposing forces—that, namely, which is derived from the heart and arterial system, urging it forward on its course, and, on the other hand, the antagonistic resistance of the great veins leading to the right auricle. Under this compression, I believe that the vessels give way, and a true hemorrhage is occasioned in the part affected. If the rupture takes place in a minute capillary, carrying the serous portion of the blood only, œdema is the consequence; but if the burst bloodvessel be one carrying red blood, a true ecchymosis is formed."

And again: "It will surely be admitted that the capillary and nutrient vessels, distributed on the extreme and sentient fibrillæ of the nerves, are affected in the same manner as the larger venous trunks. I believe these distended capillary vessels are the real seat and cause of the painful phenomena of Gout. Is it not credible that such vessels, dilated so as to admit fluids for which they were not intended, and bound down by the firm fasciæ, in which Gout has its usual seat, may give rise to much suffering?"

The great objection to Dr. Gairdner's views is, that there is no proof of the occurrence of hemorrhage during the gouty seizure; no one has seen the ecchymosis spoken of by him as a constant attendant on it, whereas other and frequent changes are invariably observed in the inflamed part. It is, indeed, true that abdominal plethora is often present in gouty habits, especially when the disease has been induced by high living; but on the other hand, in many cases, even when the disease is of an inveterate character, no symptoms indicating such a condition are discoverable. Furthermore, congestion of chylopoietic organs, accompanied with obstructed cardiac circulation, is very frequent and of long duration, and yet no Gout is developed.

Many other authors have, within the last half century, published views as to the nature of Gout; amongst whom, in this country, may be mentioned Murray, Forbes, Parkinson, Wallaston, Parry, Sutton, Sir E. Home, and Sir Henry Holland. The reflections of the last-named physician on the subject are well worthy

of attention. In France the principal authors have been Cruveilhier, C. Petit, Barthez, Guibert, and Trousseau. Some of these have been inclined to favor the views of the solidists, but perhaps the opinions of the majority have had a decided tendency to humoralism.

After these short expositions of some of the principal opinions, which have been held of the nature of Gout in recent times, and which have proved to be wholly or in part fallacious, it is necessary to lay before our readers the view which we think accords more completely with facts than any other which has been proposed, and one which will explain nearly all the phenomena presented by the disease. In the first place, "it is essential to the production of this form of articular inflammation that the blood should contain an abnormal amount of uric acid, or rather of urate of soda; and even the phenomena which constitute irregular Gout, or are regarded as gouty manifestations, demand for their occurrence the presence of the same salt in the circulating fluid."

The truth of this proposition has been proved by some hundred clinical observations upon cases of articular inflammation; and although it has not been frequently demonstrated in cases of irregular Gout, yet this has been done sufficiently often to make it matter of certainty. It will, therefore, be unnecessary to bring forward any further evidence of the correctness of the proposition.

In must not, however, be supposed that an excess of urate of soda in the blood constitutes Gout; this would be erroneous, for the salt is occasionally present in large quantities, and yet no gouty phenomena are manifested; but the individual so circumstanced may be looked upon as especially prone to its development, if other circumstances arise which favor its production.

In the next place, "gouty inflammation is invariably accompanied with the presence of urate of soda in the inflamed tissue."

We have already given much evidence of this fact, and in no case in which real gouty inflammation has been shown to have occurred have the morbid appearances failed to present themselves when sought for.

Furthermore, "it can be shown that the amount of deposited urate of soda is not in proportion to the intensity of the inflammation, and that in some structures the infiltration may ensue and scarcely give rise to any inflammatory action; facts tending to the supposition that the deposited matter may be looked upon as the cause rather than the effect of such inflammation."

It is most important, as bearing considerably upon the true pathology of Gout, that the above proposition should be fully substantiated, and it is not difficult to bring forward much evidence in its favor. If an opportunity occurs of examining a joint, as the knee, which has been but once attacked, no great amount of alteration may be exhibited, although the inflammation has been intensely acute; on the other hand, after a joint has become, as it were, callous, considerable deposition can be shown to occur without the production of much local inflammation. The same fact is better illustrated in the case of the ear, in which, as before stated, urate of soda is frequently deposited in the fibro-cartilaginous tissue. This phenomenon, although at times recognized from the sensation of heat, pricking, and tenderness of the part, yet more commonly takes place without the production of any symptom—the patient being, in fact, quite unconscious of its occurrence. In many chronic forms of Gout it is not unusual to find large collections of the white matter formed near the surface, with comparatively little constitutional disturbance, and any one watching the progress of such a case must soon become convinced that the elimination of the salt is not an effect of the inflammation. To show that it is probably the cause is equally easy: let us compare, for example, the articulating surfaces of the knee-joint with the fibro-cartilage of the external ear, in respect to their liability of becoming inflamed from the presence of foreign bodies or the infliction of injuries; irritation of the former is known to be most serious in its results, from the acute action which is set up, whereas considerable damage may be done to the latter without any but the slightest inflammation ensuing. Would not a like difference exist in the same tissues if a substance foreign to their constitution became infiltrated by the action of disease?

"The inflammation of the gouty paroxysm tends to the destruction of the urate of soda in the blood of the inflamed part, and probably also of the salt which has been thrown out."

When describing the blood and secretions of gouty subjects, allusion was made to the fact that, although the fluid effused from the action of a blister usually contains uric acid, yet an exception occurs in the case of its being applied over an acutely inflamed surface. Under these circumstances, it would appear that the uric acid is destroyed by the presence of the inflammatory action; and, if this be true, the gouty paroxysm is, at least to some extent, a salutary process, tending to rid the system of accumulated uric acid; but, as the fit is always accompanied by local mischief, the good effected is by no means unalloyed.

"Gouty deposits do not take place indiscriminately in any situation or in any

tissue; but a selection is made, in close relation to the vascularity of the part."

If the remarks on the morbid anatomy of Gout be referred to, it will be observed that the structures most liable to become affected are those possessing little vascularity; as, for example, cartilage, fibro-cartilage, ligament, tendon, and synovial membrane. It will be seen, also, that the deposit, as it were, avoids the contiguity of bloodvessels, as exemplified in the knee-joints, in which the surfaces in contact with the synovial fringes are free. Again, in the cartilage itself, the deposition, although intestinal, commences near the free surface, and gradually penetrates deeper into the tissues, but, even in extreme cases, scarcely extends beyond a third of its thickness, and is always at a considerable distance from the blood-vessels of the bone.

It is probably owing to this freedom from deposition which vascular tissues enjoy, or to the rapid destruction of the urate of soda when placed under circumstances which bring it into contact with bloodvessels, that acute gouty inflammation does not affect the covering or lining membranes of the heart, as is the case with rheumatic inflammation. The morbid changes which are often found in the valves of the heart, or the lining membrane of the aorta, form no exception to this statement, for they are not due to the presence of urate of soda, but to an alteration of an entirely different character.

"The kidneys are often, if not always, implicated in Gout, and the affection, possibly only functional at first, soon becomes structural. The urinary secretion is likewise altered."

Under the "Morbid Anatomy" of the disease it has been affirmed that, in all cases where Gout has existed for any length of time, some alteration is found in the kidneys; deposition within or external to the tubuli uriniferi is discovered, and the normal structure of the secreting apparatus is injured. In chronic cases the peculiar shrivelled or gouty kidney is frequently met with. As yet I have never seen this latter form of kidney disease without finding the white deposition, and I cannot help thinking it probable that the presence of the urate may be the exciting cause of the subsequent changes which ensue in the structure ot the kidney.

The kidney affection, whether functional or organic, readily explains the altered state of the urinary secretion: The functional disturbance may cause the defect and irregularity of the secretion of uric acid; the organic alteration accounts for the further diminution in the excretory power of the organ, and the frequent presence of a small amount of albumen in the urine.

"The impure state of the blood, due to the presence of urate of soda, is probably the cause of the disturbance which often precedes the gouty paroxysm; that is of the so-called premonitory symptoms, as well as most of the anomalous affections (irregular Gout) to which such patients are liable."

That suppression of the whole urinary secretion such as occurs in intense renal congestion and advanced forms of albuminuria gives rise to many and alarming symptoms, is a well-known and acknowledged fact; and hence it is reasonable to suppose that a suppressed excretion of one of its constituents should manifest itself by symptoms of a less intense and fatal character. Possibly some of the symptoms may be due to the reaction consequent upon an attempt at deposition of urate of soda in certain unusual situations. Dyspepsia, for example, is very common in albuminuria; it is also frequent in persons of a gouty diathesis.

"The causes which predispose to Gout, independent of those connected with individual peculiarity, are either such as produce an increased formation of uric acid or which lead to its retention in the blood."

Although our knowledge of the causes which lead to the undue formation of uric acid is most imperfect, yet there can be little doubt that over-feeding, especially in regard of animal food, portal congestion, and deficient exercise, aid very much in the production of its excess; and it is known from clinical experience that these are predisposing causes of Gout, as appears to be also lead impregnation, the use of malt liquors, wines, and so on, which probably lead to a defective elimination of the same acid. Deficient nervous energy, arising from mental or other causes, seems to act in the same manner.

"The causes exciting a gouty fit are those which induce a less alkaline condition of the blood, or which greatly augment for the time the formation of uric acid, or such as temporarily check the eliminating power of the kidneys."

The deposition of urate of soda is caused by its insolubility, and this may arise either from the large amount which is formed, or from the serum of the blood becoming less capable of holding it in solution. The blood serum is alkaline in reaction, and this condition may become lessened from various causes, especially from deficient action of the skin, the taking of a large amount of acid into the stomach, and, perhaps, an increased formation of some acid, arising from dyspepsia: all the above-named circumstances will, it is known, often excite a gouty attack. It seems probable that there may be at times a great temporary in-

crease of uric acid found in the system, by causes giving rise to an attack of dyspepsia.

"Deposits of urate of soda in the textures of the body never occur but in true Gout."

It has already been shown that urate of soda invariably accompanies gouty inflammation, and it can be equally proved that it is not thrown out under other circumstances. There are, indeed, many statements which appear at first to throw doubt upon the correctness of the above proposition, but which upon closer examination can be shown to be erroneous. I have seen white nodules on the ears of young people who have apparently had no gouty tendency: these have been found to contain fat and amorphous granular matter, but no crystals of urate of soda. I once was shown a large tumor, taken from the scalp of a young woman, and was assured that it had been analyzed and found to consist of urate of soda, and that there certainly was no gouty tendency in the patient. On examining a portion of the tumor, no trace of uric acid could be discovered in it.

Diseased joints from rheumatoid arthritis, and other chronic diseases, have been also asserted to be covered with a urate deposit: in all such cases I have shown that bone-earth concretion has been mistaken for urate of soda.

In examining the large toe-joints of a large number of bodies, a little white spot was seen in two instances; yet no Gout had been known to have occurred during life. One of the individuals had been a cabman, and had granular kidneys—he had died from an injury; the other had died of delirium tremens. In each case one foot only was affected, and the space covered with the deposit did not exceed a sixteenth of a square inch. These spots were doubtless indicative of very slight gouty inflammation, and in the investigation of cases of confirmed Gout it is extremely common to find that patients have complained of twinges and slight tenderness of the great toe for several years before the occurrence of a distinct and unmistakable paroxysm.

Explanation of other Phenomena which occur in Gout.—There are certain peculiarities in the history of Gout which still require explanation, one of which is the fact that gouty inflammation in its first visitation generally attacks the ball of the great toe. Boerhaave and Van Swieten tried to explain this by supposing that Gout chiefly attacked those tissues in which the fluids have most difficulty in passing through, as the periosteum, tendons, nerves, membranes, and ligaments, and such as are most remote from the heart, most pressed upon and injured, and most subject to cold and moisture. I believe that there is much truth in these remarks, although expressed in terms which are not conformable to the pathology of the present day. Let us endeavor now to explain the phenomenon.

The great toe contains a considerable amount of tissues peculiarly liable to become the seat of the deposition of urate of soda; as, for example, the cartilages and ligaments, tissues having either little vascularity or nourished independently of bloodvessels: the great toe being very remote from the heart, the circulation is weaker there than in many other parts, weaker than in the hips or knees. These remarks, however, both with regard to the tissues and the distance from the heart, apply even with greater force to the phalangeal joint of the great toe than to the metatarso-phalangeal joint, and apply also to the joints of the smaller toes; but, on the other hand, this latter joint is more subject to injury by pressure; it often has to bear the whole weight of the body, and sudden shocks—as, for instance, from false steps—are first felt in this articulation. In cases where the great toe has not been attacked, some peculiarity has been present in the conformation of the foot, which has had the effect of throwing the pressure on some other part.

That the metatarso-phalangeal joint is liable to injury, I have been able to ascertain from the examination of several great-toe joints in subjects who had never had Gout; and it was found that in six instances only out of twenty were these joints absolutely healthy, there being in the rest more or less evidence of ulceration of the cartilages. The reasons for the great toe of one side of the body being affected apply equally to the other; and hence the disease not uncommonly attacks first one and then the other, within the space of a few hours or days.

It is not difficult even to explain the sudden shifting of the inflammation from one joint to another: it must be remembered, that the deposition precedes the inflammation, and it is well known that the establishment of inflammation in one part is often followed by its subsidence in another. The nervous connection through the spine may also explain the alternation of inflammation so often observed in symmetrical joints.

In explanation of the reason why numerous joints are attacked as Gout gains ground, or becomes more engrafted into the system, it may be advanced that the cartilages and ligamentous structures of the earlier implicated articulations being infiltrated with the urate, and the blood still remaining impure from the presence of the salt, other surfaces are required to be selected. The defective circulation in the external ear, from the nature of its

structure and its exposed situation, is probably the reason why the small urate nodules are so frequently found upon it. I may remark that, up to the present time, I have never seen the concretions on the ears of females; this immunity may arise from their being usually covered: individuals with cold ears seem to be most frequently affected with them. The cartilaginous, fibrous, and ligamentous tissues are peculiarly susceptible of becoming the seat of the deposit, partly from their little vascularity, and probably also from the fluids in these structures being less alkaline in reaction than the blood itself, and liable to become neutral or even acid. After death, in chronic gouty cases, the synovial fluid has been found in a few instances distinctly acid.

The explanation of the comparative immunity from Gout enjoyed by females is to be sought for in their freedom from the influences of many extraneous causes, and their possession of a function which has a tendency to rid the system periodically of superfluous blood. As a rule, women take much less wine and beer than men, and altogether lead lives of greater prudence. After the cessation of the catamenia, women become more liable to gouty paroxysms. Women who inherit Gout strongly, even if they live very carefully, are apt to suffer from the irregular manifestations of the disease; the same remark applies to men who, inheriting the disease, and having the fear of it before their eyes, have from early life studiously avoided the causes which engender it. The reason of the almost certain recurrence of Gout, unless the greatest care be taken to overcome the tendency, must be sought for in the fact that the causes of the increased formation of uric acid in the system, and of its defective elimination, are generally irremovable; the periodicity of the disease may be due to the gradually increasing impurity of the blood from the time of the purification which occurs during the fit, and also to periodicity of the exciting causes—as the recurrence of the vernal and autumnal changes.

Diagnosis of Gout.—To make a correct diagnosis in cases of joint disease is a matter of importance, not only as regards the treatment, but as respects the prognosis: it is likewise often one of great difficulty, and always requires great care, even with those who have had most experience in the subject.

To determine if a case be of a true gouty character or not:—

The history of the case should be fully inquired into; it must be remembered that Gout is strongly hereditary, and therefore, if either parent or grandparent of the patient suffered from it, the probability of his joint affection having the nature of Gout is much strengthened.

The age should be taken into account; Gout is very rare before puberty, not common till after thirty-eight or forty years of age; it may occur at a very advanced age.

The sex of the patient influences the diagnosis; Gout is much more frequent in males than females; in the latter it is seldom seen till after the catamenia have ceased.

The mode of life of the patient for the several past years should be taken into consideration. Wine, malt liquors, and much animal food, tend to produce Gout; spirits have little effect. The history of the disease in its early stages should be inquired into. If we discover that the ball of the great toe was first and specially affected, and that the intervals between the attacks were of considerable duration, the conclusion that it is true Gout is almost certain to be correct; but if the history has not been of this characteristic nature, it must not be concluded that the affection is not gouty. An error may sometimes arise from laying too much stress upon the toe affection, to the exclusion of other symptoms; I have seen a great toe swollen, tense, red, and hot, and having every appearance of being attacked with intense gouty inflammation, which has afterwards been proved to depend on pyæmia. In this instance, a day or two from the commencement of the seizure, other parts were implicated and the presence of pus was manifest; from the very first the amount of constitutional disturbance was far beyond that which occurs in Gout affecting one small articulation.

The character of the symptoms should not be neglected. It must be remembered that in Gout the pain is generally severe; during the early stage of the inflammation the joint is very tense; that it subsequently pits, or is œdematous, and, lastly, desquamates; that the febrile disturbance is usually moderate, and in proportion to the extent of the local inflammation. The presence or absence of periodicity in the attacks must not be overlooked; Gout, especially for the first few years, is almost invariably periodic, complete and long intervals occurring between the paroxysms.

In true Gout, acute inflammation of the heart does not occur; in rheumatism cardiac complication is frequent.

If a deposit of urate of soda can be discovered either in the external ear of the patient, in the tips of the fingers, the bursæ over the olecranon, or in any other situation, it is a matter of certainty that the patient has the gouty diathesis. Great care, however, must be taken not to confound enlargements of other kinds, as of the ends of the phalanges, or simple bur-

sal swellings, with those produced by the deposition of urate of soda.

An examination of the blood of the patient almost decides the point: this can be effected if only a single ounce is drawn from a vein, as not more than one or two drachms of the serum are required for the thread experiment. The fluid from a blister may be used instead of blood serum, although a negative result obtained from it is not so satisfactory.

Lastly, the presence or absence of a trace of albumen in the urine, if the case be of a chronic character, may afford some assistance, as this symptom is very frequent in Gout, and dependent on the slight kidney affection which so commonly ensues after the disease has lingered in the system for a few years.

A case, showing the importance of attending to the above differential points, has within the last few weeks come under observation.

A woman aged 35, married, with one child, has for many years worked in a laundry, and has been necessarily exposed to damp, and great alternations of temperature; says she has always been temperate, but has drunk beer, and now and then a little spirits.

The patient is very deaf, which renders it difficult to ascertain every point either in her previous history, or that of the disease, and which at first obscured the diagnosis. About two years since she had swelling of the left knee, and thinks this joint was alone affected; she was unable to move about for five or six months. About a year from the commencement of the knee affection the knee and ankles were attacked and the great toe was implicated; does not remember whether it was the metatarso-phalangeal or the phalangeal joint; the attack lasted a month or so. From this time to the present has been frequently obliged to keep her bed from joint disease, both the upper and lower extremities being involved. When first seen, both knees were tender and swollen, as likewise the ankles; some tenderness also of two or three of the phalangeal joints of the hands, and the first phalanx of the left index, and the same joint of the right middle finger; these are considerably thickened and swollen, and their mobility much impaired; no visible deposits either in ears or elsewhere. No amount of febrile disturbance present. Catamenia regular. Urine free from albumen. None of her relations had suffered from joint disease.

There was considerable difficulty in arriving at a safe conclusion in this case.

Against the disease being Gout, and in favor of its being rheumatoid arthritis, there was the following evidence: the sex of the patient, her somewhat early age, and the catamenia being still present; the supposed moderate abstinence from malt liquors (though she always took three pints of beer a day, with some spirits); the absence of any affection of the great toe in the first attack and the question as to which joint of the toe was implicated in subsequent seizures; the duration of the disease and its almost progressive character from the first; the non-discovery of chalk-like deposits in the body; and, lastly, the probability of the joint affection being brought on by cold.

On the contrary, in favor of its being Gout were the following circumstances: the appearances of general good health; the distinct interval of six months between the first and second seizures; and the probability of the patient taking more malt liquors than she herself allowed.

To remove any doubt upon the subject a very small venesection was performed, and upon analysis the serum was found to yield a large quantity of uric acid by the thread experiment. This was almost decisive of its being Gout; but on very close inquiry, finding that the elbows had been affected several times, the state of the bursæ over the olecranon process was examined, and in the right bursa some thickening was detected, as likewise the presence of two or three flattened little masses, doubtless of urate of soda. The presence of these little masses in the bursa, conjoined with the fact of the blood being rich in uric acid, fully established the nature of the case; and this discovery was of no small importance, not only in the treatment of the attack, but as to the means to be taken in future to keep the disease from making further inroads in the system.

Prognosis of Gout.—An attack of acute articular Gout is probably never fatal, and individuals are often seen who have suffered from severe paroxysms for many years, and yet appear to have experienced little or no injury beyond their sufferings at the time. If the intervals between the seizures continue to be of fair duration, as one year or half a year, when the patient is beyond middle age, the prognosis is favorable, and there is no reason why any appreciable shortening of his life should ensue, provided he is willing to live according to rule, and is not exposed to accidents or other powerful causes of the disease. In confirmation of this statement is the fact that robust-looking persons of very advanced age are not infrequently seen, who have been the subject of periodic visitations of Gout for a great number of years. When, however, in comparatively early life, the attacks are frequent and prolonged, the prognosis becomes much less favorable, and especially if the urine exhibits any trace of albumen, either during the pa-

roxysms or in the intervals of freedom from them.

The appearance of the patient's urine helps us in making a prognosis. If it was formerly turbid from urates, or if it gave rise to a deposit of crystallized uric acid, and has become of late clear and of a paler color, the change probably indicates that the kidneys have to a considerable extent lost their power of eliminating uric acid, and that which seems to the patient a favorable change is in reality a sign of a serious structural alteration in an important secreting organ.

It is a grave sign in Gout to find the urine pale, the specific gravity exceedingly low, and the fluid devoid of uric acid; and if, in addition to this condition, albumen is likewise present, the indication becomes still more unfavorable.

Chronic Gout has a decided tendency to shorten life, and this fact is recognized by insurance companies, who, however, do not appear to make much distinction between the acute and chronic forms of the disease: at different offices varying rates are adopted.

In the early attacks a patient is likely to inquire of his physician, if it is possible to prevent a return of his ailment—a question tantamount to asking if there is any known method of absolutely eradicating the tendency to Gout from the system.

There are records of individuals who have experienced but one attack, though they have lived to a great age. I have known thirty-five years elapse between a regular attack of Gout in the great toe and the patient's death, which took place after he had attained his seventieth year. Several cases have come under my observation in which the disease, after having recurred periodically for many years, gradually declined in intensity and duration, and at last altogether disappeared.

The appearance of Gout can never be looked upon as a good omen; a statement contrary to a once popular opinion.

The greater the age at which Gout first seizes the individual, the more satisfactory the prognosis.

If it attacks very young subjects, the future prospects are bad.

Hereditary is generally much less tractable than acquired Gout.

The appearance of chalk-stones on the surface is always inauspicious, even if confined to the helix of the ears.

Gouty patients are more liable to suffer severely from accidents and exposure than the majority of people; the more the kidneys are implicated, the less able are they to withstand the effect of shock upon the system.

In concluding the subject of the prognosis of Gout, I will remark, that I consider that a single fit of Gout, however slight, should be looked upon as an intimation that the patient cannot go on with impunity in his then habits of life; it is a warning that either he must change them or expect returns of the disease, which, as time advances, are certain to increase both in frequency and duration, and both embitter and shorten existence.

On the other hand, I am equally persuaded that if proper regiminal and medicinal precautions be taken, the gouty patient may be saved from such an alternative and the disease, instead of increasing in intensity, may be gradually mitigated, and probably interfere but little with the comforts of life.

TREATMENT.—The subject of the treatment of Gout naturally divides itself into, first, the treatment of the articular inflammation; secondly, the management of the gouty subject during the intervals of the attacks; and, thirdly, the treatment of the complications and irregular manifestations of the disease. Under the head of Treatment we shall discuss not only the medicines which it may be necessary to administer, but likewise the dietetic and regiminal management.

Treatment of Acute Gout.—Let us first examine if there is any necessity for giving medicine at all, and whether or not it is prudent to leave the joint disease to pursue its own course uninfluenced by any drug.

Cases are now and then met with in which the affection has been left to itself, and several such have come before me. From the opportunities thus afforded, I have ascertained that many of the early and slighter attacks of Gout will subside in a few days, provided the patient is moderately careful in diet; but that, if the usual mode of living is indulged in, the attack may be prolonged, even to many weeks or months; or, if slight remissions take place from time to time, they are soon succeeded by exacerbations, until at last the patient's general health gives way, the appetite fails, and thus under a necessarily altered diet the disease exalts itself; even then the attack may last a long time, as is likewise the case under homœopathic treatment, which, if honestly practised, and with the use of infinitesimal doses, is, I should imagine, exactly equivalent to the non-exhibition of medicines.

When the articular inflammation is allowed to run its own course, and has been endured for a long time, it leaves a considerable amount of injury in the affected parts, the bloodvessels become weakened, the distension of veins and the local œdema remain, and the joints are left in a condition liable to take on unhealthy action from trifling constitutional disturbances.

Assuming then, that medicinal treatment can be of real benefit, it is for us to determine the remedial agents best adapted to diminish or cure the inflammation. There is one drug which has an undoubted influence in controlling gouty inflammation, and its action in articular Gout appears as marked as that of cinchona bark in the cure of ague; this remedy is colchicum. It signifies not what part of the colchicum plant is taken, whether the corm, the seeds, or the flowers, for the same principle pervades the whole plant; neither does it signify what preparations are made use of, whether the wine, the tincture, or the extract, provided equivalent doses be administered, for the effects of all are the same.

Colchicum, as before stated, has a direct controlling power over the joint disease, and I cannot call to mind a single instance in which its influence was not well marked, although in many cases a question may arise as to the propriety of its exhibition. Colchicum in full doses produces a marked sedative effect upon the nervous and vascular systems; it has likewise a distinct influence upon the intestinal canal; and if continued too long and in too large doses, causes tormina, and a very troublesome form of diarrhœa. It also produces a peculiar change in the fecal excretions, so that those accustomed to its use can detect it by this circumstance alone, even when otherwise unaware of the exhibition of the drug; this alteration in the alvine excretions is probably due to the influence of colchicum upon the secreting apparatus of the bowels or their appendages, more especially the liver and pancreas. Colchicum has generally been supposed to cause a more copious flow of urine, and to favor the elimination of its solid constituents; but of this there will be occasion to speak further on.

Although colchicum causes purging, still its peculiar influence is quite apart from this effect. Occasionally an almost magical change is produced by a single large dose, without the appearance of the least increase in the secretion from any organ, the effect being manifested in the rapid subsidence of the pain and other symptoms of the joint inflammation; and simple purging, even though copious, will often fail to produce any notable effect under the same circumstances. I am of opinion that, in articular Gout, colchicum may be advantageously administered during the time that the inflammatory symptoms are present; and the dose of the wine of colchicum may be from ten to twenty or even twenty-five minims repeated every six hours. Colchicum given in the above manner will of itself be sufficient in most cases to cut short the gouty attack, and I have often depended on it alone; but, at the same time, in the majority of cases, it is advantageous to combine it with other remedies, which must necessarily vary in different cases. From what has been stated of the condition of the blood and of the urine, it will at once appear that some moderate alkaline plan of treatment is likely to prove advantageous, both for the purpose of increasing the alkaline state of these fluids, and also to keep in solution the salt of uric acid, which is liable to be deposited in the cartilaginous and ligamentous tissues. There can be no doubt of the value of alkaline remedies in the gouty paroxysm, and, in many cases, such salts, given in a freely diluted form, are sufficient of themselves for its removal, and are peculiarly applicable when there are circumstances rendering the administration of colchicum undesirable. Alkalies may be given either in the free state, or combined with carbonic acid, in the form of the carbonates or bicarbonates, or united with some vegetable acid, as the citric, tartaric, or acetic acids. If the stomach is irritated, and an over-secretion of acid be present, then the free alkalies or their carbonates may be administered; but if, on the other hand, there be no such condition, then the salts with the vegetable acids may be used, which produce an alkaline state of the blood and urine, although they do not act as antacids in the stomach. These alkaline remedies not only tend to keep up and restore the normal reaction of the blood, but likewise augment the excretion of urine, and with it the elimination of those solid matters which are unduly retained in that fluid in gouty states of the system. It is important, likewise, to make a selection of the alkali, and unless there are circumstances which render the use of soda desirable, such as an imperfect action of the liver, or a deficient secretion of bile, this alkali is the least fitted for exhibition, as it has much less power than other fixed alkalies of dissolving or holding in solution uric acid. The salts of potash are, in the majority of cases, more suitable than the salts of soda, as they not only exert a much greater solvent action upon urate of soda, but likewise augment in a greater degree the excretion of the urine. A third fixed alkali or its salts can be employed in lieu of soda or potash, namely, lithia, an alkali now able to be procured in quantities sufficient for medicinal use.

Besides the administration of alkaline remedies and the cautious use of colchicum, it is important in attacks of acute Gout to attend to the state of the intestinal canal and the skin. If the bowels be confined, some aperient must be given, and the selection of the drug should depend upon the peculiarities of the patient. If mere constipation exists, a simple purgative, as the compound colocynth ex-

tract, may be administered at night, followed by a saline aperient, as a seidlitz powder, or the effervescing citro-tartrate of soda and magnesia; or, if a more active dose be required, the common black draught can be substituted for these latter. If, however, the portal system shows evidence of congestion, and the function of the liver is disordered, some more powerful cholagogue will be useful, especially if the patient has been in the habit of taking purgatives. A small amount of blue pill or of calomel can be combined with the colocynth, or podophylline, in quarter or half-grain doses, may be substituted for the mercurial. It should be borne in mind that, in gouty habits, mercurials must be used with great caution, as in many cases there is a considerable susceptibility to their action, and very unpleasant consequences may follow their administration in repeated doses.

Saline purgatives are very desirable; many of them act remotely as antacids, and all tend to relieve portal congestion. A very useful combination, and one often employed in acute attacks of Gout, is a draught containing sulphate and carbonate of magnesia, to which colchicum alone or bicarbonate of potash and colchicum, may be added.

The function of the skin, if very defective, may be promoted by the use of a hot-air or vapor bath, and at the same time the acetate of ammonia may be given, combined with other remedies.

The action of the kidneys is usually sufficiently promoted by the saline treatment, especially if accompanied by the free use of diluents.

Blood-letting, in the form of venesection from the arm, was frequently had recourse to in former times; but this practice is now almost abandoned. The use of small bleedings has still perhaps a few advocates, and of such treatment I had much experience some years since. There can be no doubt that in some cases of very acute Gout, especially when many joints are implicated and fever runs high, speedy and marked relief is procured by taking a small amount of blood from the arm; but it is questionable whether even in such instances it would not ultimately have been better for the patient to have obtained the relief a little more slowly, and without the loss of so valuable a fluid as the blood. The doubtful advantage of the practice will be more apparent when it is stated that everything that produces lowering of the vital powers tends to engraft the disease more permanently upon the system.

A question now arises,—Although general blood-letting is undesirable, should not local depletion be resorted to? The appearance of a joint when acutely inflamed, the state of the tension and redness, the high temperature, and the exquisite pain, all seem to point to the necessity of, or at least the advantage likely to accrue from, the abstraction of blood from the part; and the indication has often been acted upon. My own experience quite accords with that of former observers as to the danger of the practice, and several instances have come under my notice of considerable and irremediable injury which has resulted from the use of leeches in these cases. I have frequently seen great-toe joints stiffened after a few attacks, when local depletion has been resorted to, and within the last eighteen months two remarkable cases, in which the patients have completely lost the use of both knee-joints from two or three attacks only: in both instances leeches had been applied very freely; in one more than thirty to each joint. I can with confidence warn those engaged in the treatment of an acutely-inflamed gouty joint never to have resort to this mode of combating the disease. It would seem that the abstraction of blood from the joint allows or favors the free deposition of the urate of soda in the tissues, and thus the ligaments become rigid, and ankylosis ensues. Although more or less stiffness is not infrequently seen as the result of long-continued gouty action in a joint where no local depletion has been employed, still, as a rule, the free movement of a joint is but little impaired by even numerous attacks of acute Gout.

As it has been shown that leeches should not be made use of in the joint affection, the next point is to consider whether any or what local remedies may be advantageously employed. If the pain and redness are slight, all that is necessary is to cover the part with flannel or some other light and warm clothing: this precaution is simply for the purpose of avoiding the chance of a chill. Should, however, the inflammation be very intense and the suffering great, carded cotton should be wrapped round the joints principally affected, and oil silk or gutta-percha sheeting so applied, that the moisture is retained, and by this means a kind of vapor bath is formed. Some care is necessary to insure the complete closure of the oil silk, so as to prevent the escape of vapor; for unless this is effected, the warm covering of cotton, instead of giving relief, heats and augments the pain.

As some patients are very intolerant of pain, it is at times desirable to apply anodyne remedies, and the most efficacious are belladonna and opium. I prefer a solution of atropia and morphia, dissolved in spirit and water, in the proportion of one grain of atropia and eight grains of hydrochlorate of morphia to the fluidounce; a small piece of lint may be dipped in

the solution and placed on the part, the oil silk being employed as above described. The tincture of belladonna and of opium may be used, but the solution of the alkaloids is much more cleanly and elegant. Aconite and its alkaloid have been proposed for lulling pain in Gout; but when strong, they may cause irritation of the skin.

Blisters have been used with advantage, when there exists great want of power in the system; possibly their value in chronic and asthenic Gout may be in part due to the fact of the serum withdrawing some of the morbid matter from the affected joint.

We have alluded above to the administration of colchicum, and mentioned some of the symptoms which may arise from its administration in full medicinal doses, and also its marked influence in controlling gouty inflammation.

Some practitioners have attributed the good effects of the remedy to its action on the bowels. That it often purges when given in full doses, and that it may give rise to a peculiar excretion from the bowels, is true; but it is equally a fact that marked and rapid relief frequently occurs from its exhibition when no appreciable influence on the intestinal canal can be detected; and, on the other hand, free purging can be induced in a gouty patient by other means, without the production of relief to the local inflammation. It may hence be safely inferred, that the peculiar influence of colchicum does not result from its purgative action. Others have been disposed to attribute the beneficial influence of colchicum to its action on the kidneys, and have regarded it as a diuretic, which not only causes an increased elimination of the watery portion of the urinary excretion, but likewise of the solid constituents, and more especially the uric acid. If these properties were possessed by colchicum, there would be little difficulty in accounting for its valuable influence in controlling Gout; but, unfortunately, clinical experience does not favor these views. It is true that some observers, as Dr. Christison and Dr. J. McGregor Maclagan and Professor Chelius, have made observations which at first sight would render it probable that colchicum increased the solid excretion, but as only single specimens of urine were taken, and no reference made to the total elimination in the twenty-four hours, a serious source of fallacy existed; the urine after the administration of colchicum might, it is true, have been higher in specific gravity and richer in urates, but this circumstance may have been due to a diminished secretion. Chelius's observations were made on patients recovering from gouty attacks, in whom it is not uncommon to find, for many days, a gradually increasing amount of uric acid without the administration of any medicine. From numerous observations[1] made some years since on the influence of colchicum upon the secretion of urine in gouty and other cases, I arrived at the conclusion that, in health, colchicum diminishes rather than increases the excretion of uric acid and urea by the kidneys, and that the elimination of the watery portion of the urine is often lessened, more especially when purging is caused by the remedy. It is probable that the statements as to the increase of uric acid have arisen from the fact that the analyses have been made on urine passed at some one period of the day only.

Having failed to discover any visible alteration in the principal secretions produced by the administration of colchicum, it is necessary to seek some other mode of explaining its action. That it is a sedative to the vascular system is a well-known fact, which has been fully proved by clinical experience; in subjects with weak hearts it causes temporary intermission of the pulse. Dr. Maclagan found on two occasions twenty minims of the tincture lowered the number of beats from eighty-seven to sixty-five and from eighty-four to sixty-two per minute respectively. This controlling power exerted upon the circulation, although it may explain to some extent the relief experienced from the drug, still is quite unable to elucidate the whole; for if the sedative action were the only effect, colchicum should be equally efficacious in acute rheumatism as in Gout; but that it is not so has been proved beyond doubt. Another explanation of the effects of colchicum has been proposed; namely, that its action is chiefly exerted upon certain tissues of the body, especially the ligamentous and cartilaginous, in the same manner as other remedies are known to affect particular organs, as belladonna the pupil of the eye, digitalis the heart, and so on; but the same objections hold good here as in the former case, for the action of the drug should be equally potent in controlling inflammation of the same tissues when not gouty in its character.

Treatment of Chronic Gout.—The treatment of the acute paroxysm having been sufficiently described, it remains for us to speak of the manner in which the chronic conditions of the disease require to be managed; if in acute Gout it is necessary to make the treatment dependent upon the state of the system and the idiosyncrasy of the patient, it is even still more so when the chronic forms are prescribed for. We have seen that a gouty fit, whether it occurs in the strong and

[1] Medico-Chirurgical Transactions, vol. xli. 1858.

robust or in the weak and spare habit, is dependent on the same proximate cause; yet that it may be excited by various circumstances in different individuals, for in one patient the state of the digestive organs, in a second the function of the skin, and in a third the secretion of the kidneys, may be principally at fault: and all these considerations must be taken into account when called upon to treat any one laboring under chronic Gout.

Value of Colchicum.—Colchicum is found equally efficacious in subduing the exacerbations in chronic Gout as in combating the early fits in the acute disease, due regard being paid to the strength of the patient, and the dose regulated accordingly.

It has been asserted, and the opinion is a very prevalent one, that the use of colchicum in the acute disease tends to cause the attacks to recur more frequently, and to induce a chronic state of the malady; but there are no good grounds for such an idea, unless the remedy has been much abused. It must be remembered that Gout, even when left to run its own course, and quite independent of medicinal treatment, has a powerful tendency to return, and the natural course of the disease should not be confounded with the effects of any treatment which may have been pursued.

It is important to disabuse the minds of both the profession and public of the prejudice against the guarded use of colchicum, as the permanent danger caused by allowing the inflammation to linger for a long period is far greater than any injury which the proper use of colchicum can entail. It is not improbable, if an attack of acute Gout is allowed to run a long course, that, at the termination of the fit, the patient is for a time more free from the disease; *i. e.*, the blood is purer than if the inflammation had been simply arrested without any care having been taken to rid the system of the morbid matter. Although colchicum given alone has a powerful influence in diminishing the subacute inflammations in chronic gouty cases, yet it may often be very advantageously combined with other medicines, and, amongst these, that which claims the first notice is guaiacum.

Value of Guaiacum.—This resin may be given either in the form of the mixture of guaiacum of the Pharmacopœia, in which the powdered resin is kept in a state of suspension by means of the acacia mucilage, or as a powder combined with aromatics, or in many instances, still more advantageously, as the ammoniated tincture of guaiacum made up into a draught.

Guaiacum sometimes acts on the mucous membrane of the alimentary canal as a purgative, but this occurs less frequently with the ammoniated tincture than with the powdered resin. This aperient action is often rather useful than not; but if it is not desirable, it may usually be prevented by the addition of a minim or two of laudanum to each dose. The resin evidently becomes absorbed, at least in part, and after it has entered the circulation acts as a stimulant to the smaller arteries and capillary system of vessels. It often promotes the function of the skin, and clinical experience appears to show that it has a specific effect upon the fibrous and ligamentous tissues, as well as on the mucous surfaces; it also increases the warmth of the extremities, and relieves pain connected with a languid circulation. Guaiacum may be administered for a long period of time without injury; I have had patients under my care who have taken it for a whole year. Within the last few years I have given this drug extensively, and with great advantage; it is especially useful in the asthenic Gout of old subjects, but to young patients it may also be given with benefit.

Value of Iodide of Potassium.—Another remedy of service in chronic Gout is the iodide of potassium. This salt undoubtedly possesses great power in controlling inflammation of fibrous tissues; its action on the periosteum is very marked in the case of nodes, also in painful neuralgic affections dependent upon an inflammatory state of the nerve coverings; it is more especially useful when the pains are increased at night and by the heat of bed. It is also useful in removing the recent thickening of the tissues around joints, but proof is still wanting of its possessing any power of causing the absorption of urate of soda.

In gouty inflammation, when fluid has been thrown into the cavities of the joints, and has been slow of absorption, the administration of the iodide of potassium has often appeared to be attended with great advantage.

Cinchona Bark and Quinine.—The preparations of bark and quinine possess an undoubted power of controlling inflammation, and within the last three or four years I have largely employed them for this purpose. With regard to the action of quinine, there are certain observations in relation to its physiological action which are of interest, and may also prove of therapeutic value.

Dr. Ranke has stated that quinine has the power of diminishing the amount of uric acid in the urine. To prove this, Dr. Ranke gave in one dose twenty grains of sulphate of quinia to a patient, and found that the excretion of the acid was only one-half the average: the influence of the dose continued for about two days.

In my own observations the average of the excreted uric acid during these days was but slightly under that which it had

been before the quinine was given. In one instance, for example, the average of uric acid in the urine for two days was 5·89 grains when no quinine was exhibited, and 5·37 grains for three days when the patient was taking eighteen grains of the sulphate each day, in divided doses. Assuming that Dr. Ranke's statement is correct, and that the elimination of uric acid is much lessened for two days after the dose, it is a matter of much interest to inquire if the effect is due to a diminished formation, or defective excretion from the kidneys. From my observations, I was inclined to ascribe the effect to the sudden and powerful impression of the drug upon the nervous system influencing the excretion of uric acid, and not to any decrease in its formation in the system. I shall, however, be unwilling to offer a strong opinion upon the subject at present, as I consider that further experiments are required. Quinine may be beneficial in controlling gouty inflammation, whether it produces one or other effect: of the influence of colchicum there cannot exist a doubt; still it has not been proved either to augment or diminish in any marked degree the elimination or formation of uric acid.

Yellow cinchona bark has been used in lieu of quinine, and where there is great vascular debility, the astringent principle of the bark appears to be of service.

It is advantageous to unite small doses of colchicum to the quinine; and when using the former drug in large doses, the addition of the latter is of service in preventing depression of the nervous system.

Constitutional Treatment of Chronic Gout.—Although due attention to the inflammation of the joints is of great importance in the management of chronic Gout, there is another object to be steadily kept in view, namely, the removal of the morbid condition of the blood, and the solubility of any uric acid which may fail to be eliminated.

It has already been shown that uric acid is thrown out entirely, or almost entirely, by the kidneys, and it has also been demonstrated that in Gout there is always some diminution of the uric-acid eliminating power, and often an almost entire suppression of this function. It follows from this, that one great object must be to increase this excreting power, and several remedies may be made use of to effect this, amongst which alkalies and salines stand out prominently.

Value of Alkalies and Salines.—These agents have long enjoyed favor in the treatment of chronic Gout, and not without reason; sometimes they are given in the form of ordinary medicine, sometimes in the form of natural or artificial mineral waters. It may be observed here that as chronic Gout is a disease which has usually been many years in becoming fully established in the system, so it is one in which benefit cannot be expected, except from a long-continued perseverance in some judicious plan of treatment; and when a mere exacerbation of inflammation has subsided, the cure must not be thought to be effected. Under these circumstances it is most desirable that the plan adopted should be simple, and neither disagreeable nor troublesome; and it will be found in practice that a patient will often persevere for an almost indefinite time with the use of mineral waters, when he would refuse treatment by the ordinary mode of administering drugs.

The alkalies and alkaline earths most commonly ordered are the salts of potash, soda, lithia, magnesia, and lime; and these are usually combined with carbonic or some vegetable acid, and occasionally with phosphoric acid.

If these bases are given in the caustic state, they act as direct antacids, and many of them influence the mucous membranes as sedatives, or, in strong doses, as irritants: hence potash, soda, and lithia are seldom given in the free state, unless there are special indications for their employment. When carbonates or bicarbonates of the bases are administered, the antacid effect is equally produced; but if combined with the vegetable acids, as the citric or tartaric, the alkalies lose their power of neutralizing acidity in the stomach. After absorption into the blood, and elimination by the kidneys, the alkaline reaction is equally produced by the vegetable salts as by the carbonates or free alkalies, for the acid is broken up *in transitu*, and the base eliminated in the form of a carbonate. It is, therefore, desirable to select either a carbonate or neutral salt, according as it is thought advisable or not to produce an alkaline effect upon the stomach.

In making a selection of the base, several circumstances must be taken into consideration, and especially the organs and functions peculiarly influenced by the different alkalies and earths.

Potash and its salts act especially on the kidneys, causing not only an alkaline state of the urine, but usually a marked increase in the secretion itself.

Observation has repeatedly shown that potash has a marked effect in augmenting the quantity of urine; its effect on the excretion of the different organic solids has not been clearly made out; no uniform results have been obtained sufficient to show its influence upon the uric acid; some experiments appear to indicate an increased, some a decreased excretion of this acid, and others, again, that it was unaffected. There are many difficulties in the investigation; in alkaline urine, uric acid is soon decomposed, and if the

quantity of urine is much increased, a portion of it may be lost in the analysis. Potash and other alkalies may act in more than one way; not only may they increase the elimination of uric acid, but they probably facilitate its destruction in the system, and certainly aid in imparting solubility to it in the blood.

Soda salts have less influence upon the kidneys; they act less powerfully as solvents of uric acid; but, on the other hand, they appear to aid the secretion of bile, or act as hepatic alteratives. They are indicated in gouty cases accompanied with marked derangement of the liver.

Lithia salts have only been used as internal remedies during the last six years. They are active diuretics, more active than salts of potash; they are also powerful solvents of uric acid, and on account of the very small equivalent of the metal, caustic lithia or its carbonate possesses great neutralizing power for acids. The urate or lithate of lithia is by far the most soluble of all the salts of uric acid.

When first introduced by the author as a remedy for Gout, lithia was supposed to exist only in a few minerals, but more recently its presence has been discovered in a variety of substances. It has been found to be a constituent of the human body, of many plants, and can be shown by means of the spectrum analysis in the ashes of the blood, and even of a cigar; it is found, also, in the waters of several mineral springs, especially those of Baden-Baden; also in the springs of Carlsbad, Aix-la-Chapelle, Marienbad, Vichy, &c.

Salts of lithia, especially the carbonate and citrate, have now been employed extensively, and apparently with considerable success. It is, of course, a matter of extreme difficulty to form a very strong opinion upon the value of any drug, especially when the effects are not at once evident to the senses, and are long in being produced; but lithia appears to have many desirable qualities; if the opinion of patients can be relied upon, lithia salts taken for a long period in a very dilute form have the power of preventing gouty paroxysms in chronic cases, and some evidence has been afforded of their power of rendering joints more movable, and of causing some solution and absorption of chalky matter which has been already deposited. Lithia salts certainly act rapidly and powerfully in preventing deposition of urates and uric acid in the urine, and in the calculous tendencies of many gouty subjects must necessarily prove of much value.

The dose of carbonate of lithia may be from five to ten grains dissolved in aerated water, or the citrate may be given in doses of from eight to twelve grains or more.

Before leaving the subject of the alkalies and their salts, it may be useful to allude to the value of administering these preparations in a very diluted form. Many soluble salts, if given in the form of concentrated solutions, will act as purgatives, whereas if very freely diluted they produce diuresis; and it must be at once evident to any one who considers the subject, that the introduction of a large amount of fluid into the system has the effect of rendering the blood more capable of holding sparingly soluble matters in solution, and of augmenting the various secretions from the body, especially the urine, and hence of facilitating the expulsion of any such matters from the system. It is necessary also to select proper times for the exhibition of these remedies; as the object is to have them rapidly absorbed, they should be given on an empty stomach, at least an hour before food; if taken at or soon after a meal, they are apt to cause distension and discomfort, and at the same time they are very slowly absorbed: any amount of free alkali, when taken at the time that digestion is going on, impedes the process very seriously by neutralizing the free acid of the gastric fluid. This is a fact too frequently overlooked in practice.

Small doses of salines, if their use is long persevered in, are preferable to large ones, as they produce no disturbance of the digestive process, act more freely on the kidneys and skin, and are not likely to cause debility.

Magnesia and its carbonates have been long used in the treatment of chronic Gout, and there can be no doubt that these salts are of value in many cases. Magnesia forms a moderately soluble salt with uric acid; it is also an alkali, and acts both as a direct and remote antacid; it is useful as an adjunct, and especially in instances in which there is great acidity in the intestinal canal, and at the same time a sluggish state of the bowels. The salt formed in the stomach by its union with the acid produces a purgative effect, which tends to relieve the portal circulation, and often aids indirectly the function of the kidneys. Carbonate of magnesia dissolved in excess of carbonic acid is an elegant form of administering the remedy.

The lime salts offer no special advantage. Lime water may be employed as an antacid if there is much tendency to diarrhœa, as the salts of lime have a constipating effect.

There are some other salts, not yet alluded to, whose virtue depends partly on the acid contained in them; for example, the phosphates of soda and ammonia.

Both these salts increase the solubility of the urate of soda, and may be used with advantage in certain instances. The

phosphate of ammonia has been employed in many cases of chronic Gout, and clinical observation appears to show that it is useful in preventing paroxysms, probably by keeping the blood in a purer state.

The result of my own experience of the use of the phosphate of ammonia is favorable; it is especially indicated in cases in which the circulation is feeble, and the function of the skin impaired: this remark applies equally to other ammoniacal salts.

Treatment of Errors of the Digestive Function. — As the stomach and other parts of the digestive apparatus are generally affected in chronic forms of Gout, it is of importance that their condition should be carefully attended to; purgatives, stomachics, and alteratives are the remedies resorted to for correcting any morbid state of these organs.

Value of Purgatives.—It has been already stated that free purgation alone will not rapidly cure gouty inflammation, and that it is impossible to explain the effects of colchicum upon this idea; that, for example, sulphate of magnesia, although it causes a free watery action, will not relieve in the same manner as colchicum, even when the latter drug produces no appreciable action upon the bowels. Purgatives, however, are often of much value, especially when there is a portal congestion present, and the occasional use of the compound colocynth extract, or some such preparation, is usually of great advantage. Mercurials should, as a rule, be avoided, as they are peculiarly prone to cause ptyalism in gouty subjects, and as their frequent use lowers the powers of the system.

Value of Stomachics and Tonics. — If there exists an irritative form of dyspepsia, accompanied with flatulence, acidity, and heartburn, this is generally relieved by the administration of free or carbonated alkalies; if pain or palpitation is present, a few drops of hydrocyanic acid may be added to each dose, taking care to relieve any sluggish condition of the liver and bowels. These alkalies are often advantageously combined with some bitter stomachic, as the juice or extract of taraxacum; or, if there appears to be great want of tone, the more powerful bitters, as chamomile, gentian, chiretta, quassia, &c. &c. If much flatulence be present, then ginger or capsicum may be likewise added.

Ash-leaves in the form of an infusion have been also recommended, and clinical proof afforded of their efficiency in chronic Gout. I have frequently made use of them, and with advantage; but the amount of infusion taken each day has always been considerable, and the action of the diluent must not be forgotten.

Value of Ferruginous Preparations —In the majority of instances iron salts are not indicated, but at times cases are met with in which their administration is attended with striking benefit. They prove most useful when the blood is impoverished from the diminution of the red corpuscles, and the circulation enfeebled from weakness of the walls of the heart, and where the whole nervous system has become exhausted. It is often a good plan to combine these with small doses of the extract of colchicum.

The selection of the ferruginous preparation should be made according to the peculiar requirements of the patient: if only the hæmatinic property is wanted, reduced iron (ferrum reductum) may be prescribed; if the astringent influence is required, the sulphate may be made use of.

Value of Diaphoretics or Sudorifics.— Friction of the skin must not be overlooked in the treatment of chronic gouty cases; we must remember, that although there is no elimination of uric acid from the healthy surface, still the skin gives off a large amount of some other acid, which, when retained, renders the blood serum less alkaline.

The salts of ammonia are useful when the skin is particularly in fault, especially those in which the alkali is combined with a vegetable acid, such as acetic acid; but it is better in most cases to promote the due performance of the cutaneous function by insisting upon a sufficiency of exercise, and the free use of water to the skin, combined with friction, and wearing flannel next to the skin.

Hot-air and vapor baths are also valuable, and, when it can be borne, the Turkish bath occasionally; hot salt-water baths may also be made use of.

Treatment of the Local Affection.—One of the slighter forms of inconvenience, arising from long-continued gouty inflammation in any part, is the production of œdema, a symptom evidently depending on local debility or weakness of the vessels of the affected parts, usually the lower extremities: this is often much increased by disease of the kidneys, or sometimes of the heart. When the swelling is dependent simply on local weakness, it is best treated by the use of some mechanical support, as the elastic stocking and slight friction, with or without some stimulating and lubricating application; the limb should be elevated, so as to facilitate the free return of blood to the heart. Œdema depending on a kidney or cardiac disease must be specially treated.

A far more distressing complication, and one far more difficult to deal with, is that arising from chalk-stones, and the distortion and rigidity of the joints produced by deposition within the structure of the articulations.

If chalk-stones appear on superficial

parts, they may be of little inconvenience, and are often spontaneously removed; for example, if the nodules on the ears of gouty patients are observed for some few years, a considerable change is generally noticed; they may escape from rupture of the cutis, or if they increase in size, so as to cause any inconvenience, they may be punctured, and by this means dispersed, the contents escaping either in the semi-liquid or solid state.

There is no danger attending an operation upon the ears, nor in many other situations, when the chalky concretions are small, and especially when in a liquid state and very superficial, provided the patient is at the time in a tolerable condition of health; but the case is far otherwise if the chalk-stones are large and solidified, and specially if their ramifications are deep-seated. In such cases the removal is apt to be attended with serious and even fatal consequences, arising from the extreme difficulty in the healing of the sore, or the supervention of a low form of erysipelas. The greater the amount of kidney affection, the greater the danger of operating. Not infrequently, when the deposits approach the surface, they burst spontaneously and abscesses are then formed; these are often very difficult to heal on account of the matter being deep-seated. I have known cases in which such abscesses acted as a kind of safety-valve, and their closure has been immediately followed by a paroxysm of gouty inflammation. The best mode of treating these abscesses is to keep upon them a water dressing, as long as there is a free flow of the matter; then to have them dressed with some stimulating ointment, and occasionally either to apply a lotion of sulphate of zinc, or touch the surface with a stick of lunar caustic. Now and then, if the opening becomes nearly closed, at the time that there is a large accumulation of the urate of soda behind, it is advisable to enlarge the opening by a slight incision.

It has been thought by some physicians, both in ancient and modern times, that the deposits of urate of soda which occur in Gout are capable of being dissipated by external applications, and alkaline preparations have usually been employed for this purpose, such as solution of potash, or soda, or lime. I have in some cases had compresses, steeped in a solution of carbonate of lithia, kept on the parts for a long time, and patients have assured me that they have found decided benefit from them; but although such a solution exerts a very powerful solvent action upon the deposits when removed from the body, it is difficult to imagine how they can act through the skin; but as such treatment can do no harm, it is worthy of a further trial.

When joints are much stiffened from gouty inflammation, considerable relief is obtained from the use of a blistering liquid. This appears more useful, and, upon the whole, less annoying to the patient than iodine paint; and it usually removes any infused liquid, and chronic inflammatory action, which may be lingering about the part. After all tenderness has disappeared, movement may be attempted, very gently at first, but gradually increased; the joints may also be rubbed with some slightly stimulating liniment, or with strong salt and water. In the treatment of these cases it must always be borne in mind, that the rigidity is often due to interstitial deposit in the ligaments, and that a restoration of these structures to a healthy state can hardly be hoped for; still, as this condition may often be complicated with a chronic inflammatory action, it is always advisable to attempt the removal of such complications.

Treatment of the Irregular Forms of Gout.—The treatment of irregular Gout must necessarily be very different in different cases, and it is a matter of extreme difficulty to lay down any precise regulations for the guidance of the practitioner; however, a few general rules may be advantageously given.

Should metastasis take place to any important organ, it seriously implicates its functions, and therefore our main object must be to take such steps as will restore this function; and, as the metastasis usually follows the sudden suppression of gouty inflammation of some joint, the one very essential part of the treatment consists in the endeavor to bring back articular inflammation. This object is best effected by the application of heat or counter-irritation to the extremities, as by hot bottles, sinapisms, &c. At times we may treat the part itself, especially if there is evidence of inflammatory action existing in it; this may be effected by leeches, blisters, and mustard poultices; local depletion, however, is rarely necessary. The peculiar condition of the system, or the existence of the gouty diathesis, must not be overlooked. A question of no little importance at once arises when the diathetic condition is considered; it is that which relates to the value of colchicum in irregular Gout. Sir Henry Holland is of opinion that it can be employed with advantage, and as far as my own experience goes I quite coincide with him, although unable to explain its action. This want of knowledge applies with equal force to the action of colchicum in the genuine articular form of the disease.

In metastic Gout of the heart and lungs, cardiac stimulants, as ammonia, ether, and other anti-spasmodics, are especially indicated. If the bladder is affected,

belladonna and henbane may be advantageously used to diminish spasm; in short, remedies directly influencing the implicated organ may in all cases be resorted to, at the same time that the means tending to reinduce the articular inflammation should not be neglected.

Value of Mineral Waters in Gout.—Mineral springs are frequently resorted to by gouty subjects, and it is important that the medical man should know when to advise and when to oppose such a step, and likewise the waters most suitable for different cases.

Although it cannot be denied that many patients receive great benefit from the proper administration and use of these waters, still it must be allowed that their action is not always beneficial, and that in some cases it is very injurious.

All mineral waters have one action in common; it is that of water itself; and there is little doubt that the value of this agent when properly employed is considerable. The waters of some of the mineral springs of great reputation contain little foreign matter, and must owe most of their efficacy to the water alone. The other waters employed in gouty cases contain either alkaline carbonates, chlorides, or sulphates. Some of the waters are impregnated with sulphuretted hydrogen, and another class owe the chief of their powers to the iron which enters into their composition. Many of the springs are of an elevated temperature; some of mean heat; others cold.

All mineral waters rich in saline matters, if taken too freely, usually set up a febrile disturbance or crisis; the system becomes oppressed, there is a feeling of heaviness, languor, or agitation, and this is followed by loss of appetite, thirst, a furred tongue, and heat of skin, sometimes by vomiting and diarrhœa. Such symptoms are probably due to the blood becoming saturated with the saline matter, from the excreting organs being unequal to the task of eliminating the whole quantity introduced during the treatment.

The different springs of Vichy are all rich in carbonate or bicarbonate of soda, containing about forty grains to the pint: some have the temperature of 101° Fahr.; others are cold. When taken internally in even moderate doses, they cause the urine to be neutral or alkaline, without affecting the transparency of the fluid; when employed in the form of the bath, the effects appear to be very similar. From the soda contained in them they probably act upon the liver; and from the amount of liquid absorbed, and the temperature at which the water from many of the springs is drunk, they also influence the function of the skin.

Vichy waters appear to be adapted for the treatment of Gout when it occurs in strong subjects in whom the function of the liver and digestive organs is at fault, and are contra-indicated in very chronic cases, especially if there is a tendency to the rapid formation of chalk-stones, or if the powers of the system have become much enfeebled: my own experience is that, in this latter class of cases, they rather tend to favor the formation of these concretions.

The internal exhibition of the waters is usually accompanied with the use of the bath; if they disturb the stomach, the bath alone should be employed.

Wiesbaden waters contain a large amount of chloride of sodium, and are of a high temperature, 160° Fahr. They are less debilitating than Vichy waters, and more stimulating to the various functions: they consequently are more adapted for cases in which the circulation is sluggish and the secretions deficient; also in cases of rigidity from thickening of the textures. They are powerless in removing any solid deposition of urate of soda.

Aix-la-Chapelle is frequently resorted to; the waters are slightly saline compared with those of Wiesbaden, high in temperature, 135° Fahr., and in addition to chloride of sodium, contain some carbonate and sulphide of sodium, with free sulphuretted hydrogen. They act as stimulants to the secreting organs, and more especially to the skin; they are indicated in cases in which the skin is in fault, and they have also been found useful in removing rigidity of the joints.

The waters of *Aix-à-Savoy* resemble closely, as far as the sulphur is concerned, those of Aix-la-Chapelle.

Carlsbad waters are in great vogue in the treatment of Gout. They are rich in sulphate of soda, and contain likewise carbonate of soda and chloride of sodium; of a high temperature, 167° Fahr. They often cause purgative action, and likewise give activity to the kidneys and skin, and are useful in cases accompanied by deficient action of the bowels, with a congestive state of the liver; but should be avoided by weakly patients.

The waters of *Baden-Baden* are saline, and from recent analysis are said to be rich in lithia. Dr. Ruef has affirmed that they have proved very useful in Gout, and possess the power even of removing visible deposits of urate of soda.

The waters of several other springs, as of *Kissingen*, *Marienbad*, *Homburg*, *Ems*, and several other localities, contain saline matters, and have occasionally been used in the treatment of gouty conditions of the habit.

The waters which possess but little solid matter, and which have acquired a reputation in gouty cases, are those of *Wildbad*, *Teplitz*, *Gastein*, *Buxton*, and *Bath*.

They are all of somewhat elevated temperature, are chiefly used in the form of the bath, and appear to be peculiarly adapted for the treatment of the disease in the old and infirm. According to numerous observers, great benefit has been often experienced from their employment.

The following rules may serve as a guide in prescribing the use of mineral waters in gouty cases :—

1. They should not be employed when there exists any appreciable amount of organic disease either of the heart or kidneys.

2. They should be avoided when an acute attack is either present or threatening.

3. The particular water should be selected according to the nature of the case. When the patient is robust, and of full habit, the alkaline springs; when torpidity of the bowels predominates, the purgative waters; when there is a want of vascular action, the saline waters; when the skin is inactive, the sulphur waters; lastly, when debility prevails, then the more simple thermal waters should be chosen.

4. In all cases the use of the water should be cautiously commenced, and care should be taken not to oppress the stomach by giving too much liquid, nor to induce debility or other injurious effects by allowing too long a sojourn in the bath.

5. In every instance, when practicable, it is advisable to avoid producing the so-called "crisis," for when febrile disturbance is set up in the system, the secretions are checked, and an acute paroxysm of Gout is almost always induced; it is far better to take a prolonged than a too severe course of a mineral water.

Diet and Regimen in Gout.—The diet in the treatment of the different forms of Gout is of great importance, far more so than in the majority of diseases. When the affection is acute in character, and the patient robust, he should be confined for a few days to a diet consisting of little more than farinaceous food and diluents; and this kind of food may be persevered in until the inflammation shows a decided tendency to abate, the thirst diminishes, and the appetite begins to return. Under the term farinaceous food are included bread, arrowroot, sago, tapioca, and such-like substance; to these may be added milk; while water, and toast-and-water, may be indulged in without restraint; provided the liquids are taken upon an empty stomach. Stimulants are scarcely required under these circumstances; but if the patient has been accustomed to live freely, a little brandy may be taken with the solid food; even a moderate indulgence in wine or malt liquor will keep up the gouty inflammation for an almost indefinite period.

When febrile disturbance has abated, a more generous diet may be allowed—at first fish, then fowl or game, and at last ordinary meat. In strong persons it is desirable to keep a moderate curb upon the appetite, for fear of inducing a recurrence of the inflammation.

As soon as possible exercise may be resumed, and it is most desirable that this should be persevered with daily; but if the lower extremities are much affected, there is a fear lest too great an amount at any one time may excite local irritation in parts which have recently been inflamed.

As yet it has been assumed that the gouty attack has occurred in a strong individual, and is of an acute and sthenic kind; but this may not always be the case, for even a first fit may find a patient broken down in constitution, and quite unable to bear the least withdrawal of nourishment or stimulus. Under these circumstances, care must be taken to give such nourishment as the digestive organs can easily assimilate, as beef-tea, strong but plain soups, eggs, milk, &c.; and, when practicable, to confine the stimulus to some distilled spirit, as brandy or whisky, giving these only to the extent of keeping up the action of the heart and the efficiency of the circulation.

A few general rules may be advantageously given, which will serve as a guide for the treatment of gouty patients in general, and apply more especially to such as suffer from the chronic forms of the disease.

It is desirable to regulate the amount of food, so that the system shall be fully nourished, and the strength kept up as much as possible, but anything taken beyond this is decidedly injurious, as it tends to oppress the digestive organs, and induce debility rather than vigor of frame.

As to the character of the solid diet, it may be stated, that every article which causes unpleasant symptoms, recognizable by the patient himself, should be studiously avoided; and hence the less what are termed "made dishes" are partaken of the better: the same remark applies to all rich and highly-spiced food, and to anything that tempts the person to take more than he otherwise would. Articles of animal food of which the texture has been hardened, as salted meats, hams, and so on, are less easy of digestion, and should be discarded, as also veal and pork, which are much less easy of digestion, than mutton and good beef; white fish is generally digestible, as also fowl and game.

There should be a due admixture of animal and vegetable food; it is an error to suppose that an animal diet necessarily tends more to the formation of uric acid than a vegetable one. The tortoise, feed-

ing on a simple lettuce, excretes a large quantity of urate of ammonia, far more in proportion to the weight of the animal, than is excreted by the dog exclusively nourished with meat.

Vegetables, as potatoes, greens, and the like, may be partaken of with advantage; the soluble salts which they contain are of value in keeping up the activity of the secreting organs.

The same remarks hold good with regard to soft fruits when partaken of in moderation, as strawberries, grapes, and oranges; also other fruits when stewed or baked, as apples and pears; but these latter, as likewise plums and stone fruit in general, should be avoided in a raw state. Extreme moderation should be exercised when saccharine fruits are eaten, as sugar is liable in many subjects to lead to the production of acidity, and hence favor the development of Gout. The same precaution is necessary in reference to the addition of sugar to other articles of diet.

As to beverages, both tea and coffee may be taken if they do not disturb the nervous system. At one time it was supposed that the latter was prophylactic against Gout, seeing that the Turks enjoyed an immunity; but, if strict Mahometans, they do not take alcohol in any shape, and hence avoid its most powerful cause.

It is an important question to decide whether alcohol is to be ever allowed, and, if so, to determine the form which is best adapted for the patient.

All malt liquors should be eschewed, as they almost always cause an increase of dyspepsia, and, if at all strong, have undoubtedly a very powerful influence in inducing the disease and in keeping up a paroxysm.

Strong wines will also prolong an attack to an almost indefinite length of time, and if they are moderately indulged in will often lay the foundation of the gouty diathesis.

The wines to be carefully avoided are port, sherry, madeira, and any in which the fermentation has been checked by the addition of alcohol. If wine is taken at all, that which is best adapted for the majority of patients is a sound claret—one free from sugar and without acidity. When red wine does not agree with the stomach, then hock or moselle may be substituted, or even a light and dry sauterne or chablis.

The beverage best suited for those of a strongly-marked gouty diathesis is undoubtly French brandy, taken in very limited quantities, and freely diluted with water.

Whisky, hollands, or gin, may in many cases be substituted for brandy; but the two latter should be avoided if there is any appreciable amount of kidney disease, or at least should not be taken without advice. The distilled spirits should only be used at the meal, and from one to three ounces may be daily allowed, the amount depending upon the former habits of the individual.

If Gout has become developed at a very early age, and the youth strongly inherits it, a question arises whether it would not be desirable to advise an entire abstinence from alcoholic drinks. Such a step would be the most likely to check the further progress of the malady.[1]

Exercise must be enjoined, for it is of the highest importance, and without it all our endeavors may prove futile. The kind of exercise must be adapted to the peculiarities of the patient; walking and horse exercise are equally useful, and may be conjoined with advantage.

Fresh air is of great importance, and in many instances a complete change during the winter and spring to some warm and dry climate will enable the patient to escape an attack.

All violent exercise likely to cause exhaustion, all severe mental application and late hours, should be studiously eschewed.

In concluding the subject of the management of Gout, the author's opinions may be thus summed up:—

1. Gout in its acute form is quite as controllable, and as much under the influence of remedies, as any other inflammatory affection. The duration of the paroxysm and the amount of injury to the joints depend much upon the treatment.

2. The more chronic forms of Gout, which are met with in every degree of severity, are likewise under the control of the physician, if not for their radical cure, yet for so much relief as will enable the patient to enjoy life, and prevent further increase of the mischief, so liable to ensue if the disorder is allowed to run its own course, and more especially if recklessly tampered with.

3. As gout is a disease which is not only apt to return with increased severity, but to acquire a firmer hold on the constitution at each visitation, it is a matter of serious moment to consider whether it may not be prudent in the intervals of the attacks, not only to regulate the diet and regimen, but even to have recourse to means, scarcely to be called medicinal, by which the blood may be kept free from the impurities which lead to the production of the paroxysms.

4. The treatment of Gout founded on Cullen's aphorism, of trusting to patience and flannel, is to be highly deprecated.

[1 This recommendation may be very advantageously extended to *all* gouty subjects, unless greatly debilitated.—H.]

It may indeed be argued that it is the natural treatment, and that nature is a sure guide; but it must be remembered that man living in a civilized state is not in a normal condition, or in all probability he would never have acquired the disease, and that when suffering from a disorder so acquired, he must be content to have recourse to artificial remedies. If he could entirely lay aside his usual habits, and follow in all respects the dictates of nature, there would probably be little need to seek relief from medicine.

5. Although a plan can be sketched out which is applicable to the majority of cases of Gout, still each individual case not only exhibits its own peculiarities, and becomes a separate study, but likewise demands, in certain respects, a separate treatment. The neglect of this consideration is apt to lead to a mere routine practice, closely bordering on empiricism.

RHEUMATOID ARTHRITIS.

By Alfred Baring Garrod, M.D., F.R.S.

Definition.—A form of inflammation of the joints, accompanied with but little febrile disturbance, and distinguished from gout and rheumatism by its progressive character, by the peculiar morbid changes which it induces, and by the absence of any known morbid state of the blood.

Synonyms.—Rheumatic Gout is the name commonly given to this disease, but equally applied to other joint affections. Dr. Adams uses the term Chronic Rheumatic Arthritis. Dr. Todd included it under Chronic Rheumatism of the Joints. It has been called Nodosity of the Joints by Haygarth and Heberden; Usure des Cartilages articulaires by Cruveilhier; Rheumatism Noueux, by Trousseau and other French writers. The term Rheumatoid Arthritis was applied to this disease by the author in 1858.

History of Rheumatoid Arthritis.—It will be essential to the clear understanding of this subject that some explanation be first given of the name proposed to be employed to designate this disease, and the reason of its adoption; and to give reasons for the rejection of the many terms which have been used at different times. The term "rheumatic gout" is one which is very commonly employed, both by the profession and the public; but it is difficult to arrive at its true significance, seeing that but few have described it as a separate disease: the term has also been used to signify very different diseases. It is not uncommon to hear gouty patients say they are suffering from rheumatic gout, simply because the disease, which for many years was manifested in the feet only, now implicates other joints, as the elbows and hands; in fact, they regard their malady as gout when it is confined to the feet, but as rheumatic gout when it affects the upper extremities.

Sometimes the subacute forms of true rheumatism are designated as rheumatic gout, and more especially if the smaller joints are the seats of the attack. There exists, however, a third disease, distinct both from gout and rheumatism, to which the name is more frequently applied: it is this malady which we have called Rheumatoid Arthritis; and it is this which will engage our attention in the present article.

The name "chronic rheumatic arthritis" has not been employed, partly from the fact that the disease sometimes assumes an acute character,—if this were the only objection, it might be argued that we could designate this form by the name of "acute rheumatic arthritis,"—and partly because the name rheumatic arthritis implies that the disease partakes of the nature of true rheumatism, which we believe is not correct. The same objections, of course, apply to the words "chronic rheumatism of the joints."

"Usure des cartilages articulaires" (wasting of the articular cartilages) is an expression limited in its meaning; it only expresses one of the morbid changes which result from the disease.

"Nodosity of the joints," and "rheumatism noueux," express the presence of a frequent alteration in appearance, caused by the affection—one not constantly found.

"Morbus coxæ senilis" could not be used except when one particular joint is implicated, and it is a matter of some doubt whether this form of the disease has the same pathology.

The term "Rheumatoid Arthritis" has been employed for the following reasons:—

The disease is one chiefly affecting the joints, and is of an inflammatory character; hence the name arthritis. It is also one which, at least in its early stages, produces external changes closely resembling those caused by subacute forms of rheumatism; but as it can be shown that the nature of the affection is not the same as that of rheumatism, the prefix "rheumatoid" instead of "rheumatic" is sufficiently expressive.

As the word typhoid is allowed for the purpose of designating a form of fever somewhat resembling, but not identical with, typhus, so no objection can be raised to the use of the prefix rheumatoid, when it is intended to signify that the articular inflammation, although not of the same nature as rheumatism, yet resembles it in some of its characters, and more especially in those which are readily appreciated by the senses.

With regard to the history of our knowledge of Rheumatoid Arthritis little can be said. It is only since the time of Haygarth that it has been looked upon by any pathologist as an independent disease, and even at the present time it is often described under the name of chronic rheumatism, rheumatic gout, &c., and classed as a variety of some other affection.

Description of Rheumatoid Arthritis.—Rheumatoid Arthritis is met with either as an acute or as a chronic disease: the former is much less frequent than the latter; in fact, it has only been described within the last few years; it will, therefore, under the circumstances, be desirable to reverse the ordinary mode of treating such subjects, and to describe first the more common form, namely, chronic Rheumatoid Arthritis.

Chronic Rheumatoid Arthritis.—The affection may occur in both sexes, and at almost every age, and its invasion often assumes a form very similar to that about to be described. A young woman has become, from some cause, decidedly out of health; perhaps from menorrhagia or leucorrhœa, or from hemorrhage during parturition, or from prolonged anxiety or physical fatigue; she is exposed to cold, and, after a few days, feels some pain in the knee; there is slight swelling and tenderness; perhaps the temperature is a little elevated; simple rest to the joint may be followed by relief, or even a cure, for the time, the swelling abating, and the tenderness and pain vanishing; or perhaps this result may have been expedited by the application of a blister, or some other form of counter-irritation.

After a few weeks or months, as the case may be, perhaps from a second chill, another joint, or even that which was previously implicated, becomes affected, and a similar train of symptoms arises, but with this important exception, that, in all probability, the inflammation does not again subside, but continues fixed to the joint, and gradually extends to others. During this time there may be no appreciable constitutional disturbance beyond the general ill-health above noticed, but in some cases dyspepsia or nervous symptoms are exhibited.

The disease, if unchecked, travels over the whole body, affecting almost every articulation of the limbs, and causing much deformity and distortion, from the enlargements and contractions which it produces; nor are its ravages confined to the limbs alone, but other joints may be attacked, especially the temporo-maxillary articulation, causing the closing of the jaw; the upper cervical vertebræ may likewise be involved, and the neck become fixed; and thus at last the patient is rendered crippled and altogether helpless throughout the remainder of life.

The above example is, indeed, one in which the disease has effected all the mischief it is capable of; fortunately, it does not always proceed to this length, but is arrested at some stage or other of its progress, and then only a limited amount of distortion is induced.

The deformities produced by Rheumatoid Arthritis are not altogether characteristic of this disease, for they are produced likewise by chronic gout; but in their advanced conditions the separation of the one disease from the other is usually a matter of no difficulty.

The changes in the arms and hands, when severely affected, are of the following kind: The elbow is flexed, perhaps at an angle of 35° from full extension; the forearm in a semi-pronated position; the joint is also much enlarged and misshaped, more or less rounded from the alteration and hypertrophy of the heads of the bones, as well as of the soft tissues. The wrists are rigid, almost straight, and scarcely admit of motion in any direction.

The hands are usually thin, from the absorption of fat, and from the wasting of the other soft tissues; the extremities of the phalanges are nodular, as also the heads of the metacarpal bones. The fingers are usually turned outwards and their joints rigid, often completely fixed. As a rule, the metacarpo-phalangeal articulations of the fingers are flexed, and the first phalangeal extended, causing the second phalanx to be thrown backwards; the second phalangeal joint is also flexed. One, or even every finger in a hand, may be thus altered. The phalangeal joint of the thumb is usually extended, or bent backwards. Sometimes the nodose condition is well marked, but not infrequently it is but slightly developed; complete dis-

location of some joints is occasionally met with. The knee is generally much enlarged and rounded in the same manner and from the same causes as the elbow; it is commonly half flexed. Sometimes there is evidence of liquid effusion, but in the later stages this may be wanting.

When the hip is affected, the thigh becomes flexed, sometimes abducted, with the foot everted; at other times adducted, with the foot turned inwards; not infrequently there is felt over the large joints a sensation as though the bones were loose.

As a rule, the hands become crippled and distorted at an earlier period than the feet. Though frequently attacked, the jaw and neck seldom become fixed.

An affection which, if not identical with Rheumatoid Arthritis, at least closely resembles it, was first made known to the profession by Heberden, under the title of "digitorum nodi," and described by him as consisting of little hard knobs, about the size of a pea, situated upon the ends of the fingers, where they remain through life, being usually attended with little or no pain, and, though they cause but slight inconvenience, are decidedly unsightly. Heberden thought that they had no connection with gout, seeing that they occur in persons never afflicted by that disease; but Dr. Begbie considers them of a gouty character, as they are frequently met with in that diathesis.

The disease is sometimes confined to the extreme ends of the fingers, but now and then extends to the other small joints. When examined, the nodular feeling and the peculiar appearances are found to be owing to hypertrophy and other alterations in the epiphyses of the phalanges, and, except in position, and the small size of the affected joints, do not appear to differ from the enlargements and distortions above described as occurring in other situations.

Ladies are often much concerned at finding these nodules on their fingers, and are willing to take any amount of trouble to arrest the progress of their formation and to attempt their removal.

Acute Rheumatoid Arthritis.—Now and then cases are met with which, in most of their symptoms, closely resemble acute rheumatism; several joints are attacked, the swelling is considerable, there is distinct increase of temperature of the affected parts, with pain, tenderness, and redness. In these instances, constitutional symptoms, as thirst, loss of appetite, heat of surface, a rapid pulse, and other evidences of febrile excitement, are often observed. There are, however, wanting some of the characteristics of rheumatic fever—namely, the profuse sweating and the proneness to acute inflammation of the internal and external membranes of the heart, so common in acute rheumatism, and likewise the erratic disposition or tendency of the inflammation to fly from joint to joint. Between cases of genuine acute Rheumatoid Arthritis and those of the very chronic varieties there is every intermediate shade of difference.

The only real difficulty in these cases is to determine whether the acute disease is true Rheumatoid Arthritis, or whether it is genuine rheumatism which has acted as the exciting cause of the former affection.

As the acute disease is so little known and recognized by the profession, it may be well to give an illustration, and the following case may be taken as a typical example.

A lady, forty-two years of age, when living in Australia, in the bush, was confined, and being unable to procure a good supply of cow's milk was induced to nurse her child for a period of twenty months: at the same time she herself had but a very deficient amount of meat. By these means she was reduced to an extremely weak state. After a short time she noticed that some of her joints became affected; at first the knees, then the ankles, afterwards the elbows and wrists, and, lastly, many of the small articulations of the hands. These parts were painful, somewhat swollen, hot, and tender, but the local symptoms were never intense, nor was the constitutional disturbance very great; that is, there was no high degree of febrile excitement. After a few weeks some of the joints were much injured; the knees, although reduced in size from the absorption of the fluid, could neither be fully extended nor flexed, and the patient was soon unable to stand by reason of their rigid condition; the movement of several of the other joints was also limited, although in a less degree. The causes of the debility being removed, the patient soon gained strength and flesh, and the tendency to the joint affection passed off, but not without having inflicted irremovable injury.

Diseases caused by the Rheumatoid Diathesis.—In both gout and rheumatism, symptoms which may be termed irregular manifestations are occasionally met with, and the same holds good in Rheumatoid Arthritis; in other words, structures other than those of the joints, but of a similar nature, may take on the same kind of diseased action. Sometimes these symptoms occur simultaneously with the joint affection; sometimes they alternate with it, or appear to be altogether independent of it. In well-marked cases of this irregular form of the disease, the inflammation has attacked the eyes, ears, or structures of the larynx; producing in the first organ sclerotitis, in the second inflammation of the internal ear, and in the last hoarseness, and a peculiar dry cough, not attributable to pulmonary disease.

Analysis of the Animal Fluids in Rheumatoid Arthritis.—Very little information has been obtained from an examination of the blood, urine, or sweat of patients, suffering from Rheumatoid Arthritis, which is calculated to throw light upon the nature of the disease.

The only analyses of the blood that the writer is cognizant of have been made by himself, and with simply a negative result. If any amount of active inflammation is present, the fibrin is increased, and the clot becomes firm, cupped, and buffed.

The serum has the ordinary properties of the serum of healthy blood, and yields no uric acid.

The analysis of the urine has likewise given negative results.

The perspiration has not been examined, but there is no reason to suppose that any peculiar alteration would be found in it.

Morbid Anatomy of Rheumatoid Arthritis.—The morbid anatomy of this disease has been very elaborately worked out by Dr. Adams, to whose volume the reader is referred for full details of the various changes which take place. In the present article a summary only of the results found in different cases, and different stages of the malady, will be given.

If a joint is examined in an early stage of the disease, when swelling is prominent, a considerable increase of synovial fluid is found, and the joint exhibits the same appearances as in cases of ordinary inflammation; the lining membrane is often red, from over-injection of the blood-vessels. It is not an easy matter to obtain an opportunity of examining joints in this condition, as Rheumatoid Arthritis is seldom fatal, except in its very advanced stages; but the supervention of other maladies sometimes enables us to do so. No deposits of urate of soda are found in any stage of this disease, and I am persuaded that the statements to the contrary are erroneous, and, as yet, there has been certainly no proof given in support of such assertions. Of course it is not impossible that a patient may have had gout

[Fig. 33.

Arthritis of Knee-joint.

Fig. 34.

Changes in the Head and Neck of the Femur and in the Acetabulum in Arthritis.]

in a joint, and that afterwards the same articulation may become the seat of Rheumatoid Arthritis; but this, if indeed it ever occurs, is most rare, and would not in any way favor the idea that deposition of urate of soda is a phenomenon of this form of inflammation.

When the effusion in a joint has been absorbed, the capsular membrane is usually found thickened. In the hip, or shoulder, the round ligament or tendon of the biceps is probably destroyed; inter-articular cartilages are sometimes absorbed, and a case has recently come under my observation in which the articulation of the jaw exhibited this alteration in a very complete degree. If the fluid becomes absorbed before much serious change has taken place in the internal structures of the joint, the ligaments have generally undergone so much lengthening as to allow of unnatural mobility, thus rendering dislocation easy.

From almost the very commencement of the inflammatory action, the articular cartilage begins to suffer, a slow process of absorption takes place, the cartilage appears to split up into fibres, vertical to the surface of the bone; little depressions are observed, and these at length coalesce, and the bone is left in part uncovered; as the disease proceeds, the whole surface may be thus denuded, and as the osseous surfaces are brought into contact with each other in the movements of the articulations, they become polished in a remarkable degree by the friction, and an ivory-like condition, termed eburnation, is produced. Sometimes this eburnation occurs in streaks or patches in the direc-

tion of the motion of the joint; sometimes the whole surface may become thus altered. More rarely the cancellated structure of the heads of the bones is exposed by the absorption of the denser matter at their extremities, and the ends of the bones become enlarged and misshapen by the deposition of osseous matter. If the bone is sawn through, it is often found unusually spongy, and contains a large amount of oily matter, from the occurrence of a species of fatty degeneration.

Within the joints vegetations and bands are frequently seen, also foreign bodies of various sizes, some cartilaginous in structure, others having the consistence and texture of bone; and these are usually adherent to the internal surface by ligamentous bands.

Causes of Rheumatoid Arthritis.—The predisposing causes of Rheumatoid Arthritis may be thus classified: First, those which are inherent in the patient; secondly, those which arise independently of the individual.

1. *Influences dependent on the Individual.*—*Hereditary Disposition* does not appear to exert any very special influence, except that children of weakly parents probably inherit their debility. In looking over the histories of a large number of cases, the writer cannot find much evidence of the direct influence of hereditary predisposition; if it exists, it is very much less powerful than in the case of gout. It is not uncommon to find one member of a large family suffering severely from the disease, and the rest entirely free from it.

Sex.—It is commonly thought that women are more liable to Rheumatoid Arthritis than men. M. Trousseau speaks of the affection as very rare among men, very frequent amongst women. Women are doubtless very prone to be attacked, as they are most likely to be subjected to the predisposing causes, especially irregularities of the uterine function; and it would appear that deranged menstruation, independent of hemorrhage, predisposes to the disease. Men, however, are by no means free, and some of the most severe cases are found among them.

Age.—Rheumatoid Arthritis may occur at almost any age. I have seen it in its worst form in children of ten and twelve years of age, and I have also seen it commence in very old people above seventy years.

Individuals of weak frame, whose circulation is languid, and whose extremities are habitually cold, are more liable to the disease than others; and it should be mentioned that patients having a tubercular diathesis are often the subjects of Rheumatoid Arthritis.

2. *Influences independent of the Individual.*—Everything which causes debility and loss of tone in the extreme circulation, as hemorrhages from the uterus or elsewhere, deep and prolonged grief, severe and protracted mental anxiety, acts as a predisposing cause of the disease. It not infrequently results from rapid child-bearing, or too lengthened lactation; also from night-watching. Cold is very frequently an exciting cause of the disease, especially if it has been prolonged, and has caused severe depression of the functions of the nervous system. In one instance I have seen the affection in its most severe form apparently the result of diabetes mellitus.

In some cases injuries or shocks appear to have acted as exciting causes.

Now and then acute rheumatism acts as an exciting cause of Rheumatoid Arthritis.

Malt liquors and wines do not appear to exercise any influence in either causing or protracting Rheumatoid Arthritis.

Pathology of Rheumatoid Arthritis.—The examination of the blood in Rheumatoid Arthritis has failed to discover any constant or pathognomonic changes in that fluid; but as yet the number of analyses has been very limited. One fact of importance they have elicited, namely, the absence of uric acid; and thus we are able to distinguish the blood in this disease from that in true gout.

Pathological anatomy has likewise enabled us to distinguish Rheumatoid Arthritis from gout, on the one hand, and from rheumatism on the other, by demonstrating the absence, in the affected joints, of deposits of urate of soda, which are constant in gout, and by showing the presence of ulceration of the cartilages, and of other structural alterations, which are not found in simple rheumatism, even after repeated attacks.

The consideration of the history and progress of the disease has shown that it differs completely, in its essential nature, from both gout and rheumatism, and fully justifies the rejection of the name "rheumatic gout," which must of necessity convey to every mind the idea of a hybrid disease, a compound of both gout and rheumatism. If a disease should be so designated, because it differs completely in its intimate pathology from both affections included in its compound name, then no fault can be found with the nomenclature.

It seems difficult to persuade those who have been brought up in the old idea of this hybrid affection that such disease has no real existence.

Dr. Aitken, in his excellent work on the Science and Practice of Medicine, although he admits that Hunter warmly opposed this compound appellation, "rheumatic gout," deems it is neverthe-

less pathologically correct, and thinks that a hybrid disease, depending on the combined cachexia of gout and rheumatism, has a real existence, as recognized by Craigie, Wood, Spencer Wells, and Fuller. On turning, however, to the last-named author, the following passage is met with: "The disease should not be regarded as of a hybrid character, or, in other words, made up in part of rheumatism, in part of gout." And again: "It has no connection with either of these diseases, beyond that which attaches to it in virtue of its being a constitutional disorder, producing local manifestations in the joints." Might not the very same be said of pyæmia? Is this not a constitutional disease, producing local symptoms in the joints?

It is a much easier task to prove what Rheumatoid Arthritis is not, than to give the slightest clue to what it is: at present I should hesitate to offer a strong opinion as to its nature. It appears to result from a peculiar form of mal-nutrition of the joint textures, an inflammatory action with defective power; but of its dependence upon the presence of any morbid principle, or upon a weakened condition of the vessels or structures of the affected parts, no evidence exists upon which any reliance can be placed. Thus much only appears to be made out: it usually occurs in weakened subjects, and exposure to cold is in many cases the exciting cause of its development.

A full and searching investigation into the nature of Rheumatoid Arthritis is still a desideratum.

Diagnosis of Rheumatoid Arthritis.—Perhaps there is scarcely a subject of greater importance in the whole range of joint affections, than the diagnosis of Rheumatoid Arthritis; for upon a correct understanding of it depends the future comfort and physical well-being of a large class of persons.

Rheumatoid Arthritis, as has been already shown, assumes various forms, and individual cases of the disease are often with difficulty distinguished. The affections with which it is apt to be confounded are gout and rheumatism. It is therefore important to be able at once to distinguish Rheumatoid Arthritis from the above-named diseases. Haygarth thus describes the difference between what he termed nodosity of the joints (Rheumatoid Arthritis) and gout, &c.:—

"The nodes appear most nearly to resemble gout: both of them are attended with pain and swelling of the joints, but they differ essentially in many distinguishable circumstances. In gout the skin and other integuments are generally inflamed, with pain, which is very acute, soreness to the touch, redness and swelling of the soft parts, but in no respects like the hardness of bone. The gout attacks the patient in paroxysms of a few days, or weeks, or months, and has complete intermissions, at first for years, but afterwards for shorter periods. The gout attacks men much more frequently than women. There is one distressful circumstance which distinguishes this disorder: it has no intermission, and but slight remissions, for during the remainder of the patient's life the nodes gradually enlarge, impeding more and more the motion of the limb; the malady spreads to other joints, without leaving or producing any alleviation in those which had been previously attacked."

The following considerations will enable us to effect the diagnosis in at least the majority of cases:—

It is questionable whether Rheumatoid Arthritis is in any marked degree capable of being inherited; whereas gout is distinctly hereditary.

The sex of the patient does not aid us much. Both sexes are liable to the disease, and the difference in their liability is too slight to enable us to give it much weight in diagnosis. Nor does the age of the patient argue much, as it has been shown that Rheumatoid Arthritis attacks both children and very aged persons.

In many instances Rheumatoid Arthritis is preceded by a condition of ill-health; there are evidences of bad nutrition and exhaustion of the nervous system.

Rheumatoid Arthritis usually begins as a sub-acute disease, and the joint affection gradually increases; but occasionally it commences in an acute form. These latter cases may be mistaken for acute gout or rheumatic fever. There are, however, peculiarities in its course by which it can generally be distinguished from either of these diseases; the most marked being its progressive character, which has been fully described above.

From acute gout it may be distinguished by the length of the paroxysm, the absence of periodicity, by the large and small joints being equally attacked at the outset, and the great toe not being specially involved.

From rheumatic fever or acute rheumatism, by the comparative freedom from constitutional disturbance, the longer duration of the paroxysm, and the absence of acute cardiac inflammation.

From chronic rhumatism it can be distinguished by the comparative absence of structural alteration in the former disease.

The most frequent difficulty which occurs is to separate chronic Rheumatoid Arthritis from chronic gout, and, on referring to the article "Gout" in the present volume, a case illustrative of this difficulty will be seen. There should always be a careful search made for evidences of urate deposits on the ear and

at the tips of the fingers, and in the bursæ over the olecranon process of the elbows; but from mere inspection it is difficult to make a diagnosis, especially if the affection has become chronic.

When the diagnosis is very obscure, notwithstanding that the above rules have been followed, it may be at once cleared up by an examination of the blood; in Rheumatoid Arthritis there is no uric acid present: there is also, in most cases, the absence of any material alteration in the urine.

One disease is with difficulty separated from the acute forms of Rheumatoid Arthritis—namely, the joint affection arising from urethral suppuration: the history of the case will of course enable us to clear up the diagnosis.

Prognosis of Rheumatoid Arthritis.—Rheumatoid Arthritis is unquestionably a very intractable disease; and this is not to be wondered at, if we consider its ordinary antecedents. Its most common predisposing cause is a thoroughly impaired condition of the system, and this in many instances has arisen from influences which have been for years in operation: it cannot, therefore, be even reasonably hoped that an affection occurring under such circumstances can be rapidly cured: it is often a great achievement to arrest its further progress.

If the disease is far advanced, and the joints severely injured, it is impossible to restore the articulations to their former state, even if the constitutional tendency to the disease is thoroughly arrested. Still, under such circumstances, it sometimes happens that patients, who for years have been unable to move, will regain their power of locomotion, a result caused by the formation of a kind of false joint in some of the more important articulations. When the disease is less advanced, when the affected joints are few in number, and their mobility but partially interfered with; when at the same time there is freedom from any disease which must of necessity keep up the impaired state of health; lastly, when the progress of the affection has not been rapid—then a more favorable view may be taken of the case, and a recovery more or less complete may be hoped for.

When the disease is at its commencement, or at least when but little damage has ensued, and at the same time the affection shows little tendency to assume an acute character, then a complete recovery may be anticipated, if proper measures be adopted: if, however, the patient is in any way weakened, or treated as if he were suffering from a sthenic malady, then the probability of the disease becoming deeply engrafted into the system, and causing serious mischief, is greatly increased.

The accurate diagnosis and proper treatment of cases of Rheumatoid Arthritis exercise a very important influence upon its prognosis.

Treatment of Rheumatoid Arthritis. — The treatment of Rheumatoid Arthritis is a subject of the highest importance, more especially in the earlier stages of the disease, when as yet the joints are not seriously or irremediably injured.

From what has been stated under the "Causes" and "Pathology" of Rheumatoid Arthritis, it will be naturally inferred that a prolonged sustaining plan of treatment is imperatively called for, and that all depleting measures must tend most materially to increase the rapidity and severity of the disease. I have witnessed a great number of cases in which depletion has been persevered in for a time, and with the effect of producing lamentable results, as the joints became perfectly disorganized; and in many of these it is probable an opposite treatment would have been attended with different results.

We must remember that, during the more acute inflammatory stages; the disease must not be treated in the manner recommended to be adopted in gout. Colchicum is always worse than useless, and the patient should be well sustained throughout the whole of the treatment; neither are the alkalies, so serviceable in rheumatism, of any permanent value in Rheumatoid Arthritis.

With the exception of stating that a supporting plan must be adopted from the first, no rules applicable to all cases can be laid down, a fact which must be evident, if we consider the different causes which lead to a condition of habit favorable to the development of the disease. If the disease has been caused by hemorrhage of any kind, and anæmia exists, then the first object must be to restore the blood to its normal state, and preparations of iron are imperatively called for. If the mere hæmatinic influence is required, the reduced iron (ferrum reductum) may be administered, or the ammonio-citrate or some other very mild salt of this metal; if, however, the anæmia is combined with a relaxed state of habit, the more astringent preparations should be employed, as the sulphate or perchloride of iron. If anæmia exists from other causes than hemorrhage, similar remedies may be had recourse to.

Ferruginous salts are also of much value in keeping up the power of the heart, but in all cases they should be given in small doses, and persevered in for a long time.

In certain conditions where the nutrition is imperfect from causes often unable to be defined, cod-liver oil may be given

with great advantage: if the habit is materially improved by it, the progress of the joint affection is usually checked. Cod-liver oil is particularly indicated in patients of spare habit, and when the disease has been attended with wasting of the body. If the nervous system has been seriously implicated by depressing causes, as anxiety, grief, prolonged attendance on sickness, then remedies more directly affecting this system must be employed, either by themselves, or in conjunction with those already mentioned. Quinine, as a nervine tonic, is often of much service, as likewise, when astringents are indicated, the preparations of cinchona bark; assafœtida, valerian, and ammonia are of value when there is much mental depression.

If the circulation is very languid, guaiacum may be administered with benefit, or guaiacum united with yellow bark; as, for example, the ammoniated tincture of the former medicine, and the simple tincture of the latter.

Guaiacum appears to exert a marked effect upon the capillary circulation, increasing the warmth of the extremities, and rendering the functions of the skin more active.

Arsenical preparations are in some cases of considerable value: how they act is a subject of considerable difficulty to explain. We have clinical evidence demonstrating their peculiar action upon the skin, and it may be that their influence is exerted also upon the fibrous and cartilaginous structures, or they may more especially affect the nervous system as tonics, and thus prove of service. Arsenic may be given either in the form of the arsenite of potash (liquor arsenicalis), or the arseniate of soda (liquor sodæ arseniatis). From repeated trials, I have come to the conclusion that arsenic, in the state of arsenic acid, is less irritating than when in the lower condition of oxidation, and can be given in larger doses.

Iodides are of service in some cases, especially during the more acute stages, or when warmth has a marked effect in causing an augmentation of the pain. Iodide of potassium may be administered alone, or in combination with guaiacum or guaiacum and bark; or the iodide of iron may be given, especially if ferruginous preparations are indicated. The syrup of iodide of iron, in doses of from twenty to thirty drops twice a day, and continued for some months, has in several cases proved of much benefit, even to the extent of completely arresting the progress of the disease.

In special cases special remedies must be employed. If, for example, menorrhagia has been in any way the cause of the debility, and still continues, bromide of potassium may be advantageously had recourse to; and should the muscular system have been wasted, especially the muscles of the affected limbs, nux vomica or strychnine can be given to restore their function.

Mineral waters are often resorted to, from a mistaken view of the nature of the malady. I have seen much injury resulting from their employment from the debility they have induced. The springs most adapted for the subjects of Rheumatoid Arthritis are those of Schwalbach, Pyrmont, and Spa, or any other ferruginous waters, which sit easily upon the stomach. The stronger saline and alkaline waters, as those of Carlsbad, Wiesbaden, and Vichy, unless used with great care, are apt to aggravate the disease. In some very chronic cases, the springs of Wildbad and Gastein have proved of much service.

Change of air, occupation, and scenery, by aiding the general health, exerts a beneficial influence upon the progress of the malady.

Local Treatment of Rheumatoid Arthritis.—In the early stages, when there is tenderness and swelling of any joint, relief is often experienced from the application of blisters, and a most convenient form is the cantharides liniment of the British Pharmacopœia: it produces, in almost all cases, full vesication, and with little annoyance; it can be frequently applied, so as to produce a series of flying blisters. Under this treatment the effusion will often quickly subside, and the tenderness become much lessened or even removed, and the liability to serious or permanent injury of the joint is thereby greatly lessened. Other forms of counter-irritation, as iodine paint, croton oil, may be used, but I believe the blister treatment above mentioned is productive of more benefit.

When the affection of any joint has become more chronic, and blisters have effected all they are able to accomplish, further benefit may be obtained from the use of plasters, which act not only by the slight counter-irritation they produce, but likewise, if properly applied, from the support they afford to the joint. The kind of plaster selected should depend on the requirements of the case. If little more than simple support be needed, soap plaster may be used, and the joint may be bandaged with narrow strips of this, spread by machine on linen. If more irritating applications are desired, then the galbanum or ammoniacal plaster can be used, or the ammoniacal and mercurial, or the iodine plaster: when using the two latter, severe irritation of the skin should be avoided.

The application of lotions containing spirit of such a strength as to cause but slight irritation of the skin sometimes

gives great relief. If there is much pain, the belladonna liniment may be used in lieu of the simple spirit.

Baths are in some cases useful, especially when the function of the skin is defective; but care must always be taken that debility be not induced, otherwise any good result is more than counteracted.

Bathing is certainly not curative in this disease, it can only be looked upon as palliative, and should be employed with this understanding.

In chronic cases, when friction can be used without increasing the tenderness of the parts, it may be employed either alone, or combined with some stimulating liniment; or the joint may be well sponged with strong brine, and then rubbed dry, so as to cause the salt to enter the skin.

It is often asked whether it is desirable to allow movement of the affected joints? I believe the best answer to be this: never allow such an amount of movement as will cause the joint to be more painful on the following day; but any motion short of this may be employed with advantage. If this rule were followed, it would prevent the use of joints recently or acutely attacked, and rest in such cases aids the subsidence of the inflammation; on the other hand, it would allow those articulations in which the disease has become chronic to be moved to such an extent as to stay the wasting of the muscles of the limb, and to prevent the stiffening of the joints.

[Many practitioners have confidence in the utility of mercurial ointment, applied once or twice a week to the inflamed joints. More benefit is likely to accrue from the employment of an ointment of carbonate of lead; two drachms to an ounce of simple cerate.[1]—H.]

Diet and Regimen in Rheumatoid Arthritis.—As far as the fluid portion of the diet is concerned, it is a matter of indifference whether malt liquors, wines, or distilled spirits are taken, provided that they agree with the stomach: it is far different in true gout.

Whichever kind of alcoholic beverage causes the patient to eat with most relish, and digest with most comfort, should be selected. There is no necessity to give enough to stimulate, but only a sufficient amount to sustain the vigor of the assimilating functions. I am convinced that wines and malt liquors have no influence in favoring the development of Rheumatoid Arthritis, although they powerfully predispose to the production of gout. The solid portion of the diet should be of the most nutritious character and of easy digestion, for our main object is to keep up the strength of the system: nor have we any proof that an excess of animal food tends to increase the disease, as is the case in gout. Meat should form a good proportion of the diet, if the stomach is capable of digesting it; and whatever articles of diet are found to improve the general health may be given with advantage.

A frequent change of air and scenery should be advocated, all prolonged mental exertion avoided, and, as far as possible, all causes of anxiety should be removed.

A residence in a moderately warm country during the winter months is desirable, but the air of the place should be dry and bracing. Such a climate will enable the patient to take exercise and have fresh air when otherwise he would be confined to the house.

If the joints allow of it, moderate exercise should be enjoined, but not sufficient to cause subsequent fatigue.

The clothing should be warm, but much perspiration avoided.

[1] This vehicle is preferable to lard on account of the softening effect of the carbonate. Vaseline is liable to the same objection, in this particular mode of application.—H.]

RHEUMATISM.

By Alfred Baring Garrod, M.D., F.R.S.

The subject of Rheumatism will be discussed in the present article under the heads of Articular and Muscular Rheumatism; the first, or Articular Rheumatism, in its more acute form, being commonly known as Rheumatic Fever. Such a division has been adopted, as it is a question at the present day whether the articular and muscular affections are in reality manifestations of the same disease.

A. Articular Rheumatism.

Definition. — (1) Acute Articular Rheumatism. A specific inflammation of the structures in and around the joints, attended with great febrile disturbance; erratic; not accompanied with deposits of urate of soda, and (?) not leading to suppuration. (2) Sub-acute Articular Rheumatism. The same affection as the above, but manifested in a much less intense degree, and with little febrile disturbance; generally following upon the acute disease.

Synonyms.—Rheumatic Fever; Arthritis.

History. — Although there is every probability that mankind was afflicted with Rheumatism from the very earliest periods, still we gather from the writings of ancient physicians that they did not separate this malady from other forms of joint disease, but comprehended all of them under the general term "arthritis," the only distinction being made dependent on the particular articulations implicated.

Monsieur Baillon, a French physician, first made use of the word Rheumatism (from ῥεῦμα, a stream, a fluxion): in a Latin treatise published in 1642, entitled "De Rheumatismo et Pleuritide Dorsali," he separates this disease from gout in the following sentence: "Gout is a disease of a certain part, and periodical; Rheumatism of the whole body, and more uncertain in its attack."

Sydenham afterwards makes a distinction between the two affections: "This disease, when unattended with fever, is frequently mistaken for gout, although it differs essentially therefrom, as will easily appear to those who are thoroughly acquainted with both diseases; and hence it is, perhaps, that physical authors have not mentioned it, unless indeed we esteem it a new disease." Cullen thus defines Rheumatism: "A disease from an external and often an evident cause; pyrexia; pain about the joints, following the course of the muscles, fixing upon the knees and larger joints in preference to those of the feet and hands—increased by external heat."

Even since Cullen's time there have been authors, both in this country and abroad, who have included Rheumatism and gout under the same category, amongst whom stands prominently the name of M. Chomel.

The late Sir Benjamin Brodie, in his work on diseases of the joints, clearly separates Rheumatism from rheumatoid arthritis, although he designates the latter affection ulceration of the cartilages.

At the present time it may be confidently asserted that, omitting purulent affections of the joints and those connected with urethral suppuration, there exist at least three well-marked articular diseases dependent on morbid states of the system; these are Rheumatism, gout, and rheumatoid arthritis: and probably to them we may add a fourth, namely, muscular rheumatism.

Description of Acute and Subacute Rheumatism.—To illustrate this subject we will give a sketch of an acute attack of rheumatism, such as is daily met with in practice. A young woman, possibly somewhat out of health, is exposed to severe cold, or to cold and damp conjoined; she feels a distinct chill or rigor, and this is followed, probably during the second or third day from the exposure, by the development of the joint affection; the ankles become painful and unable to bear the weight of the body, and on examination they are found tender to pressure, swollen, unduly hot, with a distinct flush upon the surface; at the same time the system exhibits a state of febrile excitement, the pulse is rapid and commonly hard, the whole surface hot and bathed in perspiration, having a peculiar acid, at least acrid, odor. The tongue is coated with a thick creamy fur; there is loss of appetite, but increased thirst and a constipated state of the bowels; the urine is usually scanty and high-colored, and gives rise on cooling to a copious red deposit. The inflammation is seldom confined to one joint, but gradually extends over the whole body. The larger joints are more frequently affected than the smaller in the earlier periods of the attack.

The erratic nature of the affection is usually well exhibited; at one time the knees and ankles, at another the elbows and wrists suffer; and not infrequently the development of inflammation in one set of joints is accompanied by its rapid subsidence in another.

A symmetry is often shown in the order of attack: the right ankle, then the left; the right knee, then the left; and so on for the other articulations. It is a matter of astonishment to observe how quickly and completely the inflammation will subside in any part. A knee, for example, will one day be intensely hot and swollen, and so exquisitely tender as not to bear the weight of the bed-clothes; but on the following day will scarcely show any evidence of its previous suffering. This condition of the patient may continue for many days or perhaps weeks, the duration depending partly on the intensity of the disease, partly on the habit of the patient, and partly on the treatment adopted. Its usual duration, when under no special treatment, is from ten days to three or four weeks; but perhaps no disease exhibits greater differences in this respect, and hence the difficulty, except when a number of cases are compared, of

accurately estimating the value of the various plans of treatment which have been employed for its cure. Cases are now and then met with which spontaneously terminate in five or six days, others which run a course of six or even eight weeks. With few exceptions, the pain of the joints and the febrile disturbance are greater at night than in the day; but the nocturnal exacerbations are less marked in rheumatic fever than in acute gout.

After a varying time the joints become free from redness, swelling, and tenderness, the pulse lowered to its healthy standard, the temperature and sweating of the surface diminished; at the same time the tongue cleans, the thirst abates, the appetite returns, the urine clears, and in short the system is restored to its healthy state, with the exception of a certain amount of wasting and debility, necessarily induced by the increased wear of tissue and the defective nourishment which have taken place during the period of febrile disturbance. From these, however, the patient soon recovers, when able to take a generous diet and enjoy fresh air and exercise.

It is not unusual to find that patients after recovering from attacks of rheumatic fever, as far as regards the constitutional symptoms, are liable to suffer from pains in those joints which have been the seat of inflammation: these pains are not constant, but assume a neuralgic character, and will generally be found to be in proportion to the length of time the joints were affected during the febrile attack.

Thus far our description has been confined to a case of acute rheumatism, in which the inflammation has been limited to the joint structures: unfortunately, however, it is not always so, for in a large percentage of cases the covering or lining membrane of the heart, or even the substance of the organ, becomes implicated, and peri- and endo-carditis ensue. Sometimes the heart disease follows a somewhat sudden subsidence of the joint inflammation, but more commonly the articular and cardiac affections run a simultaneous course.

When inflammation of the pericardium takes place, the patient usually experiences a sensation of tightness or pain in the chest, but not always, as the acute discomfort caused by the condition of the joints draws the attention altogether from the slight uneasiness due to the cardiac complication. On applying the stethoscope a distinct friction or rubbing sound is heard, often limited at first to a small extent of surface, generally at the base of the heart, but it commonly spreads over nearly the whole of the surface of the organ; when the amount of effused fluid is large, there is increased dulness of the percussion note over the cardiac region, and the heart's sounds are diminished in intensity, and heard as if from a distance. When, on the other hand, the endocardium is implicated, abnormal heart sounds are produced usually at the aortic or mitral valves, giving rise to basic or apex systolic murmurs; or, if the closure of the aortic valve becomes imperfect, a basic diastolic murmur is also developed. These various phenomena are due to the pouring out of lymph and serum upon the surfaces of the pericardium, or the secretion of plastic lymph or fibrinous coagula upon the valves of the heart. When the cardiac substance is involved, great irregularity and extreme feebleness of the action of the heart ensue, often leading to sudden and fatal collapse. For further details concerning the physical signs and symptoms which occur in pericarditis and endocarditis the reader is referred to the articles devoted to those diseases, as these affections, when they arise from rheumatic inflammation, follow the same course and exhibit the same phenomena as when due to other causes.

Pleurisy of a true rheumatic nature sometimes occurs during the progress of rheumatic fever, and the phenomena are the same as in the ordinary forms of acute pleuritis.

More rarely the peritoneum becomes affected by rheumatic inflammation.

When the heart is implicated, delirium is usually present, especially at night; but in some few cases the rheumatic condition appears to attack the membranes of the brain, and then the symptoms of cerebral meningitis are produced. Sometimes the membranes of the spine are involved, and symptoms of spinal meningitis are set up.

Chorea is apt to supervene in young subjects after the cessation of the febrile disturbance in rheumatic fever, where the heart has been implicated.

Description of Subacute Articular Rheumatism.—At times, from various causes—as the nature of the patient's constitution or the presence of cardiac complication—articular rheumatism assumes a form to which the name subacute may be properly applied; the joint symptoms remain, but in a much less severe degree, and there is an almost complete absence of febrile disturbance. Such a condition may continue for weeks or even months, at one time relieved, at another aggravated, and the disease may then be compared to a similar subacute form not infrequently seen in gout, and to which the term chronic is applied.

In subacute rheumatism there is often some tenderness of the joints, slight swelling, and heat, but the disease differs from both chronic gout and rheumatoid arthritis, inasmuch as it may continue for a

long time without leading to any great deformity or permanent injury to the articulations.

It will be seen that the statements made above are at variance with those commonly met with in works on this disease, in which the results ascribed to the subacute affection are so formidable. The difference is easily explained: most authors attribute to chronic rheumatism the changes that in the present work are described under the head of rheumatoid arthritis.

The occurrence of one attack of rheumatic fever imparts a great susceptibility to the system for its return, which a second augments, and thus after a time the patient is liable to become the victim of frequent seizures, though these will probably assume a less sthenic form, and are often of such a character as to entitle them to be called subacute rather than acute. When a patient has been brought to this condition, the most trifling exposure to cold, or even the slightest depressing cause, may prove sufficient to light up the disorder.

Even in individuals who have suffered long and severely from repeated attacks, it is unusual to find any very serious alterations produced in the affected joints; they may for a time remain more or less swollen from effusion of fluid, the ligaments may become stretched and relaxed, but there is no permanent thickening left either from the deposits of the chalk-like matter (urate of soda) so constantly found in gout, or from the formation of false cartilages and other changes, the effect of rheumatoid arthritis.

Consideration of the different Phenomena in Acute Rheumatism.—The various symptoms which are observed in cases of acute rheumatism demand further investigation than could be given them in the above sketch, and the first which claim attention refer more especially to the joints themselves. The pain is generally very severe, but less intense than in gout; the swelling also is usually less marked than in the latter disease; there is rarely found extreme tension of the skin, and seldom œdema or desquamation of the cuticle; the redness is likewise less in Rheumatism than in gout, although this symptom may sometimes be present in a marked degree; and, lastly, the enlargement of the veins leading from the inflamed joint is certainly much less prominent in this disease than in true gout.

Rheumatic inflammation has a decidedly erratic disposition, and, as it were, flies from joint to joint, or at least one articulation suddenly ceases to exhibit inflammatory symptoms, and another, probably the corresponding articulation, becomes as suddenly implicated; and this alternation may occur many times during a single attack. A certain amount of symmetrical action is likewise observed, as in almost all diseases the symptoms of which depend on an altered condition of the blood. On reflection it will be found that our surprise should rather be excited by a want of symmetry than by its occurrence; for supposing that any morbid element has a peculiar attraction to any one joint—a knee, for example—it is necessarily drawn to the second knee more powerfully than to any other joint; or supposing again that the circumstances in one particular joint more especially favor the development of any specific form of inflammation in it, the conditions of the corresponding joint on the other side of the body must do so likewise.

It has been stated that the swelling of the inflamed joints is less in Rheumatism than in gout, and the same holds good with the pitting on pressure, and the subsequent desquamation of the cuticle. This last symptom, so constant in gout, is not observed after rheumatic inflammation: but it must not be concluded that because pitting is present the inflammation is not rheumatic; this would be erroneous, for cases of genuine rheumatic fever are now and then met with in which pitting of the skin is well marked: when this phenomenon occurs, it is generally in weak subjects. In several such instances I have proved, not only by carefully examining all the symptoms, but also by analyzing the blood, that the cases were of a true rheumatic character, and had no relation to gout.

The constitutional symptoms which are most prominent in acute articular rheumatism next require consideration. The first of these, one of great importance, is the temperature of the body. That there is a well-marked heat of surface is evident to the touch, but the indications of the thermometer are far more trustworthy; for they show correctly the amount of tissue-waste going on in the system, and cannot be masked by the influence of the mind or other circumstances which render some of the subjective symptoms of comparatively little value.

Dr. Sidney Ringer, whose researches on temperature in disease are of much value and interest, has made observations on three cases of acute rheumatism which were communicated to Dr. Aitken, and published in his work on the Science and Practice of Medicine. In some fatal cases, complicated with severe pericarditis, the temperature rose as high as 106° Fahr., 109° Fahr., and 110° Fahr. before death.

In some instances of acute articular rheumatism I noticed the temperature, and found it to vary from 100° Fahr. to 104° Fahr.; but these observations have been made in cases in which recovery has

taken place. The pulse, as already noticed, is usually quiet in acute rheumatism; it is also hard and full; but exceptional cases are now and then met with in which the frequency is never great, although the febrile disturbance and temperature run high. More reliance can be placed upon the heat of the body than upon the rapidity of the heart's action in all cases of acute inflammation, and this especially holds good in Rheumatism, as the cardiac complications so frequently present in this disease alter the character of the pulse, independently of the amount of tissue-change which is going on in the system.

The perspiration is generally considered to be intensely acid in acute rheumatism; in several cases I have found it less acid than in healthy subjects; but it must be remembered that the amount of perspiration is excessive. The peculiarity of the skin secretion in this disease depends on its acridity perhaps more than on excessive acidity.

Condition of the Blood in Acute Articular Rheumatism.—In acute rheumatism, if blood is drawn from a vein, the clot is found to exhibit a buffy coat, and is frequently cupped—appearances which indicate that the fibrin is increased in quantity, and that this principle is also probably somewhat altered in quality.

Many discrepant statements have been made upon this subject; according to Haller, the blood yields a thick and firm clot in this disease, but others assert that under the buffy coat the clot is found to be loose and friable;—probably both are correct in different instances.

Andral and Gavarret analyzed the blood in fourteen cases of rheumatic fever, and found the maximum amount of fibrin to be 10·2 parts in the 1000; the minimum 2·8 parts; the mean was 6·7: healthy blood yields, according to these observers, three parts per thousand.

The serum of the blood does not appear to be very sensibly altered in composition; Andral and Gavarret found an increase in the solid residue left by evaporating this fluid, the mean amount being 86·0 per thousand against 80·0 per thousand in healthy blood.

The same chemists found a diminution in the total solids: thus the mean in the above-mentioned fourteen cases was 194·6 parts of solid matter to 805·4 parts of water in the 1000 parts of blood; whereas in healthy blood the ratio between the solid residue and water is 200·0 to 790·0.

In several analyses which I have made of the blood in acute rheumatism, I have found the amount of fibrin notably increased—namely, from four to six parts in the 1000—and the clot has usually been buffed and cupped. The serum in thirty-five cases was found distinctly alkaline in reaction, it presented a healthy appearance, its specific gravity was somewhat less than in health, and in no case was any uric acid detected either by the thread experiment described in the article on Gout, or by the ordinary method of separating this acid from blood. The absence of uric acid or urate of soda is important, as it at once shows essential difference between gout and Rheumatism.

Urea does not exist in the blood in acute rheumatism in quantities larger than in health, except in cases in which the kidneys have been either previously affected, or have become congested during the progress of the disease. As above stated, the serum was always distinctly alkaline in reaction: this fact was particularly observed and noted, as it has been affirmed that the serum is sometimes acid in rheumatic fever. There are, as yet, no recorded observations indicating the amount of the alkalinity. No abnormal principle has been found in the blood; lactic acid has been assumed to exist in it, but no proof has been given of its presence.

In the subacute and chronic varieties of Rheumatism no marked alterations are found; the fibrin is increased but slightly, and only in proportion to the amount of the febrile disturbance. In ten cases the maximum of the fibrin was 5·1, the minimum 2·6, and the mean 3·8 parts in the 1000 parts of blood.

Urine in Acute Articular Rheumatism.—Examinations of the urine in acute rheumatism have been frequently made with the following results: To the eye its appearance varies much in different cases; as a rule it is high-colored and scanty, clear when first passed, but speedily becomes turbid from the deposition of urates; when copious it sometimes remains bright even after it is cold. These characters are by no means peculiar to rheumatic fever, but are seen in many febrile affections. The deposited urates are often of a deep tint from the coloring matters of the urine being in excess and attracted to the uric acid salts, and if the fever runs very high, and especially if portal congestion is present, the color becomes bright red or deep pink. On a more minute examination, it is found that the secretion of water by the kidneys is usually diminished; the solids of the urine are increased. The increase in the solid matter is due chiefly to the augmentation of the urea, partly also to an increase of the coloring matter. The uric acid is augmented: Dr. Parkes has found as much as seventeen grains in one case; the largest amount I have obtained has been fifteen grains. It should be remembered that this increase of uric acid in the urine is not peculiar to rheumatic fever; it occurs likewise in other febrile diseases, unless the excreting power of the kidneys is injured. The

chlorides are diminished during the febrile disturbance, but less so than in pneumonia. Dr. Parkes found the sulphates much increased in several cases in which the inflammation of the joints and the febrile disturbance were severe. When alkaline remedies are not administered, the acidity of the urine is usually great. Albumen is now and then met with during the course of this disease, sometimes only for a day or two, but its occurrence is much less frequent in rheumatic fever than in pneumonia. No proof has yet been given that lactic acid exists in the urine in rheumatic fever in greater amount than in healthy urine.

Cardiac and other Inflammatory affections in Acute Rheumatism.—It has been stated that in a large percentage of cases of rheumatic fever the structure of the heart becomes involved, and as such complications are of vital importance to the patient, they deserve in this place further consideration.

It has long been suspected that some relation existed between acute articular rheumatism and inflammatory diseases of the heart. In the first volume of the Medico-Chirurgical Transactions there is a paper by Sir D. Dundas on this subject, in which he states: "In all cases which I have seen, this disease has succeeded one or more attacks of rheumatic fever. In one case, the affection of the heart appeared at the commencement of the rheumatic fever, and its action was so rapid that the pulse could not be counted for many days; much difficulty of breathing and oppression, attended with a sense of debility, took place; and the inflammation, pain, and swelling of the extremities, after having shifted from one joint to another for many weeks, subsided; but the affection of the heart continued, generally attended with great pain, producing in the progress of the disease, and towards its close, a considerable disposition to dropsy, under which the patient lingered for many months."

As far back as 1788, it would seem that Dr. Pitcairn had noticed that persons subject to Rheumatism were attacked more frequently than others with symptoms of heart disease, and he considered that the two diseases often depended on a common cause. The connection between Rheumatism and cardiac disease was also known to Dr. Wells, Dr. Baillie, and others. The heart affection, however, was regarded by these authors rather as the effect of a metastasis of the rheumatic inflammation from the joints to this organ, than as an essential part of the disease itself. Some French authors claim the merit of the discovery of the close relationship for M. Bouillaud, who in his work, published in 1840, called special attention to the coincidence of pericarditis, endocarditis, and pleurisy with acute articular rheumatism. Since the above period, the relationship has been fully determined, and these diseases are now looked upon as portions of the rheumatic affection, and not as mere accidental complications.

There is considerable discrepancy of opinion among different authors as to the kind of cases of rheumatic fever in which cardiac inflammation is most likely to occur. Some are of opinion that even in the slightest forms of the disease, when febrile disturbance is moderate, the structures of the heart may become seriously affected. Others think that the liability to the heart disease is far greater in the severe forms of articular rheumatism, and that even when the joints are not much implicated, still the systemic disturbance is always great, and the peculiar phenomena of the disease strongly marked. As far as my own experience goes, it amounts to this, namely, that although the severer forms of the articular disease are very apt to be complicated with cardiac inflammation, yet even in the very slight forms, measured by the febrile and joint symptoms, serious mischief may arise in the heart; and several such cases have come under my own observation. When the heart is much influenced, this will of itself be a source of great constitutional disturbance. It is supposed by some pathologists that the vegetations which take place on the valves of the heart are not always due to endocardial inflammation, but may arise entirely independently of such, and are owing rather to an altered state of the blood itself.

It is a matter of extreme difficulty to determine the relative frequency of the cardiac affection in rheumatic fever; in young subjects the heart is much more prone to be attacked than in adults; the kind of treatment adopted in the early stages appears likewise, from the statistics of Dr. Dickinson, to have a considerable influence upon the result. Bouillaud thought the heart became affected in one-half of the cases, but Dr. Macleod in about one-fifth only.

Morbid Anatomy of Articular Rheumatism.—The opportunities of examining joints affected with rheumatic inflammation are not numerous, as the articular affection never kills, and a fatal determination, even when the heart or other important organ is attacked, is fortunately rare. In a few instances, where death has occurred when inflammation of the joints was actually presented, I have found considerable redness of the synovial membrane, and increased vascularity of the synovial fringes; an augmentation of the synovial fluid, and sometimes little gelatinous coagula of fibrin; under the microscope nucleated cells were seen but

none of the ordinary appearances of pus. In one case of mono-articular disease, the fluid was indeed turbid with pus cells, but in this instance, some doubt existed as to the true nature of the affection.

In Dr. Fuller's work a detailed account is given of the post-mortem appearances in sixteen cases of acute rheumatism in St. George's Hospital. In eight of these cases the joints were examined: in some of them the only morbid appearances were increased vascularity, a thick tenacious fluid, with granular globules or a few pus cells; in some the fluid was turbid, and soft fibrinous coagula were found; in other cases pus is described as being present both in the joints and likewise along the tendons.

In two cases nothing abnormal was observed: but in one the inflammation had subsided some weeks before death; and in the second, as far as the history is given, the local mischief was not great.

Chomel, who examined a knee-joint two days after the cessation of acute rheumatic inflammation, found an excess of synovial fluid, but no other morbid change.

In examining joints which had been frequently and severely affected in rheumatic fever, but in which the inflammation had completely subsided, the only appearance I have observed, indicating any deviation from the normal condition, has been a lax state of the ligaments of the articulations, and an opacity of the articular cartilages, or a loss of the natural bluish-white opalescence of this tissue.

No mention has been made of ulceration of the articular cartilages, and I have had proof that at least a dozen attacks of rheumatic inflammation may occur in a joint without any such change taking place.

In no case has the slightest trace of urate of soda been found, although very carefully looked for, and on one occasion I had the opportunity of examining and comparing two knee-joints—one that of a young man who had died from heart disease at the time that the joint was suffering from recent rheumatic inflammation; the other, that of a man who had been killed by an accident, which had previously caused the development of gouty inflammation of the part. The difference in the two cases was very striking: the vascularity in each was about the same; in the latter there was the characteristic white thickening of the cartilages from the crystalline deposition, and in the former the complete absence of such an appearance.

For the changes which ensue when rheumatic inflammation attacks the pericardium, endocardium, or substance of the heart, as likewise when the pleura or membranes of the brain are affected, we must refer the reader to the articles on the various diseases of those structures.

Causes of Rheumatism.—The causes of acute rheumatism may be divided into (1) those which belong to the individual, and (2) those which are external to and independent of the patient.

(1) *Influences dependent on the Individual.*—*Hereditary Predisposition.*—Although the influence derived from this cause is not so well marked as in the case of gout, still it is very powerful. Chomel and Requin stated that, in 72 cases, 36 had rheumatic parents, 24 had healthy parents, and 12 were unable to give any information upon the point. This statement, however, is of little value, from the fact that these physicians did not distinguish cases of Rheumatism from those of gout.

Dr. Fuller traced an hereditary influence among the rheumatic patients admitted into St. George's Hospital, in nearly 29 per cent.: the writer has found that in about one-fourth of his patients hereditary predisposition could be traced; many other authors have come to a similar conclusion; and it may be looked upon as an established fact, that a disposition to Rheumatism can be inherited.

Age.—Young people are much more liable to be attacked with rheumatic fever than those more advanced in years, and in this respect Rheumatism differs essentially from gout. Heberden gives an instance in which Rheumatism occurred in a child only four years old. Others have made similar statements. Haygarth states that rheumatic fever affects persons of all ages from five to seventy-two inclusive; more frequently from six to thirty, but most frequently from sixteen to twenty years of age.

Sex.—In a table given by the writer of 51 consecutive cases of rheumatic fever which came under his care, 31 were females, and 20 were males. There are, however, many discrepant statements with regard to this point. From many statistics it would appear that men are more subject to Rheumatism than women: thus it was found that out of 289 cases of acute rheumatism admitted into St. George's Hospital, 151 were males, and 138 were females: and again, out of 136 cases of the same disease recorded by Dr. Latham, 75 were males, and 61 females. In Dr. Haygarth's cases, 99 were males, and 71 were females.

It has been thought that women, after the catamenial period, are as liable to be attacked with Rheumatism as men. I have no hesitation in saying, that if this remark applies to true articular rheumatism, it has little or no value; for after the age of forty-eight it is very rare to find either men or women affected for the first time with this disease. Women, however, are prone to have both gout and rheumatoid arthritis upon the cessation of the menstrual function.

State of Health.—There can be little doubt that articular rheumatism is more apt to become developed in individuals who have from some cause or other become weakened, either from insufficient nourishment or from disease: hence one cause of its more frequent occurrence amongst the poor and ill-fed than amongst the rich. It is very common to meet with it in women who are suckling, the debility arising from over-lactation giving them greater liability to be attacked.

(2) *Influences external to the Individual.*—Cold is certainly a very common exciting cause of articular rheumatism, and on referring to tables which have been constructed by different observers it will be found that this cause is far more potent than any other. The majority of patients are able to trace their malady to some one direct exposure to cold, and especially to cold combined with moisture. The more liable the person is from causes within himself, the less is the exposure which is necessary to develop the disease; a cold draught when over-heated, a damp bed, getting thoroughly wet through and becoming chilled, are the circumstances most prone to excite articular rheumatism. It would appear probable that cold acts by causing a sudden check to the function of the skin.

Scarlatina is often followed by an articular affection which has all the characteristics of acute or subacute rheumatism, but which must be separated from the fearful purulent disease of the joints occasionally met with under these circumstances. If the Rheumatism which follows scarlatina is of the same character as rheumatic fever, its occurrence may be explained by the fact, that the scarlatina poison acts especially upon the skin, and greatly influences the cutaneous function, which is proved by the subsequent severe desquamation of the cuticle.

It has been supposed that other causes, as the suppression of the menstrual discharge, or of habitual fluxes of various kinds, can act as the exciting cause of articular rhematism; but if the recorded instances are carefully inquired into, it will be found that they are either cases of gout, or of some kind of purulent joint affection.

In a certain number of cases of rheumatic fever, the patients are unable to state any exciting cause of the attack, and if the predisposition to the disease is very great, it may often occur without any appreciable cause.

Effects of Climate, Seasons, and Weather.—Dr. Aitken gives, in his work on Medicine, the returns, by Colonel Sir A. Tulloch, of Rheumatism in the regiments stationed at home and in the different colonies belonging to this country. Out of 1000 soldiers admitted at the military hospitals, in Jamaica, 29; Nova Scotia and New Brunswick, 30; Bermuda, 33; Malta, 34; Ionian Islands, 34½; Gibraltar, 38; Canada, 40; Mauritius, 46; Windward and Leeward Command, 49; United Kingdom, 50; and Cape of Good Hope, 57, were sufferers from Rheumatism. The following passage from the above work contains all that is known on this subject:—"It is not, therefore, in the coldest climate that Rheumatism is most prevalent, but in those seasons and in those climates remarkable for damp and variable weather; and thus, says Sir A. Tulloch, 'we find in the mild and equable climate of the Mediterranean or the Mauritius the proportion of the rheumatic affections even greater than in the inclement regions of Nova Scotia and Canada; and though some of the provinces of the Cape of Good Hope have been without rain for several years, yet Rheumatism is more frequent in that command than in the West Indies, where the condition of the atmosphere is as remarkably the reverse.' Exposure to heat, however, would appear to have much influence in the production of Rheumatism; for we find that returns of the navy show a considerably larger proportionate number of attacks than those of the army—the number per thousand annual mean strength attacked in the Mediterranean fleet being 63·9, in the West Indies and North American station 69, and in the South American station 72·3."

Chomel remarks that Rheumatism is seldom met with near either the equator or the poles, but that it becomes more frequent as we proceed from these regions, and that it is more especially rife in the temperate zones.

In England the occurrence of rheumatic fever appears to be much more common in the eastern than in the western counties: this is usually accounted for by the exposure of the former portion of the country to the northeast winds.

As far as seasons are concerned, it may be stated that no part of the year is exempt. Haygarth's tables give the following results in 150 cases:—The disease began in January in 21; February, 11; March, 9; April, 18; May, 11; June, 11; July, 14; August, 5; September, 8; October, 13; November, 13; December, 16.

It will be found, however, that in different years the relative number for each month is liable to considerable variation.

PATHOLOGY OF RHEUMATISM.—The name Rheumatism implies that the disease has been considered to be dependent upon some altered condition of the blood, and the ancients, confusing Rheumatism with gout, necessarily regarded it as having the same humoral origin.

One set of pathologists look upon Rheu-

matism simply as an inflammation of certain fibrous and serous membranes. This was the view held by Cullen, who remarks that, although some have attributed the disease to the existence of a peculiar acrimony in the system, it was without reason.

Sir C. Scudamore held the same views as Cullen, and makes the following remark:—"It may be stated, that the predisposition to Rheumatism consists in a deficiency of healthy tone in textures connected with joints and muscles, and in nerves, so as to be affected in this peculiar manner by the influence of variable temperature. If we lose sight of the humoral term Rheumatism, we shall come to the simple fact, that, in a condition of susceptibility, cold or sudden reduction of temperature, makes a particular impression on the vessels and nerves near the surface, and produces a painful affection of certain textures, which is attended with more or less inflammation, the phenomena of which are so far of a peculiar nature, that we either consider the disease specific, inasmuch as the symptoms differ in their constituent character from those produced by other inflammations, or we may view the effect in the light of common inflammation, modified on the one hand by the nature of the exciting cause —the external one cold—and on the other hand by the particular species of textures which become affected."

With slight modifications, quite unnecessary to detail, this view resembles that of some authors of the present day who look upon Rheumatism as unconnected with any essential morbid state of the animal fluids.

Similar objections to those which have been raised against Cullen's views of gout apply to the above attempted explanation of the pathology of Rheumatism.

A second set of pathologists consider the phenomena of Rheumatism as produced by the presence of some peculiar principle in the blood, either one altogether foreign to its healthy constitution, or an augmentation of some normal ingredient.

Dr. Prout first threw out the idea that lactic acid was the principle in question, and this view has found many supporters. It has been argued that the skin is the great eliminator of lactic acid; that cold is the chief exciting cause of Rheumatism; and that, perhaps, bad or insufficient nourishment may lead to an increased formation of this acid, and cold to its defective excretion, and the combined influence may cause a large accumulation in the blood. This explanation of the disease has much in it which is probable, but at present it is devoid of anything like absolute proof. The only observations which appear to give direct support to it are those of Dr. W. B. Richardson, who has shown that when lactic acid is injected into the peritoneal cavity of dogs, it causes both peri- and endo- carditis. The post-mortem appearances in different animals exhibited a red, thickened, and œdematous state of the mitral valve, with fibrinous bead-like deposits on its surface; the endocardium lining the left ventricle was intensely vascular, resembling bright-red velvet; and the aortic valve was of a deep-red color, its fine borders being thickened and everted; the pericardium was red and injected with effused lymph upon the surface of the ventricle. In some cases the tricuspid valve exhibited a similar injected condition. No swelling of the joints was observed during the life of the animals, and no morbid appearances exhibited after death.

These experiments are instructive, but they only prove that the absorption of a large quantity of a free acid by the perineum leads to inflammation of other surfaces of a similar nature; and it has yet to be shown whether like phenomena would not be induced by the absorption of other acid or acrid substances. If the injection of other acids, as acetic and formic acid, should produce the same results, there would be no proof that Rheumatism has any relation to lactic acid.

The pathology of articular rheumatism must be allowed to be in a very unsettled state, and further observations and experiments are required before we can arrive at any satisfactory conclusion with regard to it.

Diagnosis.—It will be necessary under this heading to consider the salient points of difference which exist between Rheumatism and the diseases with which it is liable to be confounded.

The affections for which acute rheumatism is likely to be mistaken are acute general gout, acute rheumatoid arthritis, pyæmia affecting the joints, and the acute forms of urethral rheumatism.

From acute general gout it may be separated by the following peculiarities:—

The absence of uric acid in the blood.

The prevalence of extreme sweating, and the acrid odor of the perspiration.

The great amount of constitutional disturbance compared with the joint affection.

The tendency to acute cardiac inflammation.

From acute rheumatoid arthritis, by the great constitutional disturbance, the acrid sweats, the liability to cardiac inflammation, and the erratic nature of the joint disease.

From pyæmia, by the erratic nature of the inflammation, by the great amount of perspiration, by the liability to cardiac inflammation, and by the absence of the

disorganization of the joints produced by the latter disease.

From acute inflammation dependent on urethral suppuration, by ascertaining the history of the case, the greater constitutional disturbance, the more general character of the joint affection, and the absence of severe disorganization of the articulations.

The subacute form of articular rheumatism may usually be distinguished from chronic gout and rheumatoid arthritis by attention to the above differences and a careful inquiry into the history of each case.

PROGNOSIS OF ARTICULAR RHEUMATISM.—Articular rheumatism, even in its most acute form, though extremely painful, can scarcely be looked upon as a very fatal malady, but it may lay the foundation of the most serious disease of the heart. Dr. Aitken states that the deaths from rheumatic fever hardly exceed one out of every thousand cases of deaths from all causes, but this applies only to those immediately resulting from the attack; it is almost impossible to arrive at anything like a correct estimate of the proportion who die from its after-effects. When rheumatic fever proves fatal, it is either from acute disease of the heart, probably involving the substance of the walls of the organ, and leading to sudden collapse, or, much more rarely, death may result from acute cerebral disease.

The prognosis in acute rheumatism is also good as far as relates to the state of the affected joints; it is seldom that any very permanent injury is produced, unless the patient is otherwise out of health, and the rheumatic inflammation becomes the exciting cause of other serious and destructive action.

For an account of the after-changes which ensue from the cardiac affection, the reader is referred to the articles on Pericarditis, Endocarditis, &c.

TREATMENT OF ARTICULAR RHEUMATISM.—Numerous methods have been proposed for the treatment of articular rheumatism, and each has had its advocates; but, until recently, few have considered it feasible to leave such cases to nature, merely taking care that the patient be placed under favorable hygienic conditions. Many practitioners who would unhesitatingly adopt either a severe depleting or powerfully stimulating treatment, would shrink with alarm from the very idea of letting nature have her own way.

For several years I have treated some of my rheumatic patients in hospital on a purely expectant plan, and although the cases have not yet been tabulated, some of the results have been so decided as not to be mistaken. I am quite certain that many cases even of severe rheumatic fever get rapidly well without the administration of drugs, and on simply colored or camphor water the improvement is often so quick and satisfactory, that had not the nature of the treatment been known, great virtue would surely have been ascribed to it; on the other hand, in many instances, the disease runs a lengthened course, with many partial relapses;—such tardiness is often found under other plans of treatment. In the Guy's Hospital Reports, 1865, are two communications containing the result of the treatment of forty-one cases of rheumatic fever, thirty-seven under Dr. Gull, and four under Dr. G. O. Rees, scarcely any medicine being given except mint water. Of the forty-one patients, twenty-two were males and nineteen females. The majority were under thirty years of age, only two above the age of forty years.

	Males.	Females.
The average number of days from the admision into the hospital to the cessation of pain; skin cool, and no relapse	13·1	15·5
The average number of days from the admission to complete convalescence; out of bed, and no relapse . .	16·6	21·4
The average number of days in hospital	27·6	26·8

The cases were all well-marked instances of rheumatic fever. In a large number of them the heart was implicated, and in such the duration of the symptoms was much longer: thus, the average duration of the acute symptoms in seven cases, in which there was no evidence of the heart being involved, was 8·5 days, but in six cases in which the heart was decidedly affected, 23·6 days.

The effect of the non-medicinal treatment on the supervention of heart disease could not be determined, as in the large majority of instances abnormal cardiac sounds were heard on admission.

Further trials of the expectant plan are much wanted, and every detail with regard to the condition of each patient should be carefully noted down and published, in order that the cases may be fairly analyzed.

We will now give a short summary of the different methods which have been adopted, and have gained favor with the profession.

Venesection has been largely employed, and considered one of the most important curative agents in rheumatic fever. Sydenham was a strong advocate for this practice, and Cullen considered blood-letting to be the chief remedy in acute rheumatism, and thought blood ought to be

drawn in large quantities, and the bleeding repeated in proportion to the frequency, fulness, and hardness of the pulse, and to the violence of the pain: he, however, thought that some bounds should be set to the bleeding; otherwise the recovery might prove slow. Bouillaud, in France, carried this plan to the fullest extent, and many English physicians during the present century have not been sparing of the lancet.

The condition of the blood in acute rheumatism, as shown by the buffy coat and cupped condition of the clot, evidently favored the idea that bleeding was necessary in rheumatic fever, as in other forms of acute inflammation.

Venesection certainly gives speedy relief to the pains of the joints, even when practised to very small amounts, but free depletion tends to weaken the patient, and retard his restoration to health; it also causes the joint affection to linger, and favors relapses. Whether there is any real advantage in small depletions is questionable; I should certainly hesitate to bleed in the majority of instances, as there is no proof that it either shortens the duration of the joint affection, or lessens the tendency to cardiac complication.

At the time when blood-letting was commonly employed, some physicians were inclined to look upon it as of doubtful efficacy. Thus, Heberden states that, as far as he has been able to observe, the benefit of large and repeated bleedings is in most cases far from being clear and unquestionable.

Fordyce also remarks that, while it was the practice to remove the general inflammation by bleeding, metastasis frequently took place to internal organs and destroyed the patient, but that during fifteen years, when he entirely left off bleeding in acute rheumatism, he only lost two or three patients, although he treated several hundreds during that period. And, lastly, Dr. Willan states that he had observed that, by frequent bleedings, the pains, swellings, and febrile symptoms were not only aggravated at the time, but often protracted indefinitely, and that sometimes under such treatment the disease had lasted upwards of two months.

Mercurials, especially calomel, but a few years since were regarded as almost absolutely essential in the treatment of acute rheumatism, and even at present there are comparatively few who would venture to treat the cardiac complication without their aid: it is, therefore, important to decide the question of their utility.

If we compare the results obtained from cases treated freely by mercurials with those treated without mercury, it will be seen that, as far as the joint affection is concerned, the latter are decidedly more favorable than the former: such being the case, it seems difficult to understand why, if the inflammation of the heart's membranes be identical with that of the structures of the joints, mercurials should be of more value in the cardiac than in the articular disease.

Opium has usually been combined with calomel, partly to check the action of this drug upon the bowels, and partly to alleviate the painful affection of the joints. That opium in this disease is far more valuable than the calomel with which it is conjoined, is beyond doubt; it gives relief from the pain, promotes sleep, and prevents exhaustion of the nervous system; at the same time it encourages the true action of the skin, by increasing the capillary circulation.

A small dose of some preparation of opium, or of a salt of morphia, may generally be advantageously administered at night, and even once or twice a day, if the symptoms calling for its employment be urgent. Moderate doses of calomel may now and then be combined with opium, to promote the excretion of bile, and prevent constipation.

It is probable that opiates act merely as palliatives, and have no direct influence on the progress of the disease.

The use of *antimonial preparations*, more especially of tartar emetic, has been advocated in acute rheumatism; but experience seems to show that they have no power in checking the duration of the joint disease or of mitigating the cardiac affection; they are not much employed at the present time, except in very small doses, and then simply with the view of promoting the action of the skin.

Colchicum is a medicine which is still much used by a large number of practitioners, from the impression that gout and Rheumatism are affections closely allied to each other, and consequently that any drug valuable in the one disease must necessarily be useful in the other. Of the powers of colchicum in subduing true gouty inflammation there is no possible doubt; but in regard to acute rheumatism the case is very different. As the result of my own experience, I may state, that colchicum possesses no influence in checking the progress of rheumatic fever; that when given in large doses, so as to lower the tone of the vascular system, it affords temporary ease, but not more than any other vascular depressant; and furthermore, that colchicum is a dangerous drug in many cases, as it acts as a direct cardiac sedative, and the chief danger to be feared in acute rheumatism is the loss of power of the heart.[1]

[1 This statement is undoubtedly true, as a rule; but it requires modification in regard to cases of Rheumatism in patients inheriting a gouty constitution.—H.]

Cinchona Bark and Quinine.—Although bark had been previously employed in the treatment of acute rheumatism, by Drs. Morton, Hulse, and Fothergill, Dr. Haygarth was the first who used it extensively, and brought forward clinical evidence of its utility; but the treatment did not appear to gain much favor with the profession; doubtless the large amount of cinchona bark necessary to produce a decided effect upon the system was a great drawback upon its administration: the substitution of quinine for the bark itself removed this objection, and the sulphate of the alkaloid has been tried very largely in France, and in very considerable doses, varying from one to six grammes in the twenty-four hours. From the statements of Briquet, Monneret, and Vinet, this remedy has a very decided influence upon the course of the fever, acting as a powerful sedative upon the circulation, and allaying the pain and swelling of the articulations; it often appears to prevent the development of cardiac complication, and even when this is present, it exercises no injurious influence. The effect of the quinine upon the disease is most decided when given in full doses, even to the extent of producing uncomfortable symptoms in the head and stomach.

Alkaline and Saline Treatment.—The treatment of acute rheumatism by saline and alkaline remedies has long found favor with the profession, and many modifications have been adopted; but before speaking of their relative merits, it will be well to define clearly what is meant by the saline and alkaline treatment.

There are certain saline remedies which, after absorption into the system, are eliminated by the kidneys in the same state as when they enter the stomach; for example: (1) nitrate of potash, chlorate of potash, and other salts in which the base is conjoined with a mineral acid; (2) alkaline salts with carbonic acid, in the form of the neutral or bicarbonate of the base; (3) salts with alkaline bases united with a vegetable acid, as citric or tartaric acid. Although these salts are neutral in reaction, when introduced into the stomach, they become speedily altered in the blood, the acid is decomposed, and a carbonate of the base appears in the urine; and hence, although they produce no alkaline effect upon the mucous membrane of the alimentary canal, yet upon the blood and the secretions their alkaline effect is well marked. Whether this effect on the blood is exactly the same as that of the free alkalies, or their carbonates, has not yet been determined.

Saline remedies have sometimes been employed in small doses, simply for the purpose of acting upon the secreting organs; at other times they have been given in very large doses, in order to alter the character of the blood itself, or powerfully influence the vascular system.

The first saline to be discussed is nitrate of potash. This remedy was used about a hundred years ago by Dr. Brocklesby, in conjunction with bleeding. He ordered a dilute solution of the salt in water-gruel (about 120 grains to the quart), and as much as an ounce or more of nitre in the twenty-four hours. With this he states that he cured many cases in seven or eight days.

Since the above date it has been employed by M. Geudinn, M. Martin-Solon,[1] and in this country by Dr. Basham.[2] From M. Martin-Solon's communication we cannot deduce the real value of the treatment, as the details are not sufficiently recorded, and it has been shown that under any treatment many cases get well in a comparatively few days.

Dr. Basham states that one, two, or even three ounces of the nitrate, freely diluted, may be taken in the twenty-four hours without inconvenience. He looked upon and employed the nitre as an adjunct to other remedies, and hence it is difficult to determine from his table of seventy-nine cases of acute rheumatism the real influence of the salt in combating the disease. Dr. Basham considers the local application of nitre of great value in causing abatement of the pain and swelling of the joints.

The nitre treatment, upon the whole, seems to have been followed by good results, and in Dr. Dickinson's record of seven cases treated in St. George's Hospital a favorable result was obtained.

Alkaline Treatment.—Although an apparently over-acid state of the body, and an increased amount of fibrin in the blood, in acute rheumatism, would naturally suggest the value of alkalies as remedies for this condition, it does not appear that they were systematically employed until the year 1847, when Dr. Wright published a communication on the subject. Since that period both Dr. Fuller and the writer have made a full trial of them, one administering the alkali combined with some vegetable acid, the other prescribing it in the form of the bicarbonate. Dr. Fuller's results will be found in his work on Rheumatism; the writer's earlier trials in a communication to the Medical and Chirurgical Society in 1855, in which an account is given of fifty-one consecutive cases, treated upon the full alkaline plan. The average duration of the disease in twenty males was 11·3 days, the duration under treatment 6·2 days; the average

[1] De l'Emploie du Nitrate de Potasse à haute Dose dans le Traitement du Rheumatisme articulaire. (Bulletin gén de Thérap. août, septembre et octobre, 1843.)

[2] Med.-Chirur. Trans. vol. xxxii.

duration of the disease in thirty-one females was 15·7 days, the duration under treatment 7·3 days. In no case did any heart disease occur after the patient had taken the remedy forty-eight hours.

The plan consists in administering a dilute solution of bicarbonate of potash in about thirty-grain doses, every four hours, until the joint symptoms and febrile disturbance have completely disappeared. These doses produce no inconvenience either to the stomach or bowels; the urinary secretion is not notably increased, but its character is completely altered, and the reaction becomes either neutral or alkaline; it usually remains clear, but occasionally gives rise to a deposition of the triple phosphates. Upon the heart the alkaline bicarbonate acts as a sedative, reducing the frequency of the pulse sometimes forty-eight beats in the minute, but not causing any faintness.

When a patient is fully under the alkaline treatment, the blood is distinctly altered, and the coagulation of the fibrin takes place more slowly.

Many other remedies have been proposed for the cure of acute rheumatism, some of which it will be only necessary to enumerate: lemon juice has been prescribed by Dr. Perkins and Dr. Ciraud on the Continent, and in this country by Dr. G. O. Rees. It is usually given in quantities of from three to eight fluid ounces each day, and it is supposed to act as a sedative to the vascular system. It is doubtless a fact that, under its use, many cases of the disease rapidly get well, but, as has been shown above, this must not be taken as a proof of its curative power. and before we can decide upon its value, it will be necessary to have the tabulated results of a large number of trials of lemon juice, and to compare them with those obtained from other methods of treatment.

Other remedies used, either for the cure of the disease or the alleviation of certain of its symptoms, are, iodide of potassium, guaiacum, aconite, emetics, purgatives, diaphoretics, and hot-air baths.

Iodide of potassium is used to relieve the obstinate pains which linger when the acute symptoms have passed off, and more especially pains which are increased by heat, and most troublesome at night.

Guaiacum is valuable in subacute cases, when the circulation is weak, and the pains relieved by the application of warmth.

Aconite has been proposed to alleviate the acute pain of the joints, but when given in efficient doses it is apt to cause depression of the heart's action, and may be dangerous.

Hot-air baths exert a soothing influence, but great care is required if used during the time of febrile disturbance; and their curative value is somewhat questionable.

No proof has yet been given of the value of a free administration of emetics or purgatives; but proper attention must be paid to the state of the bowels.

[Since its introduction to the notice of the profession by Stricker, of Berlin, in 1876,[1] the treatment of acute Rheumatism with salicylic acid has received much commendation from practitioners on both sides of the Atlantic. Amongst those early observing its efficacy have been Traube, Broadbent, Maclagan, Sée, Hérard, Beaumetz, Jaccoud, and Lépine.

The doses reported by Stricker and Sée were large. The former gave twenty or thirty grains every hour for six doses; the latter, a drachm and a half daily, in five doses; or, an equivalent amount, two drachms and a half of salicylate of sodium. Other practitioners, however, have found that smaller quantities will answer the desired purpose; while very large doses are not free from danger. Empis and Jaccoud have reported cases of sudden death, not improbably explained by excessive doses of the drug. Probably ten grains every hour for five or six hours will usually suffice to make the powerful impression required. After that, the same or a less amount may be given thrice daily, until convalescence is assured. Under its influence two or three days will often be the whole period of severity of the symptoms. Cardiac complications are, there is reason to believe, less frequent under this than under any other treatment.

Salicylic acid may be given in glycerin, or dissolved in alcohol with the aid of citrate of ammonium.[2]

Salicylate of sodium is generally accepted as, on the whole, the most available form of combination of the acid. From a drachm to two drachms or more of the salt may be given during the first day of treatment; with diminished doses afterwards. No other mode of treatment of acute Rheumatism has, as yet, received so much testimony in its favor.—H.]

Treatment of Heart, Lung, and Brain Complications in Acute Rheumatism.—Having given an account of several of the more important methods of treating rheumatic fever, it is desirable that we should inquire if any deviations are necessary when inflammation attacks the structures of the heart, lungs, or other internal organs. It has been already stated, that

---

[1 Berliner Klin. Wochenschrift, Nos. 1 and 2, 1876.]

[2 An ounce of alcohol, with a drachm of citrate of ammonium, will dissolve two drachms of salicylic acid. Cassan, of Paris, first proposed this solvent. *Bull. Général de Thérapeutique*, April 30, 1876.—H.]

the articular inflammation leads to but little mischief, and that the joints rapidly recover their healthy state. It is, however, very different in the case of the heart; for there is a great disposition, both in the endocardial and pericardial serous membranes, to throw out lymph, which may lead to the thickening of the valves and adhesion of the surfaces of the pericardium; it is therefore a matter of no little moment to ascertain whether any plan can be adopted either to prevent such mischief supervening, or of rapidly and efficiently checking it if it has already taken place.

There appears to be every probability that the inflammation of the serous membranes of the heart is of the same kind as that of the joints, but it must be remembered that the structures themselves are of a somewhat different character, and remedies which produce little or no effect upon the joints may cause a decided action upon the cardiac tissues. It must also not be forgotten that inflammation lingers much longer in the heart, and is modified by the incessant movement of the organ.

It is a very common practice in cases of pericarditis, or even of endocarditis, to apply leeches over the cardiac region; and it is a clinical fact, that the tightness of the chest and pain are decidedly relieved by their application. Although it is difficult to explain the value of the local abstraction of blood in these cases, still I believe it is undoubted, and I cannot therefore hesitate to recommend it. The loss of blood need not be large, from three to twelve leeches are generally sufficient, and the bleeding should never be allowed to produce any appreciable weakening of the patient. Cupping may be employed in lieu of leeches, but I am inclined to prefer the slow loss of blood by the use of leeches, to its more expeditious abstraction by the cupping-glass.

Blisters applied to the heart's region are also of much value, either before or after the application of leeches: care should be taken by previously applying collodion to the leech bites, and covering the surface of the plaster with tissue paper, to avoid the absorption of cantharidine, and the production of renal irritation. Now and then, if there be much effusion into the pericardium, the blister may be kept open, either with savine ointment, epispastic papers, or by some other means. When leeches have not been previously employed, the above precautions are less necessary. The simple application of a blister over the cardiac region is productive of much relief to the patient, and is followed by a decided improvement in both the heart's movements and sounds.

A very important question in the treatment of the cardiac complications relates to the value of mercurials. It has been shown that the mercurial treatment of the joint affection does not prevent the occurrence of inflammation of the heart, and it only remains to be ascertained whether this metal has any power of arresting inflammation after it has once ensued. *A priori* it appears scarcely probable that a remedy, which has no influence in preventing inflammation, should have the power of arresting it when it has already commenced; but as the mercurial treatment is strongly advocated in such cases even by some who do not consider it as a preventive, it will be necessary to inquire a little further into the matter.

A few years since, almost every practitioner would have given this drug, and many at the present day scarcely dare omit its administration; for it is supposed to limit and control the inflammatory action, and to cause the absorption of the products which have been thrown out.

During the time of great febrile excitement it is very difficult to get the system under the influence of mercury, and it may be that, when this is effected, it is rather due to the prior abatement of the inflammation than from the influence of the metal.

For many years I was in the constant habit of administering calomel in cases in which inflammation of the heart was present, but for the last eight or ten years I have not done so as frequently, and have seen no reason to regret the change of practice: the cardiac inflammation appears to have yielded quite as readily, and the patient, on the subsidence of the fever, has not had to suffer from ptyalism in addition to debility.

As the question cannot be considered in any way fully settled, it must be left to each practitioner to follow his own course, and form his own judgment on the value of the mercurial plan of treatment. When it is adopted, the common method is to give calomel in doses of from one-half grain to three or four grains, every four or six hours, usually combined with a small amount of opium, to prevent the purgative action of the mercurial. If much difficulty is experienced in producing the effect upon the gums, then inunction is often had recourse to, and this may be practised by causing a drachm or so of the blue ointment (unguentum hydrargyri) to be rubbed thoroughly into the skin of the inner side of the thigh, or into the axilla; sometimes a blistered surface over the heart's region is dressed with the mercurial, instead of the green ointment. I have frequently adopted this latter method, and should prefer it, if mercurials are employed at all: it may be that the peculiar effect of the metal is produced upon the tissues in the neighborhood of the blister, before the general system becomes affected by it.

During the whole course of treatment of the cardiac inflammation, the plan thought to be most advantageous for the joint affection should be steadily persevered with, as anything which favors the abatement of the systematic disease must also relieve the internal complications.

It is always a matter of the highest moment to insist upon the most complete quiet; constant movement of the organ must necessarily take place, but everything should be shunned which increases this movement, or adds to the work which the heart has to perform. All chance of mental agitation, as well as bodily exertion, should therefore be strictly avoided.

After the inflammation has subsided, the heart is generally left in an irritable state; to allay this, a belladonna plaster is useful, and the administration of small doses of digitalis, combined with some salt of iron. More or less anæmia is always produced during the febrile disturbance, and this tends to keep up the excitement of the heart; and hence the value of the ferruginous preparations.

If the pleuræ become inflamed during the progress of acute rheumatism, the affection may be treated upon the same principles as the pericarditis; leeches and blisters may be prescribed, and calomel and opium given, if considered of value by the practitioner; and the same remarks apply to the management of the brain and spinal complications which now and then arise.

Local Treatment in Acute Rheumatism.—In the majority of cases little or no local treatment is needed; the affected joints should be protected by some light covering, as cotton-wool, flannel, or even a light handkerchief—but nothing further is called for: but now and then the pain is so excessive, or the patient is so sensitive to it, as to render it desirable that some direct application should be made use of. Hot-water fomentations may be first tried, but if not found sufficient, belladonna may be added; a very convenient preparation for the purpose is the liniment of the British Pharmacopœia, which may be either sprinkled upon the hot flannel, or diluted with two or three times its bulk of hot water, and applied by means of a piece of lint, taking care to prevent evaporation by oil-silk or some other impermeable tissue. A solution of atropia, or atropia combined with morphia, forms a very clean and elegant substitute. The extracts of belladonna, henbane, conium, and aconite have been recommended, as also the tincture of opium and decoction of poppy-heads.

Alkalies and salines have also been extolled as topical remedies in acute rheumatism; Dr. Basham has employed a solution of nitrate of potash, and Dr. Fuller one of the carbonate of potash or soda. Leeches have sometimes been used to the inflamed joints: I have never seen occasion for their employment in genuine rheumatic fever.

Blisters applied so as to cover all the inflamed joints have been highly spoken of by Dr. Dechilly and others; Dr. Martin-Solon made a report on the subject to the Academy of Medicine in 1850, and in the discussion which ensued it was stated, that the treatment had no other effect than to quicken the subsidence of the inflammation in the joints. Dr. Herbert Davies has recently revived the use of free blistering, and orders armlets, wristlets, and even fingerlets of blister plaster, at the time when the inflammation is most acute; he recommends linseed-meal poultices to be subsequently applied, in order to promote the free flow of serum; he places these blisters entirely around the affected limbs, and in the case of the knees, orders them of at least three inches in width, regarding any slight strangury which may arise as of little importance compared with the benefit afforded by the free vesication.

According to Dr. Davies, the blister treatment causes a speedy diminution in the frequency of the pulse, rapid subsidence of the joint affection, and lessens the liability to cardiac inflammation; within twenty-four hours after the removal of the blisters, the urine is stated to become alkaline in reaction. Dr. Davies's results in a large number of cases appear to be favorable.

Quino-alkaline Treatment.—During the last ten years, since the publication of his paper on the treatment of Rheumatism with large doses of bicarbonate of potash, the writer has made a very extensive use of the following plan, from which he thinks he has obtained more valuable results than from any other; it may be termed the quino-alkaline treatment, and is thus practised: Sulphate of quinine is ordered to be rubbed up with a solution of bicarbonate of potash, to which a little mucilage and some aromatic, as tincture of cardamoms or spirit of chloroform, is subsequently added; each ounce-and-half dose contains five grains of the quinine and thirty grains of the potash salt, the quinine being reduced to the state of carbonate. To the adult the above dose is given each four hours, and persevered with until the joint affection and febrile disturbance have completely abated. When the quino-alkaline treatment was first made use of, a few days were allowed for the exhibition of the alkali alone, and then the quinine was added, but of late the quinine has been given from the first; it neither increases the thirst nor the furred state of the tongue, and its influence upon the heart is to lower its pulsations, but not to weaken them,—and

hence, when peri- and endo- carditis are present, its employment is not contra-indicated. If cardiac complications exist, local depletion and counter-irritation may be made use of.

In the subacute forms of articular rheumatism, the same plan may be employed, but in a milder form; that is, the doses may be smaller, or fewer in number.

If desired, the citrate of potash, or some other alkaline salt with a vegetable acid, may be substituted for the bicarbonate: when irritation of the intestinal canal is present, the bicarbonate appears to act as a valuable sedative, but when there is a tendency to constipation, the citrate or tartrate may be advantageously given, care being taken that absorption of the salt be not too much prevented by its action on the bowels.

The potassio-tartrate of iron may be added to the quino-alkaline draught after a time, and often with much advantage if any amount of anæmia is present.

Although the results obtained from a large number of trials have not yet been tabulated, the writer feels assured that the above treatment is much more efficacious than the simple alkaline plan, that there is far less tendency to the occurrence of relapses, and that the patient is left in a more satisfactory condition after the cessation of the febrile disturbance.

Diet and Regimen in Rheumatism.—In acute rheumatism, when the febrile disturbance runs high, food can only be advantageously given in the liquid form: it is, however, important to sustain the strength of the body as much as possible, and for this purpose milk, if it can be assimilated, is one of the best forms of nourishment, as it contains all the elements necessary for repairing the waste of the system. If milk in an undiluted state is too heavy, it may be mixed with an equal measure of soda water. Besides milk, beef-tea, mutton and other meat broths, and jellies, may be given. It is desirable also to introduce a certain amount of amylaceous and saccharine matters into the system; hence arrow-root, made with water, milk, or beef-tea, may be administered. Soda water, lemonade made with cream of tartar or citrate of potash, and flavored with lemon, toast and water, or plain water, may be employed to allay thirst. Alcoholic stimulants are seldom needed in the young subject, unless there is great depression from the presence of serious cardiac mischief: wine, in true Rheumatism, has no tendency to keep up the specific inflammatory action.

As the fever abates, and the power of the digestive organs returns, the diet may be impaired; at first bread, light puddings, then white fish, fowl, and afterwards ordinary meat: but care must be taken that the stomach be not distended nor the system disturbed by the presence of food incapable of being digested. Throughout the whole course of the treatment of Rheumatism, both in its acute and subacute varieties, the great object should be to sustain as much as possible the strength of the patient, for by this means the duration of the disease is diminished, and the subsequent recovery rendered less tedious.

During the height of the malady, when the skin is freely perspiring, care should be taken not to allow the surface to be suddenly cooled; for although the free action of the skin does not appear to give relief to the pains, yet a sudden check to it cannot fail to be injurious, and may lead to metastatic action.

Individuals who have once suffered from rheumatic fever should be extremely careful as to their clothing; they should always be completely clad in flannel, which may vary in thickness at different times of the year; the feet should be kept warm, and every precaution taken to avoid a chill.

B. Muscular Rheumatism.

Definition.—An affection of the voluntary muscles, of an inflammatory nature (?), but unaccompanied with swelling, heat, redness, or febrile disturbance.

Synonyms.—Designated according to the situation of the affected muscles, Lumbago, Torticollis, Pleurodynia, &c.; termed also Myositis, Myo-rheumatism, and Myodynia. [Myalgia, Inman.[1]—H.]

History.—By British writers this disease is often included, with other affections, under chronic rheumatism, a name manifestly incorrect, as the malady often assumes an acute character. On the Continent some authors have claimed for it a separate existence, and the disease can scarcely be looked upon as a chronic form of articular rheumatism, seeing that its pathology is different.

Description of Muscular Rheumatism.—Muscular rheumatism usually commences as an acute disease, but has a considerable tendency to assume a chronic form: it may affect any of the voluntary muscles, but is prone to attack certain sets rather than others. The seizures are frequently sudden; sometimes, for example, a patient awakes in the morning and finds himself incapable of turning in bed

[1 Dr. Inman applied the term myalgia, however, to pain of any origin, located in the muscles, including, therefore, that resulting from over-fatigue, &c.—H.]

or of twisting the neck, and the attempt to do so gives exquisite pain. If the affection is very acute, he may suffer pain even when quiet, for the muscles are often involuntarily thrown into a state of spasm; in the less severe forms the patient may be comparatively comfortable when at rest, but on the least movement he experiences agony by the paroxysm thereby excited. On examining the seat of suffering, no external phenomena are visible, but there may be slight tenderness on pressure; there is also a freedom from febrile excitement, at least at the onset of an attack, but as it progresses, thirst, loss of appetite, and heat of skin may ensue, probably due simply to the continuance of the pain, and the loss of sleep thereby occasioned. In muscular rheumatism the pulse is but little affected, and the urinary secretion preserves its normal state. A very important feature in this disease is the absence of inflammation of the heart, so characteristic of the articular affection. In the acute stages the symptoms increase towards evening, and are augmented by heat; but when the disease becomes chronic, the pain is not unfrequently relieved by its application.

The duration of acute muscular rheumatism is generally short, usually not more than a few days, seldom exceeding a week: but when its intensity has become mitigated, it often proves tedious, and may be prolonged for an indefinite period of time; it is also apt to be again lighted up, if the patient be exposed to any of its exciting causes.

The principal varieties of muscular rheumatism are as follows:—

Lumbago (lumbodynia), when the large masses of muscles on each side of the spine in the lumbar region are implicated. When acute it renders the patient utterly helpless; the most intense agony is induced on the slightest attempt to rise in bed, or even to turn in any direction.

Torticollis (cervicodynia, or crick in the neck) is a term applied when the muscles on one side of the neck are affected. The patient is compelled to hold his head awry, in order to relax the muscles. Sometimes the sterno-mastoid is principally implicated, sometimes the disease extends to the muscles at the back of the neck.

When some of the intercostal muscles are the seat of the malady, the affection is called *Intercostal Rheumatism* (pleurodynia): the symptoms are pain in some part of the chest, rendered intense by the act of breathing, but relieved by such pressure as prevents the movement of the ribs.

The above three names are in common use, but many others have been occasionally employed by authors desirous of designating diseases simply from their locality; and thus have arisen the terms Scapulodynia, Dorsodynia, Cephalodynia, according as the muscles about the shoulders, or of the dorsal region of the spine, or those of the head, are particularly implicated.

As any of the voluntary muscles are capable of taking on morbid action, these names might be multiplied indefinitely; the walls of the abdomen, the muscles of the limbs, the tongue, pharynx, diaphragm, are stated to be sometimes implicated; and even some of the involuntary muscles, as of the œsophagus, stomach, intestines, and uterus, appear to be susceptible of the disease.

Causes of Muscular Rheumatism.—These may be divided into 1st, those dependent on the peculiarities of the individual, and, 2d, those which are altogether external.

Causes dependent on the Individual.—True muscular rheumatism is seldom seen in very young subjects; it generally occurs after the age of puberty, and more commonly in full adult age.

One attack of the disease engenders a liability to its return, as is the case with articular rheumatism.

Individuals of a gouty habit are frequently attacked with a muscular affection, which cannot well be distinguished from ordinary muscular rheumatism.

Causes external and independent of the Individual.—The only external causes of muscular rheumatism appear to be cold and damp, more especially if combined with an over-use of the muscles: lumbago, for example, is often produced by straining the lumbar region; and cold draughts of air, as from a partially open window will frequently prove the exciting cause of an attack of torticollis.

Pathology of Muscular Rheumatism.—The nature of muscular rheumatism is usually regarded as closely allied to articular rheumatism, the difference in the symptoms being supposed to depend on the peculiarities of the structures which are affected in the two diseases. Some reasonable doubt may, however, be entertained of the correctness of this opinion, for the following reasons: If the proximate cause of the muscular affection is the same as that of the articular, the heart would probably become inflamed in a certain proportion of the cases; but it is not so: and again, there is an absence of the peculiar secretion from the skin, so marked in articular rheumatism. The state of the blood has not yet been ascertained, but there is reason for believing that the fibrin is not much augmented.

For the present we must be content to await the result of more numerous and searching inquiries, before attempting to

explain the true nature of muscular rheumatism.

Diagnosis of Muscular Rheumatism. — Acute intercostal rheumatism may be mistaken either for pleurisy, or for a neuralgic affection so frequent in hysterical subjects: from the former it may be separated by the absence of the friction sound and other signs of inflammation of the pleura; from the latter, by the absence of well-marked spinal tenderness. It is sometimes difficult to distinguish the chronic form of muscular rheumatism from the dull pains arising from other causes, as from progressive muscular atrophy, the presence of metallic poisons in the system—especially lead and mercury—and likewise the poison of syphilis; also from ordinary inflammation of the muscular tissue. Lumbago may be mistaken for a painful affection of the loins, arising from calculi or gravel in the kidneys.

A careful examination of the history is usually sufficient, in each instance, to enable us to arrive at a correct diagnosis.

Prognosis of Muscular Rheumatism. — Muscular rheumatism, although difficult to cure, is not attended with serious results; there is not the fear of cardiac inflammation, the great source of danger in articular rheumatism. The sciatica arising from lumbago is often both painful and obstinate.

Treatment of Muscular Rheumatism.—In the acute stages of muscular rheumatism, especially in lumbago, local depletion, generally by cupping, is resorted to. That this remedy gives temporary relief, there cannot be a doubt, but whether it be necessary or not is another question. My own opinion is, that it is seldom or never called for, and that the same relief may be obtained from other topical applications which are less open to objection. Hot fomentations are very valuable; as, for example, spongiopiline, wrung out of hot water, and applied either alone or sprinkled with tincture of opium, or the liniment of belladonna, or a combination of the two. When the affection becomes more chronic, then a thinner piline lightly sprinkled with belladonna liniment, with or without the addition of the volatile oil of mustard, proves very useful, acting as a counter-irritant, and at the same time relieving pain by the anodyne properties of the belladonna.

Turpentine fomentations, or stupes, may be substituted in some instances for the above applications.

Occasionally blisters are applied, and if the affection prove obstinate, they may be kept open, with the addition of morphia or some other anodyne.

The internal remedies which have been employed in cases of muscular rheumatism are numerous. When the affection is acute and recent, benefit often arises from the use of salines, which act on the skin and kidneys, and alter the state of the blood. From my own experience I should recommend the acetate of ammonia in large doses, combined with some alkaline salt, as the bicarbonate of potash, and with the addition of iodide of potassium. The time, however, soon arrives for the use of some medicinal agent, as quinine, which exerts a marked action upon the nervous system; and this may be advantageously combined with the other remedies. Sometimes large doses of the hydrochlorate of ammonia are useful; and in cases which occur in gouty habits, colchicum alone, or in combination with other medicines, may be prescribed with much benefit.

When the disease becomes chronic, medicines which increase the capillary circulation are generally useful: guaiacum stands at the head of the list, and may be given either in substance or as the ammoniated tincture; after guaiacum follows sassafras, mezereon, turpentines, resins, and balsams.

Sulphur in small doses is frequently of much advantage, and it can be administered in substance or in the form of the sulphur waters of Aix-la-Chapelle, of Aix in Savoy, or Barèges.

Arsenic is likewise occasionally adopted as a remedy in long-standing, obstinate cases.

When the pain has subsided, friction and electricity are often resorted to, in order to diminish the stiffness and to restore tone and activity to the muscles.

The diet should be generous, with a moderate amount of stimulants; the patient should be warmly clad in flannel, and every precaution taken to avoid chills.

GONORRHŒAL RHEUMATISM.

By Bernard Edward Brodhurst, F.R.C.S.

The affection which is known as Gonorrhœal Rheumatism consists of inflammation of and about the joints, following upon urethral irritation, and preceded for the most part by a muco-purulent urethral discharge.

Swediaur and Monteggria are said to have described this affection, and to have published cases illustrative of the disease; but there is not anything to be found in their writings which entitles them to the merit which has been claimed for them; and indeed there ought to be no hesitation in saying that the disease does not seem to have been recognized until Sir Astley Cooper mentioned it in his lectures. Mr. South says: "Gonorrhœal Rheumatism and gonorrhœal ophthalmia were, I believe, first mentioned publicly by Astley Cooper, and the first of these affections he considers is not an infrequent disease; but it appears to have been previously observed by the elder Cline, for, in reply to the question put to him by Cooper, whether he had ever seen rheumatism produced from gonorrhœa, he said, 'Several times.'"[1]

Sir Benjamin Brodie has given some pathological account of this disease, and having described its nature, he continues thus:—"The disease is usually described under the name of Gonorrhœal Rheumatism, though it is plain, from the course of its symptoms and from the effects of remedies, that it differs from ordinary rheumatism in many essential circumstances, and though there seems to be no doubt that, while it occurs in most instances as a consequence of gonorrhœa, it may take place quite independently of gonorrhœal infection."[2]

Sir Astley Cooper relates the following case:—"An American gentleman came to me with a gonorrhœa, and after he had told me his story, I smiled, and said, 'Do so and so,' particularizing the treatment, and that he would soon be better; but he stopped me, and said, 'Not so fast, sir; a gonorrhœa with me is not to be made so light of; it is no trifle; for in a short time you will find me with inflammation in the eyes, and in a few days after I shall have rheumatism in the joints. I do not say this from the experience of one gonorrhœa only, but from that of two, and on each occasion I was afflicted in this manner.' I begged him to be careful to prevent any gonorrhœal matter coming in contact with the eye, which he said he would. Three days after this I called on him, and he said, 'Now you observe what I told you a day or two ago is true.' He had a green shade on, and there was ophthalmia of each eye. In three days more he sent for me rather earlier than usual for a pain in his left knee; it was stiff and inflamed. I ordered some applications, and soon after the right knee became affected in a similar manner. The ophthalmia was with great difficulty cured, and the rheumatism continued many weeks afterwards."

That which is here so graphically related may be observed in almost every instance of Gonorrhœal Rheumatism; and, with the exception of the ophthalmia, it is exactly the manner in which every first attack of Gonorrhœal Rheumatism arises and proceeds. Gonorrhœal ophthalmia is much less frequently observed than gonorrhœal articular rheumatism: probably it does not occur more than once in a dozen instances of Gonorrhœal Rheumatism.

Gonorrhœa then being established, one or more joints become, in the course of ten days to three weeks, stiff, painful, and swollen, the patient having, perhaps, exposed himself to the weather, sitting or walking in wet clothes, or to a draught of cold air. At the same time the feet may be painful and the conjunctivæ inflamed; there will be considerable fever, with dry skin and a furred tongue. Probably, as the articular inflammation increases, the urethral discharge will diminish; again it will become more abundant, and at length cease or degenerate into a gleet.

The first attack of Gonorrhœal Rheumatism is invariably preceded by a specific gonorrhœal discharge; a subsequent attack may be preceded by urethral discharge which is not of a specific character; and, also, the same character of articular disease may be re-excited without the urethral discharge being developed.

Both the robust and the debilitated suffer from this disease, but they suffer differently. The fever and inflammation

[1] A System of Surgery, by Chelius, translated by John F. South, vol. i. p. 217.

[2] Pathological and Surgical Observations on the Diseases of the Joints. 5th edit. p. 43.

are proportionate to the plethora which may exist. In the young and plethoric the inflammation is of an acute character, and lymph is for the most part deposited on the synovial membranes, giving rise to false ankylosis; whereas, in the debilitated, serum will probably alone be effused. In both cases the joints are liable to be destroyed; in the former through the deposit of lymph and the production of false ankylosis, and in the latter through the destruction of cartilage.

The joint having become inflamed, a large effusion of serum takes place into the synovial cavity; but, though there may be great tension, suppuration never occurs. Absorption of the effused serum takes place, and the joint may resume its healthy action. The limb remains in a semi-flexed position during the period of effusion into the joint; for in the flexed position of the limb the surrounding structures are somewhat relaxed, and consequently they yield to the bulging membrane with its contained fluid. When, however, the hip is inflamed, the limb remains much more extended than in ordinary hip-joint disease.

The knee is more frequently affected than any other joint, being a large and complicated joint, and less protected by muscle from atmospheric influence.

After the first attack of this disease, the patient is exceedingly liable to a recurrence of it. Usually the second attack is, as the first, the result of gonorrhœa and exposure to cold and wet. But, although Gonorrhœal Rheumatism will now almost certainly be excited by a specific discharge, any urethral discharge or urethral irritation would seem to be sufficient to re-excite the disease when the patient has once suffered from it.

On this second occasion probably the inflammation will be less, but the joints will be longer in recovering mobility, and one or more will possibly remain ankylosed. Sir Benjamin Brodie relates the case of a patient who suffered from four attacks of this disease in the course of some few years, in whom he says that "inflammation of the urethra was in all of them the first symptom, which was followed by purulent ophthalmia, and afterwards by inflammation of the synovial membranes and swelling of nearly all the joints. In two of these attacks he attributed the discharge from the urethra to his having received the infection of gonorrhœa; and in the two others to the use of the bougie."[1]

Slight stiffness may remain for several weeks, and a crackling sensation will probably be communicated to the hand on moving the limb; but this may also at length cease, and the joint will resume its normal condition both in regard of size and motion.

Each attack is more virulent in its character than the preceding, and in proportion to the debility of the patient. The female seldom suffers from this disease, but whenever I have observed an instance, it has always terminated in ankylosis.

When gonorrhœal ophthalmia occurs, the conjunctiva, sclerotic, and iris may all become affected. It is not for the most part a severe form of ophthalmia, and it readily yields to treatment.

Although the effusion into a joint may be very considerable, dislocation never occurs, and in this point the disease differs widely from ordinary rheumatism, where the tendency is for the articular surfaces to become more or less displaced; but in this disease ankylosis is induced rather than dislocation.

In no case that I have met with has there been so much effusion into the joints, and so much pain, as in the following instance; neither have I met with a case where the patient enjoyed such redundant health. A dark-haired young man, who was attached as lieutenant to one of our regiments serving in a tropical climate, had contracted gonorrhœa, and having laid himself down on a low broad wall he fell asleep, and so remained for some hours, and until after sunset. He awoke in great pain, and, as it seemed to him, this pain was spread all over the body, but especially it was felt in one hip: here, however, the pain was so acute that it was with difficulty he could be removed to bed. Besides the hip, the shoulders, knee, and ankle became affected; but in none of these joints was the inflammation so acute as in the hip, and they all passed through this inflammatory condition without material injury, and recovered perfectly. In the hip the effusion was so great that it was thought suppuration must take place: the swelling, however, subsided at length, leaving the joint stiff and immovable. Until this effusion was removed he suffered an agony of pain.

Occasionally the urethral discharge alternates with articular inflammation, or it degenerates into a gleety discharge, while the articular inflammation assumes a chronic character. Thus, a young Jew, who had contracted gonorrhœa, was soon after seized with pain in several joints. The urethral discharge ceased as the articular inflammation became developed, and it recurred as the pain in the joints was removed. This continued for several weeks, until on a winter's evening he was exposed to cold, when, an access of inflammation occurring, he found during the night a finger-joint excessively painful and somewhat swollen. The joint became ankylosed in the course of some few

[1] Op. cit.

days. All the other joints recovered well. The gleet remained for many weeks.

The articular inflammation appears to be of a more injurious character when a second or third attack of Gonorrhœal Rheumatism occurs. The first attack may leave no trace behind; but a subsequent attack seldom fails to do so: it may cripple the patient for life, as happened in the following case of a young man, nineteen years of age, of a somewhat strumous diathesis. In this patient gonorrhœa appeared on the seventh day after infection. In the course of a fortnight the knees became painful and swollen, and they continued in this state for about two months, when the inflammatory condition subsided, and the joints resumed their normal condition. Two months after this first attack had disappeared, he was again infected with gonorrhœa. In ten days several joints became inflamed, as the right temporo-maxillary articulation, as well as the hips, the knees, the ankles, the shoulders, the elbows, and the thumbs. The upper limbs and the ankles recovered well and regained their mobility, but the hips, knees, and the jaw became ankylosed.

The following is a remarkable case, in which, after several attacks of Gonorrhœal Rheumatism, the disease was again set up without urethral discharge appearing. A gentleman, twenty-five years of age, acquired gonorrhœa, which was soon followed by pain and swelling of the knees. The urethral discharge appeared on the seventh day, and some few days later synovitis supervened with great effusion into the knee-joints. The skin was hot and dry, and he suffered acutely, so that every movement was attended with much pain. The urethral discharge continued for two months, and then ceased entirely, and the swelling and stiffness of the knees also at length disappeared, having lasted for three months. After another interval of three months this individual was again affected with gonorrhœa. The urethral discharge again appeared on the seventh day, and it continued for two months. After some days the left hip-joint became inflamed, as well as the ankle and tarsal joints. This attack was of a much more severe character than the former one—the effusion was greater and the pain more acute. After a period of ten months my patient was able to walk with the help of sticks. Stiffness and a painful condition of the limbs continued yet, however, for many months; but at length he regained the use of his limbs. After a lapse of several months, again he contracted gonorrhœa. On this occasion the symptoms closely resembled those which occurred on former occasions; but he never entirely recovered from the stiffness which resulted from this attack of articular inflammation. Now, both hips, both ankles, and one knee became inflamed, and he also suffered from ophthalmia. Ankylosis did not occur, but there was a certain amount of stiffness of the joints which prevented him from rising after he had been seated for some hours. About six months after he was able to walk about, he married. Painful attempts were made to consummate the marriage, but it was found to be impossible: Dr. Lever discovered that occlusion of the vagina had taken place. At this time, namely within a very short period of marriage, articular inflammation recurred. There was now, however, no urethral discharge whatever. But the articular inflammation proceeded, and at length produced ankylosis of every joint in succession; so that in five years the whole skeleton was implicated: the atlas was ankylosed together with the axis, and in consequence the head could not be moved; and all the vertebræ were ankylosed together; and the hips, knees, ankles, shoulders, elbows, wrists, and jaw were so firmly fixed that no movement whatever could be obtained,

An interesting example of this disease has been for some time under treatment in St. George's Hospital, of which the following are the principal points: Edward G——, aged 46, having been treated as an out-patient by Dr. William Ogle for some time, was at length admitted into Fitzwilliam Ward, on account of effusion into and great thickening about the knee-joint. The left wrist was firmly ankylosed, and the spine in its entire length was motionless—ankylosis of all the vertebræ having taken place. The ribs also were ankylosed, and the breathing in consequence was purely diaphragmatic.

In the year 1855 (thirteen years prior to his second admission), he was a patient of Dr. Wilson's, in Cambridge Ward, for Gonorrhœal Rheumatism.

Three months before he was admitted into the hospital, he was exposed in the hunting-field, being a groom, to wet and cold, at the same time that he was suffering from an attack of gonorrhœa. He was admitted with pain in the feet and shoulders, in the right elbow, and over the clavicles; but at this time the gonorrhœal discharge had stopped.

Since that period he has never suffered from gonorrhœa; but he has had pain in various joints, and gradually his back and neck have become stiff, as well as the left wrist and the right knee. He was scarcely aware that any morbid process was going on in the spine, for there was little or no pain, and during the whole of the time between his discharge from the hospital and his re-admission he was able to perform his duty as a coachman.

Treatment. — When Gonorrhœal Rheumatism is vigorously treated in the commencement of the attack, the joints may become affected in a slight degree only. If there be much constitutional disturbance and inflammatory action, purgatives may be exhibited, and a small quantity of blood may be taken from the arm; while local fomentations may be employed, and a splint of gutta-percha to keep the affected joint perfectly at rest. After venesection, a full dose of opium gives great relief, and if it is administered with ipecacuanha, as in Dover's powder, the secretion of the skin is increased. The sweating which is thus produced is beneficial; but this increased action of the skin is best promoted in the Turkish bath. I have known the pain about the joints to cease entirely in the bath. Some time since, I saw a gentleman who suffered very acutely from pain and inflammation consequent on this form of disease of the joints, and in whom the tension from effusion was also excessive. He was lodged by my desire in a house attached to a Turkish bath, and each day he was carried down into the bath. When profuse perspiration was obtained, the pain left him; and absorption of the fluid within the synovial capsules was certainly promoted by exposing the body to the high temperature of the bath.

In an acute attack, abstinence from flesh meat, as well as from fermented and distilled liquors, is absolutely necessary.

When such measures are promptly taken, an acute attack of Gonorrhœal Rheumatism may generally be cut short. When, however, inflammation tends to become chronic, iodide of potassium may be given with advantage: thirty to forty grains being administered daily. It is preferable to abstract a small quantity of blood from a vein than to apply leeches to the inflamed joints: leeches not unfrequently aggravate some of the symptoms, and induce also suppuration in the cellular tissue. For these reasons, any benefit to be derived from their use is doubtful, and at times they certainly increase the evil they are intended to mitigate.

After the first or second attack, or when the patient is debilitated, the treatment should be of a slightly stimulating and tonic character: depletion will aggravate all the symptoms, and increase the effusion. Opium may be given freely, and iodide of potassium in small doses. Gutta-percha splints should always be used during the period of effusion to prevent motion.

So soon as pain and swelling have ceased, gentle frictions with shampooing should be employed to restore mobility. Much time will probably be required to effect this object, and it may be necessary, if adhesions have formed, to flex the limbs forcibly after chloroform has been inhaled. In many cases mobility may be restored, even after very firm adhesions have been formed.

PART II.

LOCAL DISEASES.

INTRODUCTION.

BY THE EDITOR.

A FEW words are necessary to explain the principle of classification adopted with regard to the diseases which find their place in this, the first, section of "partial diseases," or "affections of particular organs."

The maladies which are treated of in these volumes have been in the first place divided into two large groups, "general" and "local," and the reason for such division has been assigned (Classification of Diseases, p. 32). Some diseases are "general,"—that is to say, they appear so to affect the whole body at once, that all its functions are impaired or altered; and not only so, but they are implicated to such an equality of degree that it is always difficult, and sometimes impossible, to say upon which system of organs, if upon any, the greater weight of the burden falls. Of such diseases Part I. of this System of Medicine contained the history. We have now to deal with another class of affections, the "local" or "partial," viz., with those in which we have little or no difficulty in localizing the disease; maladies with regard to which we say at once that they are diseases of the nervous system, or of one portion of the digestive system. It is not intended that such diseases are accurately limited to the particular systems from which they derive their names; for we know well that the digestion is, or may be, disturbed in epilepsy, in apoplexy, and in paraplegia; and, on the other hand, that no severe perturbation can occur in the stomach without the simultaneous development of some corresponding disturbance in the nervous centres. Furthermore, we know that there is sometimes great difficulty in determining whether we have to deal with a disease universal in its distribution at its onset, or with some primary lesion of a particular organ, the secondary effects of which are general; or, even allowing that some particular organ is especially affected, the affection may be of such kind that it is sometimes a matter of grave doubt as to the system of organs to which that particular one, in this special instance, may be considered to belong. Tubercular meningitis has been mistaken for typhoid fever, and *vice versâ;* abscess of the brain has been confounded with malarial poisoning; and, again, tumors in the brain have been regarded as diseases of the stomach; while all the symptoms of cancer of the stomach have been explained away by the fiction of "spinal irritation." Each organ of the body has something in common with every other organ; and although two viscera may be, locally, as remote as the limits of the human body will allow, there are between them bonds of union so intimate that they are, in reality, brought very close together in the minute conditions of pathologic change. Blood, bloodvessels, lymphatics, connective tissue, and nerves are common to all organs which take part in the more active processes of life, and in each of them there is in progress that common nutrition-change which is the necessary condition of all functional activity. Although, therefore, the function of the stomach may be that of exerting a particular effect on food, it must not be forgotten that the stomach has nerves, vessels, and connective tissues, and that diseased processes, exhibiting themselves mainly in its disordered functions, may be the expression of something wrong in innervation, in blood supply, or in general nutrition. And, again, although the brain is the organ which ministers to the higher functions of the nervous system, and records its diseases in changes of mind, sensation, or motility, it must be remembered that the brain has vessels which undergo nutrition-changes of degeneration or decay, and that many of its so-called special diseases are often but the outcome of a more general mal-

nutrition, which may have had its starting-point in heart, arteries, or veins. While, then, we may speak of "softening of the brain" among the diseases of the "nervous system," it would be quite as correct, in many instances, to place it among the effects of disease or degeneration in the "vascular system."

All the more important organs of the body are so complex in their structure, and all have so much in common, that we are bound to admit that the lines we draw between them, in regard to pathologic change, are often determined rather by the consideration of their practical utility than by the fact of their scientific accuracy. Knowing, however, the inherent difficulties of the case—viz., those which arise out of the fact that all the systems of organs have marvellously close relations with each other—and being aware, moreover, of those accidental hindrances which arise out of either our own ignorance or want of tact in the application of such knowledge as we have attained, we still hold it to be desirable that, in this System of Medicine, we should maintain the distinction between "general" and "local" diseases, and that we should take as our basis for classification of the latter, the particular systems of organs. We believe this to be so, because the lines which are drawn in making these distinctions include groups of diseases, the individual members of each of which have *inter se* closer clinical relations than have those which belong to different, although contiguous groups; and because in the vast majority of cases it is comparatively easy, and sufficient for all the practical purposes of "classification," to say that a particular disease under consideration is "general" in its character, or is "partial," in the latter instance especially affecting this or that great system of organs, such as the nervous, the respiratory, or the digestive.

The principle of division thus established with regard to all diseases—viz., that of limitation or localization—is again applicable, as a means to be employed for the primary subdivision of the large group of diseases forming the first section to be considered in Part II.; and accordingly "diseases of the nervous system" are distributed under two headings—the "general" and the "special" or "partial."

Under the former, the "general," are described those affections which exhibit their phenomena in all parts of the nervous system—those in which brain, spinal cord, and nerves seem to be *all*, more or less intensely, and more or less widely, involved; while under the latter, the "partial," are detailed those in which the brunt of the malady is borne by particular parts of the nervous system—the brain, the cord, the nerves, or their appendages.

It will be seen at once, by a reference to page 584, that under the former, the "general," are enumerated, together with those which strictly merit that designation, some diseases which appear to affect certain portions of the nervous system either more profoundly or more essentially than they influence others; and further that some morbid conditions are described which, although limited in the distribution of their symptoms, have as yet no such definite pathological anatomy that we can affirm, with anything like satisfaction, what part of the nervous system is in them primarily at fault. The first large group, therefore, is made to include, together with those in which the whole nervous apparatus is equally disturbed, some diseases apparently partial in their distribution, and others which, in the present state of science, are of "undetermined seat." Among the members of this first large group, for example, there are placed epilepsy, hysteria, and such diseases as show themselves in altered functions of the brain, cord, and nerves—occasionally one great division of the nervous system, and sometimes another, presenting the most marked derangement, but all divisions being more or less involved in morbid change of either function or structure, or both:—and in the same large group we find wasting palsy, chorea, writers' cramp, and allied affections, which, although they exhibit the maximum of their obvious symptoms in particular parts of the nervous system, are yet of such uncertain pathology, that it would be injudicious at present to describe them as diseases of either brain, cord, or nerves, exclusively.

So far as the pathology of these will allow, they will, when examined in detail, be referred to their proper places; but it is thought, for the simple purpose of arrangement, desirable to keep within the limits of ascertained facts and principles, by retaining them in the positions already described. The other alternative, that of placing them under particular headings, while it might confer a greater amount of apparent scientific precision, would, I believe, be attended by less real scientific accuracy; inasmuch as it would give an undue prominence to many hypotheses, very valuable in themselves, as forming the framework of both thought and investigation, but which, being as yet no more than hypotheses, are not entitled to hold high rank among the conditions upon which classification should be based. The diseases known, for example, as meningitis, myelitis, and neuritis, respectively, have clinical histories and well-known pathological conditions related to one another in a manner much more definitely ascertained than have such affections as ataxy, paralysis agitans, and wasting palsy. We have referred the symptoms

of the first series to their anatomical changes, whereas it cannot be said that we have done so with regard to the second group. Much more is known about the latter than was known a few years ago; but observations, during life and after death, have to be multiplied and verified before we can ascribe those diseases, with scientific precision, to particular localities and special kinds of structural lesion.

Again, the kind of distinction between hemorrhage into the corpus striatum and hemorrhage into the spinal cord, is, in its clinical relations, widely different from that which can be established between either of those two affections and ataxy, or chorea. In the one we are dealing with what is definitely known; in the other, with what is as yet indefinite, and only approaching scientific arrangement. Upon these grounds, therefore, the primary division is made into diseases of "general" distribution, or of undetermined locality, and "partial" diseases, or those having a recognized pathological anatomy.

In the second group of diseases of the nervous system—viz., those described as "partial"—the principle of arrangement is sufficiently obvious to need but little elucidation. In the first place, a subdivision is made upon simply anatomical grounds—viz., into affections of the cranium, the spinal column, and the nerves; and each of these is again subdivided upon an anatomical basis—diseases of the cranium being distributed under the categories of "meninges" and "nervous tissues," such as brain, cerebellum, and the like; while diseases of the spinal column are distributed in a similar manner. The next principle of division is that determined by the nature of the anatomical changes which these tissues, respectively, undergo. Here an attempt has been made to place in close proximity those affections which have the most highly-marked clinical similitude—an attempt, however, which is only carried to such a degree as shall not interfere with the more general arrangement.

It is not necessary to enter further into the detail of this classification, as the principles upon which it is based are sufficiently obvious for all the practical purposes of this System of Medicine.

DISEASES OF THE NERVOUS SYSTEM.

These diseases are divided into two large groups, viz., A, those of general distribution, or of uncertain seat; and B, those which are partial, and which occupy known relations to particular portions of the nervous system.

A.—General Nervous Diseases, and those of uncertain seat:—

Insanity.	Sunstroke.	Convulsions.
Hypochondriasis.	Alcoholism.	Epilepsy.
Hysteria.	Vertigo.	Locomotor Ataxy.[1]
Ecstasy.	Chorea.	Muscular Anæsthesia.
Catalepsy.	Paralysis Agitans.	Wasting Palsy.
Somnambulism.	Writers' Cramp.	Metallic Tremor.

INSANITY.

By Henry Maudsley, M.D., F.R.C.P.

Synonyms.—Insanity; Madness; Mental or Cerebro-Mental Disease; Mental Derangement; Mental Alienation; Mental Aberration; Unsoundness of Mind; Lunacy.

Definition.—So many and various in kind and degree are the forms of mental derangement included under Insanity, that it is not possible to give a definition of it that shall be at the same time comprehensive and exact. If the definition is wide enough to comprise all varieties, it will include eccentricities that fall short of disease; if exact enough to be definite, then it must exclude many cases of undoubted mental disease. As various as are the features or the voices of men, so various are the characters of their minds; and as no two persons are exactly alike in mental character and development, so in no two instances of the degeneration of mind do the morbid features correspond exactly. The development of other organs of the body, taking place before birth after a common type, is very much alike in different persons, and the diseases of them have a great resemblance; but the real development of the brain as the organ of conscious life, taking place after birth in relation to surrounding circumstances, and thus gradually issuing in the formation of individual character, is different in different persons, and accordingly cerebro-mental diseases present manifold varieties of features. As regards any particular case of Insanity which we may have to decide about, it is necessary then to fix attention on two points: first, on the change of individual character—the alteration from the former self; secondly, on the want of harmony, or the discord, between the individual and his surroundings. For although the morbid phenomena of the diseased mind witness in some measure to the degree of its previous development, yet the degeneration which disease implies must needs display itself in an alteration in the kind of manifestation of feeling, thinking, and acting—in other words, in a changed self; while again the import, as morbid, of the phenomena displayed can only be rightly weighed in relation to the individual

[1] Transferred to description of Spinal Cord.

sphere of life. It is, for example, quite possible, though apt to be forgotten in practice, that sentiments and acts which are habitual in the lowest strata of life may be sure signs of mental disease when uttered and done by one in a high social sphere.

Bearing in mind the difficulties inherent in the nature of the subject, which have led to every sort of definition by every writer who has not forborne the task in despair, I may declare Insanity to consist essentially in a *morbid derangement, generally chronic, of the supreme cerebral centres—the gray matter of the cerebral convolutions or the intellectorium commune—giving rise to perverted feeling, defective or erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations.*[1] We may safely go so far as to affirm the mind centres to be in the vesicular neurine of the convolutions, and Insanity to consist essentially in disorder, primary or secondary, of their functions —in disordered feeling, disordered intelligence, and disordered will.

This definition has the merit of fixing attention, first, on the reception of impressions from the external world—the mode of *feeling*, or the *affective* life; secondly, on the mental fashioning or elaboration of impressions—the modes of *ideation* or *intellection*, the *intellectual* life; and, thirdly, on the reaction of the individual on the external world—the mode of *action* or *conduct;* it answers also to the best psychological division of mind into *feeling*, *cognition*, and *will*. How desirable it is not entirely to overlook the social relations, will be plain when we reflect that it is in the irregularities of the individual, as an element in the social system, that the morbid character of Insanity fundamentally consists. Certainly the definition is far from being perfect, as in the nature of things must be the case so long as it is impossible to draw the line where sanity ends and Insanity begins, or even to say positively whether a particular person is insane or not; but against its manifest defects may be put its positive merits—namely, that it fixes the gray matter of the convolutions, the undoubted nerve-centres of intelligence, as the principal seat of morbid action in Insanity; that it distinctly declares that Insanity may be exhibited either in moral perversion only, or in the actions of the patient, or in delusion; and, lastly, that it sets forth how Insanity destroys the relations and responsibilities of the individual in the social system, making him very much like what a morbid element is in the organic system—something which cannot take its due place in the general harmony, and which must either be eliminated from it or sequestrated and rendered harmless in it. A man may certainly have disordered feeling, may think and judge erroneously, and act extravagantly, without being insane; but if he does so as a regular thing, and without any adequate cause in external circumstances—if he does so in fact by reason of a steadily acting internal cause, a derangement of his supreme cerebral centres—then he is insane. The standard by which to measure the perversion is, first, that of the *kind*—that which is fixed by the general consent of mankind; and, secondly, that of the *individual*—that which is justified by the degree of his previous mental development.

Many and varied as are the forms which madness takes, there are still beneath superficial differences certain characters of essential agreement; and accordingly genuine groups or types may be described, notwithstanding the fact that cases marking every grade of transition between one group and another are met with in practice. Of Insanity may still be said what Burton long ago said of it: "I could give instances of some that have had all three kinds *semel et simul*, and some successively. . . . . What physicians say of distinct diseases in their books, it much matters not, since that in their patients' bodies they are commonly mixed."

CLASSIFICATION. — The classification commonly adopted in this country, and yet indispensable for practical purposes, is a modification of that proposed by Esquirol, and is as follows:—

I. Mania . . . . . { Acute, or Raving Madness. Chronic. Recurrent.
II. Monomania.
III. Melancholia.
IV. Moral Insanity.
V. Dementia . . . . . { Primary. Secondary.
VI. Idiocy, including Imbecility.
VII. General Paralysis or Paresis.

[1] *Ideation*, now so commonly used, was first suggested and employed by Mr. James Mill in his "Analysis of the Human Mind." Dr. Darwin, in his "Zoonomia," aptly designates the common centres of intelligence as the *Intellectorium commune*.

In Germany, the classification which finds most favor stands thus :[1]—

I. Die Depressionzustände.
  1. Die Hypochondrie.
  2. Die Melancholie.
II. Die Exaltationzustände.
  1. Die Tobsucht.
  2. Der Wahnsinn.
III. Die psychischen Schwächezustände.
  1. Die Verrücktheit.
  2. Der Blödsinn.
  3. Idiotismus und Cretinismus.
IV. Der paralytische Blödsinn, Die allgemeine Paralysie der Irren.

I. Conditions of depression.
  1. Hypochondria.
  2. Melancholia.
II. Conditions of exaltation.
  1. Acute Mania.
  2. Monomania.
III. Conditions of mental weakness.
  1. Craziness or Incoherence.
  2. Dementia or Fatuity.
  3. Idiocy and Cretinism.
IV. Paralytic dementia, General Paralysis of the Insane.

It is easy to perceive the defects of such purely psychological classifications. They are vague and artificial, embracing in the same class forms of disease distinct enough to demand a separate description ; moreover, there are forms of mental disease which, presenting the characters of two or more of the different classes, might be placed in one or the other, or cannot be placed satisfactorily in either. Dr. Skae has proposed to classify all the varieties of Insanity in natural orders or families, grouping them in accordance with the *natural history* of each.[2] Why, he asks, should we attempt to group and classify them by the *mental* symptoms, and not, as we do in other diseases, by the *bodily disease* of which the mental perversions are but the signs ? In pursuance of this aim he has sketched the outlines of twenty-nine natural orders or families, having, as he believes, each its natural history, its special cause and morbid condition, a certain class of symptoms more or less peculiar to each, its average duration, and probable termination. They stand thus :—

Idiocy, Imbecility, } Moral and intellectual.
Insanity, with Epilepsy.
Insanity of Masturbation.
Insanity of Pubescence.
Satyriasis.
Nymphomania.
Hysterical Mania.
Amenorrhœal Mania.
Post-Connubial Mania.
Puerperal Mania.
Mania of Pregnancy.
Mania of Lactation.
Climacteric Mania.
Ovario-Mania (Utero-Mania).
Senile Mania.
Phthisical Mania.
Metastatic Mania.
Traumatic Mania.
Syphilitic Mania.
Delirium Tremens.
Dipsomania.
Mania of Alcoholism.
Post-Febrile Mania.
Mania of Oxaluria and Phosphaluria.
General Paralysis, with Insanity.
Epidemic Mania.
Idiopathic Mania, { Sthenic. Asthenic.

M. Morel, of Rouen, has propounded a classification of mental diseases according to their apparent causes—an *etiological* classification. He makes six principal groups, each of which has two or three classes under it: the *first* group being that of hereditary Insanity ; the *second* consisting of Insanity produced by *toxic* influences ; the *third*, of Insanity produced by the transformation of other nervous diseases, such as hysteria, epilepsy, hypochondria ; the *fourth*, of idiopathic Insanity; the *fifth*, of sympathetic Insanity; and the *sixth*, including all cases of dementia.

[What may be called a *physiological* classification has met with favor on the part of some modern authors, as follows : 1. *Sensorial* Insanity ; in which hallucinations of the senses are mistaken for realities, and so mislead (*alienate*) their subject, by getting him out of normal relation to the world around him. 2. *Intellectual* Insanity ; characterized by delusions, or confusion of the reasoning powers, upon one, many, or all subjects. 3. *Emotional* Insanity ; commonly called Moral Insanity, involving the affections, or propensities, with morbid impulses too strong for the will to control. The term Impulsive Insanity is sometimes applied to the same group of cases. Either of the

[1] Die Pathologie und Therapie der psychischen Krankheiten. Von Dr. W. Griesinger. Zweite Auflage. 1861.—Die Pathologie und Therapie der psychischen Krankheiten. Von Dr. M. Leidesdorf. Zweite Auflage. 1865.

[2] On the Classification of the Various Forms of Insanity on a Rational and Practical Basis, by David Skae, M.D.; Journal of Mental Science, October, 1863. For a fuller account and a criticism of this classification, I may refer to my work on "The Physiology and Pathology of Mind," Second Edition.

above forms may be acute or chronic; and they may be, and in most cases are, especially the last two, combined together.

Dementia, upon such a scheme, would be retained to indicate a failure or wreck of all, or nearly all, the psychical powers.—H.]

Adopting for the purposes of description the classification in common use, artificial as it is, it will be most convenient to describe the special features of the different varieties of mental disease in the course of the account of its causation, symptomatology, and treatment.

CAUSES. — These are usually divided into *physical* and *moral*, though without any exactness in such discrimination being really practicable. Two persons are exposed to like severe mental trials; one of them becomes insane, the other does not. Has the madness, then, been produced by a moral cause? In the former case, there was probably some innate vice of nervous element—some predisposition of it to disease, or some accidental nervous depression, by reason of physical disease, or other cause, whereby Insanity has been produced by a moral cause that has had no such ill effect in the latter case. The entire causes have not, then, been in reality the same. What should ever be borne in mind is, that all the conditions which conspire to the production of an effect are alike causes, alike agents, and that there is, in most cases of Insanity, a concurrence of conditions, not one single effective cause. Mental alienation often appears as the natural issue of all precedent conditions of life, mental and bodily —the outcome of the individual character as affected by certain circumstances; in such case, the germs of disease may have been latent in the foundations of the character, and the final outbreak is but the explosion of a long train of antecedent preparations. In vain, then, is it to try to fix always upon a single cause, moral or physical; a common mistake on the part of those who think to do so being to fasten upon that which is in reality an early symptom as the supposed cause. On this rock have hitherto foundered all etiological classifications of Insanity. It will be most convenient to set forth certain general considerations respecting sex, age, and the like, and then to proceed to treat of the proximate or exciting causes of Insanity. It is obviously unscientific to enumerate sex and age as causes of Insanity: no one goes mad because he or she happens to be a man or woman; but because to each sex, and at certain ages, there occur physiological changes that are apt to run into pathological effects in those who are predisposed to nervous derangement.

(*a*) *General Considerations.*—There are general causes, such as the climate of a country, the form of its government and its religion, the state of its civilization, the occupation and habits of its inhabitants, which work together in the course of generations to the formation of a national type of character, wherein there may be greater or less proneness to Insanity. Reliable data respecting the frequency of Insanity in different countries are, unfortunately, still wanting, and even the question whether it has increased with modern civilization has not been positively settled. Travellers certainly agree that it is a rare disease among barbarous people; whilst in the different civilized nations of the world there is, so far as can be ascertained, an average of one insane person in 500 inhabitants. The undoubted steady increase, again, of the insane under care and observation, would seem to be greater than can be fairly accounted for by the greater attention now given to their welfare: while theoretical considerations indicate that the feverish activity of life, the numerous passions and the great strain of mental work incident to the multiplied industries and eager competition of an active civilization, cannot fail to augment the liability to mental disease. Though not yet exactly provable by statistics, there is still some reason to believe that, with the progress of mental development through the ages, there is a correlative degeneration going on, and that Insanity is a penalty which our present civilization necessarily pays.[1]

Sex.—Though Esquirol and Haslam thought Insanity to be a little more frequent among women than among men, it is now generally agreed that the converse is true. Dr. Thurnam affirms men to be more liable to mental disorders than women; and Dr. Jarvis came to the same conclusion from the examination of the statistics of different countries. Recently, however, it has been stated that the female sex is more liable to suffer from hereditary Insanity.[2] Pregnancy, the puerperal state, the catamenial functions, and the climacteric change are conditions in women that will favor the disturbance

[1] In 1859 the total number of lunatics in England and Wales was 36,762; in 1869 it had increased to 53,177. The proportion of lunatics to the population had risen from 1 in 536 to 1 in 411 of the population. In France the ratio of lunatics to the population was, in 1851, 1 to 796; and in 1861, 1 to 444. In both countries, however; it is certain that the main portion, if not all, of the increase has been due to the operation of the lunacy laws, by which more accurate registrations of the insane have been gradually brought about.

[2] Statistics of Insanity of the Crichton Roya' Institution, by H. G. Stewart, M.D.; Journ. Ment. Science, 1865. Also, Hereditary Insanity, by H. G. Stewart, M.D.; Journ. Ment. Science, 1864.

of the mental balance, especially where there is any predisposition thereto; but against these must be weighed the larger exposure of men to mental wear and tear in the competition of life, and their more frequent addiction to intemperance and other excesses. Women, too, very seldom suffer from general paralysis. On whichever side, male or female, the uncertain difference lies, it is probably inconsiderable.

Period of Life.—Insanity is rare before puberty, though every form of it, except general paralysis, may occur even so early in life; it is far more frequent between the ages of 16 and 25; but it is most frequent of all during the period of full mental and bodily development—from 25 to 45—when there is the widest exposure to its causes. The internal revolution which takes place in women at the climacteric period leads to many outbreaks of Insanity in them between 40 and 50. In men there appears to be a climacteric period between 50 and 60, when Insanity sometimes supervenes: an old man may be found to be keeping a mistress in secret, or to be making foolish proposals of marriage, when, forerunning complete dementia, sensual impulses, clothed in the morbid habit of delusion, mock the extinction of sexual function. In childhood and early life idiocy and imbecility, moral and intellectual, are most commonly met with; after puberty mania and, later on in life, melancholia; in old age senile insanity occurs; and general paralysis seldom before 30, the years between 30 and 50 being the favorite years of its attack.

Condition of Life.—The statistics hitherto collected in regard to this point are of little or no value. Whether a particular profession or trade favors the production of Insanity is generally a question of the habits incidental to its pursuit—whether those who follow it live soberly and temperately, or whether they are addicted to intemperance and riotous living. On the whole, however, those who work with the head are more liable to mental disease than those who work with the hand, and they are less liable to recover when once attacked. It is an unproved and indeed ill-founded assertion that governesses are the victims of Insanity in greater proportion than other persons. The statement has originated in the fact that a great number of governesses are received into Bethlehem Hospital, as many as 110 having been admitted in ten years. The reason of this is that Bethlehem is intended especially for persons of the class of governesses—those who are not paupers, but yet cannot pay for care and treatment.

Other things being equal, it is certain that Insanity is proportionately more frequent amongst the unmarried than the married.

Individual Predisposition.—The heritage which a man has from his parents may alone, or together with the circumstances of early education, give rise to an individual predisposition to mental derangement. Unquestionably some persons have what may be called the insane temperament—a certain *neurosis* or *diathesis*, easily prone to degenerate into actual disease; they feel impressions in a way which other people do not feel them, are disposed to sudden impulses of strange feelings and desires, to whimsical caprices of thought and eccentricities of action, and they not unfrequently carry in their countenance and bearing the marks of their evil heritage. They have what Willis long ago called the *diathesis spasmodica*, an irritable weakness of nervous constitution, in which, if there be not positive disease, there is the well-prepared ground of disease. Authors are not agreed as to the proportion of cases of Insanity in which positive hereditary taint is detectable: some, like Moreau,[1] putting it as high as nine-tenths; others, as low as one-tenth. The most careful researches fix the proportion as not lower than one-fourth, if not so high as one-half; and there can be no doubt that the tendency is to increase the proportion as investigation becomes more searching and exact. When a person cannot endure the ordinary trials of life, or a natural physiological function, such as the development of puberty, it is plain that there must be some native infirmity or instability of nerve element. It must be borne in mind that hereditary predisposition may be of every degree of intensity, so as, on the one hand, to conspire only with certain more or less powerful exciting causes, or, on the other hand, to suffice of itself to give rise to Insanity even amidst the most favorable external circumstances. Again, not Insanity only in the parents, but any form of nervous disease in them—epilepsy, alcoholism, hysteria, and even neuralgia—may predispose to Insanity in the offspring, as, conversely, Insanity in the parent may predispose to other kinds of nervous disease in the offspring. Procreation during the temporary insanity of drunkenness, and too much interbreeding in families, are both recognized cause of a predisposition to mental degeneration. Some, like Lugol and Schroeder van der Kolk, have maintained that scrofula of parents may generate a predisposition to Insanity in the children; and whether this be so or not, it can admit of no question that the undoubted transformation which diseases undergo through generations is a subject deserving of further and

[1] Psychologie Morbide dans ses Rapports avec la Philosophie de l'Histoire. Par Dr. J. Moreau.

more exact study.[1] Baillarger has proved, what Esquirol observed, that Insanity descends more often from the mother than the father, and from the mother to the daughters more often than to the sons. Children born before the outbreak of an attack are less likely to suffer than those born after an outbreak.

An injudicious *education* may aggravate an inherent mischief; the parent not only transmitting a taint or vice of nature to the child, but fostering its increase by the influence of a bad example, and by a foolish training at that period when the young mind is very susceptible, and the direction given to its development decisive for life. Where there is no innate taint, mischief may still be wrought by enforcing an unnatural precocity, wherein is often planted the germ of future disease. Parental harshness and neglect, repressing the child's feelings, stifling its need of love, and driving it to a morbid self-brooding, or to take refuge in a world of vague fancies, is sometimes not less injurious than a foolish indulgence, through which it never learns the necessary lessons of renunciation and self-control. There can be no doubt that by the influence of good education and sound training a predisposition to Insanity might often be much neutralized and rendered almost harmless; but the mischief is that those who procreate children so afflicted are commonly least fit to train them well.

(*b*) *Exciting Causes.* — The so-called moral causes are generally, though not universally, held to be more frequent than the *physical* causes: Pinel thought them to be twice, Esquirol four times, as frequent; while Guislain attributed 66, Parchappe 67, out of 100 cases of Insanity to moral causes.[2] It is not the way of great intellectual exercise, when unaccompanied by emotion, to lead to mental derangement; mental exercise is favorable to length of days and health of mind; it is when the feelings are deeply engaged, when the mind is the theatre of great passions, that it is most moved and its stability most endangered. The depressing passions are most effective in this regard: grief, religious anxiety, disappointed affection or ambition, jealousy, the wounds of an exaggerated self-love, and the painful feeling of being unequal to responsibilities, or other such conditions of mental agitation and suffering, are most apt to reach a violence of action by which the balance is lost. It is especially when the individual has by a long concentration of thought, affection, and desire on a certain aim or object grown into definite relations with regard to it, and made it, as it were, a part of the inner life, that a sudden and entire change, shattering long-cherished hopes, is most likely to produce Insanity; for nothing is so fraught with danger to the stability of the strongest mind as a sudden great change in external circumstances without the inner life having been gradually adapted thereto. Hence, also, it is that a great exaltation of fortune, as well as a great affliction, rarely fails to affect for a time the strongest head and sometimes quite overturns a weak one; though the strong mind succeeds after a time in establishing an equilibrium between itself and its new surroundings, which the feeble mind cannot do. Men do not, however, often become insane from joy; and when one of the expansive passions, as ambition, religious exaltation, overweening vanity in any of its Protean forms, leads to mental derangement, it does not, like a painful passion, act either directly as the sudden cause of an outbreak, or indirectly by producing organic disorder and subsequent Insanity, but it exhibits its effects slowly, as a gradual development or exaggeration of a particular vice of character.

Among the causes of mental disturbance which it would be difficult to pronounce other than moral, but which are really due to physical conditions, are those incident to the great mental revolution produced by the development of the sexual system at puberty; when there occurs, as Goethe aptly expresses it, "an awakening of sensual impulses which clothe themselves in mental forms, of mental necessities which clothe themselves in sensual images." The great moral commotion produced at this period is the cause of an unstable equilibrium of mind, which is just as dangerous as if it were produced by some external cause; and which, if hereditary predisposition exist, may, without further auxiliary cause, issue in Insanity.

Of the *physical* causes of Insanity, intemperance occupies the first place; acting not only as a direct cause, but indirectly through the emotional agitation incident to an irregular life of dissipation and excess. Opium, Indian hemp, and other narcotics notably give rise to temporary disorder of mind, and, if abused by long indulgence, they may lead to permanent degeneration. Self-abuse in men is the cause of a particularly disagreeable form of Insanity, characterized by intense self-feeling and conceit, indolence and vacillation of character, and profound moral disturbance in the earlier stage, and later, by failure of intelligence, nocturnal hallu-

[1] Die Pathologie und Therapie der Geisteskrankheiten auf anatomisch-physiologischer Grundlage. Von J. L. C. Schroeder van der Kolk. 1863.

[2] Pinel, On Insanity, translated by Dr. Davis; Esquirol, Traité des Maladies Mentales; Guislain, Traité sur l'Aliénation Mentale; Parchappe, Traité de la Folie.

cinations, and suicidal or homicidal propensities. Epilepsy is sometimes followed by a most violent and dangerous mania, and, when of long standing, produces loss of memory and general failure of intelligence. Sometimes an outbreak of mania precedes or takes the place of an epileptic attack; and it may happen that a painful form of moral derangement, with periodical exacerbations—a masked epilepsy—precedes for months the appearance of the genuine epileptic convulsions.

In some instances hysteria produces or passes into Insanity. An attack of acute maniacal excitement, with great restlessness, rapid and disconnected, but not entirely incoherent, conversation, sometimes tending to the erotic or obscene, evidently without abolition of consciousness; laughing, singing or rhyming, and perverseness of conduct, which is still more or less coherent and seemingly wilful,—may occur in connection with, or instead of, the usual hysterical convulsions. Or the ordinary hysterical symptoms may pass by degrees into chronic Insanity. Loss of power of will is a characteristic symptom of hysteria in all its Protean forms, and with the perverted sensations and disordered movements there is always some degree of moral perversion. This increases until it swallows up the other symptoms: the patient loses more and more self-control, becoming capriciously fanciful about her health, imagining or feigning strange diseases, and keeping up the delusion or the imposture with a pertinacity that might seem incredible, and getting more and more indifferent to and impatient of the advice and interference of others. Outbursts of temper become almost outbreaks of mania, particularly at the menstrual periods. An erotic tinge is sometimes observable in the behavior.

More or less dulness of intelligence and apathy of movement, giving the seeming of a degree of imbecility, is common enough in chorea, and in some cases there is a violent delirium or mania; but besides these cases there are, I believe, in children others in which, without disorder of movements, there is a true choreic mania: it is an active delirium of ideas which is the counterpart of the usual delirium of movements, and its automatic character and marked incoherence are very striking; hallucinations of the special senses, and loss or a perversion of general sensibility usually accompanying delirium.

Chronic diseases, constitutional and local, favor the production of Insanity in many instances. Anæmia plays the same weighty part as in the causation of other nervous diseases. It is not without influence in many cases of hysterical insanity, as well as in the asthenic form which occurs during lactation; and when suddenly produced by great loss of blood, it may be the cause of an attack of puerperal mania. The syphilitic virus is now known to affect nervous element injuriously, and of late an extreme form of dementia has been ascribed to a syphilitic exudation, circumscribed or diffused, on the surface or within the substance of the brain. Tuberculosis is frequently associated with mental disease, one-fourth of the deaths in asylums being due to phthisis; and a form of suspicious melancholia, having something of the character of dementia about it, has been described as phthisical insanity.[1] The disappearance of a skin disease, or the suppression of an accustomed discharge, has of old been known to be at times followed by an attack of mania or melancholia; and there are on record numerous cases of mania which have been caused by retrocedent gout. Of local diseases favoring the production of Insanity, the influence of those of the heart seems to have been overrated; out of 602 post-mortem examinations made in the Vienna asylum, the heart was found to be affected in one-eighth, and in some of these only very slightly. Abdominal diseases are sometimes genuine causes of melancholia; and diseases of the sexual organs in women have always had a high place assigned to them in the scale of causes. It is certain that an attack of mania has followed the suppression of the menses, and that the return of menstruation is often followed by the recovery from Insanity; but it is certain also that outbreaks of maniacal fury, or of suicidal or homicidal violence, have coincided with the period of menstruation. Schroeder van der Kolk had a patient profoundly melancholic, who suffered also from prolapsus uteri, and in whom the melancholia disappeared directly the uterus was restored to its place. Flemming relates two similar cases in which the melancholia was cured by the use of a pessary, in one of them regularly returning whenever the pessary was removed; and I have seen, in one case, severe melancholia of two years' duration disappear after the cure of a prolapsus uteri. Instances are on record in which a woman has regularly become insane during each pregnancy; and, on the other hand, Guislain and Griesinger mention a case, respectively, in which Insanity disappeared during pregnancy, the patient at that time only being rational.

Under the name of Puerperal Insanity are frequently confounded three distinct varieties — the Insanity of Pregnancy, Puerperal Insanity proper, and the Insanity of Lactation. The first and last

[1] Tuberculosis and Insanity, by T. S. Clouston, M.D.; Journ. Ment. Science, April, 1863.

usually have the form of marked melancholia with suicidal tendency; the second appears as an acute and incoherent mania.

The Insanity which sometimes breaks out at the change of life in women is commonly a profound melancholia, with vague delusions of an extreme character.

After acute febrile diseases, as typhus and typhoid fever, the acute exanthemata, acute rheumatism, and pneumonia, Insanity sometimes follows. In such cases it either takes the form of acute dementia, or of the mild delirium of nervous exhaustion, from which recovery takes place in a few days; or it steadily passes into a chronic and persistent form, especially if there be hereditary taint; or it is acute, recovery taking place for a time, but, as happens after injuries to the head, being followed by subsequent marked change of temper, and finally Chronic Insanity.[1]

Injuries of the head, when not followed by any immediate ill consequences, may still, after a time, lead to incurable Insanity, through the degenerative changes which they induce in the cortical layers. Insolation notably acts perniciously on the cerebral centres, either by causing acute hyperæmia and œdema, or, as is more probable, by over-stimulation and consequent exhaustion of nervous element. Abscesses and tumors of the brain, cysticerci,[2] effusions of blood, do not directly produce mental disorder, which is indeed often absent; and when they do give rise to such disorder, they seem to act indirectly by a reflex or sympathetic action. Professor Gerhardt relates one case in which mental derangement was the first symptom of an embolism, the paralytic phenomena following later; and in a case recorded by L. Meyer, chronic tubercular meningitis gave rise to mental disorder.[3] Instances are on record in which Insanity, like tetanus, has been caused by peripheric injury of nerve; and of great interest are those cases, long since observed by Dr. Darwin, in which it occurs as the transference of disorder from the spinal centre.

Let it be distinctly understood, however, that of the above enumerated causes of Insanity, it scarcely ever happens that one acts singly; many of them would have no such ill effect, except through the co-operation of hereditary predisposition, and the latent hereditary taint might remain happily dormant, but for the concurrence of unfavorable conditions, physical or moral. Whenever such inborn taint does exist, it is certain that any great revolution in the system, whether arising out of external circumstances, or from internal causes, such as puberty, pregnancy, and the climacteric period, will be fraught with danger to the healthy balance of the mind.

[As a fair representation of American statistics in regard to Insanity, we may refer to those of the Pennsylvania Hospital for the Insane, in Philadelphia. Dr. Kirkbride, in his Annual Report published in 1876, states the supposed causes of 7167 cases of Insanity, treated in that Hospital during 35 years. To *ill health of various kinds*, 1290 cases are ascribed. To *intemperance*, 637 cases; *loss of property*, 230; *disappointed affections, domestic difficulties, grief, loss of friends*, &c., in all, 570 cases. *Mental anxiety* is credited with 441 cases; *intense study*, with 52; *intense application to business*, 56; *want of employment*, 40; *puerperal state*, 284; *masturbation*, 93; *exposure to the sun, or other intense heat*, 72; *opium*, 27; *tobacco*, 17 cases.

Of the whole number thus reported upon (7167 patients), there were admitted between the ages of 20 and 30 years, 2080 cases; between 30 and 40 years, 1951 cases; in each of the other decades of life smaller numbers.—H.]

FORMS OF INSANITY AND THEIR SYMPTOMATOLOGY.—A glance at the symptoms of the various forms of mental disease reveals at once the existence of two well-marked groups: one of these including all those cases in which the mode of *feeling* or the *effective* life is chiefly or solely perverted—in which the whole habit or manner of feeling, the mode in which the individual is effected by events, is entirely changed; the other, those cases in which *ideational* or *intellectual* derangement predominates. More closely scanning the symptoms it is seen that the affective disorder is the fundamental fact; that in the great majority of cases it precedes intellectual disorder; that it co-exists with the latter during its course; and that it frequently persists for a time after this has disappeared. Esquirol rightly, then, declared "moral alienation to be the proper characteristic of mental derangement." "There are madmen," he says, "in whom

[1] De la Folie Consecutive aux Maladies Aigues. Par le Dr. E. Muguier. Paris, 1865.—Griesinger, op. cit.—On the Delirium of Acute Insanity during the Decline of Acute Diseases, by Hermann Weber, M.D.; Med.-Chir. Trans., 1848.

[2] On Cysticerci, Archiv der Heilkunde, 1862, Prof. Griesinger. A case of insanity in which several cysticerci were found in the brain is related by Joire in the Gazette des Hôpitaux, 1860; another by Dr. Snell in the Zeitschrift für Psychiatrie, 1861; another by Baillarger in the Arch. Clin. des Maladies Mentales, 1860; and another by Dr. Saunders in the Report of the Devon County Asylum for 1864.

[3] Prof. Gerhardt, Wiener Med. Presse, No. 7, 1865; L. Meyer, Zeitschrift für Psychiatrie, 1858, p. 716.

it is difficult to find any trace of hallucination, but there are none in whom the passions and moral affections are not perverted and destroyed. I have in this particular met with no exceptions." This experience is in entire accord with that of every observer of Insanity, and with the principles of a sound psychology. It is the feelings that reveal the genuine nature of an individual; it is from their depths that the impulses of action come, while the intellect guides and controls; and accordingly in a perversion of the affective life is revealed a fundamental disorder of the innermost nature, a disorder which will be exhibited in acts, rather than, as ideational disorder is, in words. To insist upon the existence of a delusion as a criterion of Insanity, is to ignore some of the gravest and most dangerous forms of mental disease.

Melancholia.—Here the fundamental fact is a deep, painful feeling of profound depression and misery, a great mental suffering. The patient's feeling of external objects and events is perverted, so that he complains of being strangely and unnaturally changed: impressions which should rightly be agreeable, or only indifferent, are felt as painful; friends and relatives are regarded with sorrow or aversion, and their attentions with suspicion; he feels himself entirely isolated, and can take no interest in his affairs; and he either shuns society and seeks solitude, lying in bed and unwilling to exert himself, or he utters his agony in sounds ranging from the moan of dull ache to the shrill cry of anguish, or in ceaseless gestures of misery, or even in some convulsive act of desperate violence. All this while there may be no delusion; the patient may be conscious of the change in himself, may grieve over his unnatural state, and strive to hide or fitfully resist it; but as he gets worse he becomes more and more self-absorbed, more and more indifferent to, or distrustful of, those around him, and, finally, succumbs entirely to his affliction. Then it is, usually, that the vast and formless feeling of profound misery takes form as a concrete idea—in other words, is condensed into some definite delusion: this now being, as it were, the expression of it. The patient believes that he has committed some great crime, for which he must suffer death on the gallows; that he has blasted the happiness of his family; that he is possessed by the devil, or is the victim of a persistent and cruel persecution, by magic or by magnetism; that he has committed the unpardonable sin, and is for ever damned. The delusion is not the cause of the feeling of misery, but is engendered of it, and takes different forms according to the degree of the patient's culture, and the social, political, or religious ideas prevailing at the particular epoch: what the uneducated person attributes to witches or to devils, the man of some cultivation attributes to magnetism or to political conspiracy. In certain cases it is striking how disproportionate the delusion is to the extreme mental anguish—how inadequate it is as the expression of it: one, whose agony is that of the damned, will aver that it is because he has drunk a glass of beer which he should not have done, or because he has muttered a curse when he ought to have uttered a prayer. With him who believes that he is doomed to infinite and eternal misery, it is not the delusion, but the affective disorder, that is the fundamental fact; there can be no adequate or definite idea of the infinite or eternal, and the insane delusion of eternal damnation is but the vague and futile attempt at expressing an unutterable real suffering. It is noteworthy, again, how much the affliction of the melancholic subsides sometimes when a definite delusion is established: the vast feeling of vague misery which possessed the whole mind has undergone systematization in definite morbid action; and when the delusion is not active, but reposes in the background, not otherwise than as ideas constantly lie dormant in the sound mind, the patient may be tolerably cheerful. A suicidal feeling is so common that the possibility of its existence should always be had in mind; in 51 cases of Insanity in which suicide had been meditated or attempted, 28 were cases of melancholia.

As many as are the varieties of mental pain or suffering, so many varieties are there of melancholia; the essential character of all of them being an oppression of the self, the weight of a great suffering, out of which springs the delusion of being overpowered by some external agency, demoniac or human, or of salvation lost through individual sins. The classification of melancholia according to the accidental character of the delusion is, therefore of little value. Two well-marked groups may be distinguished: the first, including those who have a definite delusion—*Lypemania;* the second, those who have no definite cause of terror, but display a fearful apprehension of everything possible and actual—*Pantophobia.*

Hypochondriacal Melancholia represents one of the mildest but most persistent forms of melancholic depression; the anxiety proceeding from an extravagant feeling of bodily disease and exaggerated notions of danger. The morbid feeling, which is not usually without some physical cause in the organism, may be general, or it may be confined to single anomalous sensations. The patient is anxious and depressed; he complains of anomalous

feelings, which he watches and analyzes very attentively; his heart flutters fearfully, a film passes over his eyes, and there are strange sensations in his head; he examines his pulse, tongue, and evacuations, and rarely fails to find something abnormal in all of them. He is commonly irresolute, sluggish, and indifferent to what is not related to the circle of his morbid ideas; but in some cases paroxysms of anguish and despair rise to such a height as to sweep away all power of self-control, and to issue in suicidal or homicidal violence. The intelligence, though generally sound in regard to all matters that are not overclouded by the morbid feelings, is still profoundly affected through these. Hence, though hypochondriacal melancholics do not often commit suicide or homicide, they may do both; a man in the Somerset Asylum, for example, cut into his belly with a piece of glass, and dragged out his small intestines, in order to let the wind out. The transition is indeed gradual from the less severe forms to those in which the anomalous sensations are not merely exaggerated and misinterpreted, but are referred to some absurdly unreal cause, as to the presence of a serpent in the stomach, or to a galvanization of the nerves, or even to those cases in which the patient supposes his legs to be glass, his body butter, or himself metamorphosed into a wolf. By this declension, hypochondriacal melancholy undoubtedly passes into true melancholia.

Climacteric Insanity usually takes the form of profound melancholia, with vague and vast delusions, as that the world is in flames, that it is turned upside down, that everything is changed, or that some very dreadful but undefined calamity has happened or is about to happen. The countenance has an expression of a vague terror and apprehension. In some cases short and transient paroxysms of excitement break the melancholy gloom; these usually occur at the menstrual periods, and may continue to do so for some time after the function has ceased.

In connection seemingly with the development of puberty, or at any rate soon afterwards, we sometimes meet with a fanciful and *quasi-hysterical melancholia* in girls, which is not very serious when it is properly treated. There are periods of depression and paroxysms of apparently causeless weeping, alternating with times of undue excitability, more especially at the menstrual periods; a disinclination is evinced to work, to rational amusement, to exertion of any kind; the conduct is capricious and soon becomes perverse and wilful; the natural affections seem to be blunted or abolished, the patient taking pleasure in distressing those whose feelings she would most consider if in health; and although there are no fixed delusions, there are unfounded suspicions or fears and changing morbid fancies. In some of these cases, when the disease has become chronic, delusions of sexual origin occur, and the patient, whose virginity is intact, imagines that she is pregnant or has had a baby.

The *Insanity of Pregnancy* is, as a rule, of a marked melancholic type, with suicidal tendency: a degree of mental weakness or apparent dementia being sometimes conjoined with it. Other cases, however, exhibit much moral perversion, perhaps an uncontrollable craving for stimulants, all of which we may perhaps regard as an exaggerated display of the fanciful cravings, the capriciousness and the morbid fears from which women suffer in the earlier months of pregnancy.

The *Insanity of Lactation* is an asthenic melancholia, often with determined suicidal tendency. The time of its occurrence seems to show that the longer the child is suckled, the greater is the liability to it.

Sensibility is commonly much affected in melancholia. There may be a general diminution of the sensibility of the skin, or a local complete loss thereof; and complaints of præcordial anguish and of strange abdominal sensations testify the perversion of organic sensibility. These anomalous sensations appear sometimes to have a relation to the confusion and anguish of mind not unlike that which the epileptic aura has to the epileptic fit. Illusions and hallucinations of the special senses are frequent: the patient seeing those round him as devils, or smelling a corpse in his room, or tasting poison in his food, or hearing voices which revile and accuse him, or which suggest impious thoughts and prompt to violent deeds—it may be to imitate Abraham, and sacrifice his child.[1]

The bodily nutrition usually shares in the general depression of tone, although it is sometimes remarkable, considering the great apparent suffering, how little it is affected. When it does suffer, digestion fails, and constipation is troublesome; the skin loses its freshness, becomes sallow, dry, and harsh; the temperature of

[1] If a person sees, hears, or otherwise perceives what has no existence external to his senses, he has a *hallucination;* if he sees, hears, or otherwise perceives that which has no such external existence as he perceives, or perceives it with erroneous form or qualities, he has an *illusion:* and if, though perceiving external objects as they really exist, he believes in the existence of such objects, or conceives such notions of the properties and relations of things, as are absurd to the common sense of mankind, he has an insane conception or a *delusion*—the ground of the falseness of conception being not error, but a morbid condition.

the body is lowered, and the extremities are cold; the respiration is slow, moaning, and interrupted by deep sighs; the pulse is feeble, sometimes very slow, and even intermittent; and menstruation is generally irregular or suppressed. Sleep is usually deficient, though patients are apt to assert that they do not sleep when they really do, so little do they feel refreshed by it. Refusal of food, which is common, and sometimes very persistent, may be due to other causes besides want of appetite: it may take place through a fear of being poisoned, or in consequence of a delusion that the intestines are sealed up, or in order to commit suicide by starvation, or in fancied obedience to a voice from heaven, or from sheer wilful obstinacy.

The behavior of the melancholic accords with, or fitly expresses, the character of his ideas and feelings; and three well-marked groups of melancholia may be made according to the different relations on the motor side:—

1. Melancholia with stupor, *M. attonita* is interesting because of its close resemblance to dementia, with which it has been confounded. The expression of the face is that of a vacant, self-absorbed amazement, or the fixed form of some painful passion; the patient, as if in a trance, or as one only partially awake, scarce seems to see or hear; there is partial or general insensibility of the skin; consciousness of time, place, and persons is lost, and the bodily wants and necessities are alike unheeded; the muscles are generally lax, or some of them are fixed in a cataleptic rigidity. The patient——who, statue-like, must usually be removed from place to place—is possessed with some terrible delusion, as that the whole world is in flames, or that he is standing on the edge of a sea of blood, and when he recovers his senses he is as one awakened out of a frightful dream. One lady under my care, who was in this state for two years, with the exception of an occasional break of lucidity for a few hours, and who ultimately recovered her senses quite suddenly, believed that every one who approached her, and even lifeless objects, were threatening to murder her. As may easily be imagined, it is not always possible to distinguish this condition from dementia: for as to live in one sensation would be equivalent to having no sensation at all, so for a mind to be entirely absorbed in one terrible delusion, to remain in one persistent state of morbid consciousness, is equivalent, for the time being, to there being no mind at all. As, however, recovery may take place rather suddenly, though it may sometimes last only for a few hours or days and then be followed by a complete relapse, it is plain that melancholia with stupor is different from the stupor of real dementia.

2. Melancholia is often accompanied with destructive impulses, to sudden acts of violence against self or against others. Suicidal impulse is very common amongst melancholics, some sincerely and bitterly grieving over the horrible propensity as the sole cause of their unhappiness: but what is very remarkable is the sudden manner in which patients usually calm are at times surprised and overpowered by a desperate impulse, and hurried into a convulsive act of violence. A quiet man, having the delusion that his soul was lost, who had been for months under my care, and of whom no one suspected any mischief, suddenly started out of bed one night, without any warning, and flung himself out of a window through which it would have been thought impossible that any man could get. He was possessed with terrible hallucinations, thought that the world was come to an end, and in a fearful state of writhing agony cried, "Let me go! let me go!" Like paroxysms recurred occasionally during the next few weeks, after which the man recovered. The time of waking from sleep is that at which the desperate impulse is most likely to arise, wherefore melancholics should never be left alone when getting up in the morning. In other cases the sudden act of violence may be directed against others; the patient injuring or killing some one by reason of a sudden hallucination, or in consequence of his anguish having reached such a height of unendurable agony as to abolish all self-control, and irresistibly to utter itself in convulsive violence, either against a fancied persecutor or a completely indifferent person (*Raptus melancholicus*). Of such are some homicidal lunatics. Others act in obedience to a delusion: an evil spirit instigates the demono-maniacs to desperate deeds in spite of the will; or its impulses intensify their misery and lead to determined suicidal attempts, in order to escape from the intolerable promptings. A melancholic mother has killed herself to escape the desperate impulse to kill her child. Nor is it inconsistent with insanity in such cases that the violent deed should have been planned with surprising cunning and effected with sustained ingenuity.

So far from the morbid impulse or act constituting the insanity, it is but the outward and visible sign or expression of a profound affective derangement, the tendency of which is to manifest itself, not as ideational insanity does, in words, but in acts, and which for this very reason is much more dangerous than ideational insanity: it is truly an affective insanity, one symptom of which is homicidal or suicidal impulse: the delusion, when there is one, and the homicidal act, are both symptoms of a deeper-lying disease; and

the morbid manifestation of one may be as little within control as that of the other, or as the suddenly arising hallucination is. In the one case the patient is the victim of a morbid idea; in the other, of a morbid movement—in both cases, of a convulsion more or less co-ordinated. Where the disease is less acute, it is the feeling of this affective perversion that sometimes drives the melancholic to commit murder in order to be hanged, or impels a mother to murder her children in order to send them from misery on earth to happiness in heaven. It admits of no question whatever, and should therefore be borne clearly in mind, that the calmest melancholic is liable to occasional unaccountable exacerbations of disease, during the paroxysms of which he may perpetrate violence against himself or others; a wonderful relief, and even an apparent sanity, with endeavor to escape penal consequences, sometimes following the accomplishment of the act.

3. There is a melancholia of acute character, with great excitement and restlessness, that may even pass into mania. It is certain that cases marking every step of the transition to mania do occur in practice: and it is not always easy, notwithstanding the painful character of the delusion, to distinguish excited melancholia from mania: there are truly melancholics who are maniacal, as there are maniacal patients who are melancholic. The more activity of movement there is as the expression of the mental suffering, the more acute the utterance of the agony in gesture-language—in the wringing of the hands and the writhing of the body—the nearer does the case approach mania. The manifestations of excitement are, however, generally of a more uniform character than those of mania, and often even monotonous.

The *course* of melancholia is generally chronic; remissions are common, but complete intermissions rare. Still, it is striking sometimes how suddenly a great change may take place: Griesinger mentions a case of deep melancholia in which there occurred a perfect lucid interval for the space of a quarter of an hour; and I have more than once seen a profound melancholic awake in the morning cheerful and seemingly quite well, remain so for the rest of the day, and yet be as bad as ever on the following day. Such sudden recoveries are, like sudden conversions, greatly to be distrusted. Still, I have met with two instances in which sudden recoveries were permanent: in one, the patient, who had been acutely melancholic for six months, recovered suddenly after a flood of tears; in the other, the patient was quite well in the morning, after a sleepless night of much mental anguish and excitement. When recovery really takes place, it is usually gradual, and from within four to twelve months from the commencement of the disease; it is rare, but not impossible, after a year, although it may occur now and then after several years, especially if some great shock has aroused the patient to exertion. Half, or even more than half, of the cases of melancholia get well under proper treatment; and of those which do not recover, about half decline into mental weakness or complete dementia—the rest remaining chronic or ending in death. Death may be caused directly by the exhaustion of excitement or refusal of food, or it may be due to intercurrent diseases, phthiscal, cardiac, or abdominal. Gangrene of the lung was found by Guislain most frequently in melancholics who had died after long refusal of food.

Mania.—In this form of mental disease there is an excitement or exaltation of the self-feeling of the individual, the expression of which takes place either in the movements and conduct or in the character of the thoughts. Accordingly, two groups of cases may be broadly distinguished, although they pass insensibly into one another and are not unfrequently mixed; the first including all those cases of acute mania or maniacal fury in which the madness is mainly manifest in the actions of the patient, who sings, dances, declaims, runs about, pulls off his clothes, and in all ways acts most extravagantly; the second group including those more chronic cases in which the derangement is expressed in the ideas, is systematized in definite delusions—in which, therefore, the morbid action has taken deeper hold of the individual. The first group corresponds in the main to *acute mania*, the second to *monomania*.

It was held by Guislain that a stage of melancholic depression, of greater or less duration, almost invariably precedes an attack of mania; and there can be no doubt that this sequence is traceable in many cases. But it is not so in every case, as some have maintained. What has been commonly overlooked is, that there is not only an affective disorder of a depressed or melancholic kind, but that there is also an affective Insanity which is rather of an excited, expansive, or maniacal kind—a deep derangement of the affective life, in which the individual's self-feeling is greatly exaggerated or morbidly exalted, without positive intellectual alienation. It is a maniacal disorder, so to speak, of the feelings, sentiments, and acts, without delirium; and it is expressed, as the corresponding affective melancholia is, not in delusion, but in the conduct of the patient. As it is from the affective life that the impulses of

action come, while the function of the intellect is to guide and control, it is in strict accordance with reason that, when there is affective derangement or perversion of the mental tone, the morbid impulses that arise should be beyond control or guidance of the will, just as the convulsion of a limb is beyond control when there is derangement of the tone of the spinal centres. This inceptive maniacal state, which may unquestionably be primary, though usually following that of melancholic depression, is displayed in a great change of moral character; the parsimonious becomes extravagant, the modest man presumptuous and exacting, and the affectionate parent indifferent to his family; there is an extreme liveliness of manner, or a restless and busy activity, as of one half intoxicated; an overweening self-esteem is a marked feature, and an extravagant expenditure of money or an excessive sexual indulgence is common. Or the *exaltation* may be less and the *perversion* of the affective life more marked; in other words, the moral *alienation* more extreme, as witnessed in the profound moral derangement which sometimes precedes a series of epileptic fits, or takes the place of an epileptic fit, and in most of those cases included by Pinel under *mania sine delirio* and by Prichard under *moral insanity*. In such cases, as with the cases of so-called irresistible homicidal or suicidal impulse, it has been too much the practice to fix attention exclusively upon the extravagant actions of the patient, to the neglect of the profound affective derangement out of which his acts spring; so that they have been set apart as special and their real relations overlooked. They are truly of the same nature as that maniacal perversion of the whole manner of feeling sometimes forerunning an outbreak of mania; and their morbid expressions in single acts of vicious or violent conduct are of the same kind as those general symptoms of acute mania which are exhibited in the movements or actions of the patients.

Acute Mania; Maniacal Fury or Frenzy; or Raving Madness.—It seldom breaks out without a preceding stage of affective derangement, the period of incubation being usually of a melancholic character, as though there were a painful forefeeling of the coming storm. After a shorter or longer feeling of such premonitory depression there follows a marked change in the inclinations, habits, and affections: the patient, "much, much different from the man he was," gets restless, and is prone to wander or travel about, is sleepless at night, or is tormented with very vivid dreams; he next becomes lively and excitable, as though half intoxicated, and the tone of his voice is sometimes strangely altered; his actions are restless, extravagant, and turbulent; and all the while he thinks himself wonderfully well, and scorns the suggestion of medical aid. As matters become worse there is an irresistible propensity to utter the internal commotion in outward gestures, acts, or words: the patient sings, dances, declaims, shouts, and laughs; or he is industriously occupied in restless and aimless work, as in polishing the floor with his saliva, in tearing his clothes to shreds, or in changing the place of every piece of furniture about him; or he explodes in furious outbreaks of rage and raving of word and action. The organic appetites or instincts come markedly into the foreground, the veil of reason being withdrawn: the appetite is ravenous and indiscriminate, garbage, or even excrement, being devoured with apparent avidity; and the patient, forgetful of decency, and abandoned to the promptings of the sexual impulse, sometimes masturbates as the monkey does, without shame or restraint. Withal there is often a certain consciousness of his state, so that he may restrain himself and seem reasonable for a time, and when seemingly at his worst he will sometimes yield to the show of energy and determination. The mood of mind may be brisk and humorous, or bitter, angry, and scornful in the face of opposition. There is no fixed delusion, nor any fixed group of delusions; but the ideas are rapid, confused, and transitory, and appear as fleeting delusions, or immediately utter themselves in automatic impulses to words and actions; the idea of an act, the moment it arises in the mind, becomes the act. Because of the rapidity of the flow of ideas in the early stages, the witty observations, acute comparisons, and fluent rhymes then sometimes made, it has been said that there is an increase of mental power. But it is only the semblance of an increase: though there is a lively revival of the past with great vivacity of expression, there is no due assimilation of the present, but an incapacity to perceive rightly the relations of things around, together with false judgment with regard to them, so that the unhappy sufferer is extravagantly joyous in a madhouse; there is an entire absence of that co-ordination of the feelings and ideas which marks the highest mental power and is the condition of true will. The lively flow of scarce coherent ideas marks the excitability of an irritable weakness, and is the forerunner of a restless succession of isolated ideas and fragmentary associations in the more advanced stages, not otherwise than as convulsion is the forerunner of paralysis.

It is striking how complete in some cases may be, during the attack, the memory of the past, and after the attack,

of all that has happened during it; whilst, in other instances, the patient will forget altogether the events of his madness, like as a dream is forgotten, though he may remember them again during a subsequent attack. It may happen also that, immediately before a second attack, thoughts and feelings displayed on the occasion of a first attack, but latent since, will recur, so that even attendants recognize the evil presages, and can predict the outbreak.

Hallucinations of the different senses are common in mania, and illusions still more so. In 178 out of 229 cases Brierre de Boismont professes to have met with such complications; they are generally fleeting, like the other morbid phenomena. Some have thought that the long endurance of the great expenditure of energy in acute mania is owing to a perversion of the muscular sense, by reason of which the true condition of the muscles is not declared. There can be little doubt that illusions of the muscular sense are at the root of the delusions with regard to bodily movements sometimes exhibited in mania; when a person lying in bed believes his limbs or himself to be flying through the air, it is certain that the muscular sense does not give correct information, but is affected with hallucinations.

The bodily functions often bear the great mental agitation of acute mania in a surprising manner. The pulse may be a little quicker in the early stage, when there is perhaps some febrile disturbance; but it is afterwards scarce raised in frequency. The temperature of the body is only slightly increased; but in cases of a typhoid type, where there is sleeplessness, restlessness, gradual wasting, and where the tendency is to death from exhaustion, Dr. Saunders has found it to be often raised from three to five degrees above the natural standard.[1] In the Insanity occurring after acute diseases, Dr. Weber's observations show only a slight increase of temperature, although this had been raised several degrees during the previous disease, and immediately rose again on the occasion of a relapse.[2] The skin may be either dry and harsh, or moist and of offensive odor. Constipation is common, but in some cases there occurs a continued and obstinate relaxation of the bowels. The urine Dr. Sutherland found to contain an excess of phosphates in acute mania; and if this were true, it would testify, like the increase of temperature, to an abnormal disintegration of tissue. More recent examinations of the urine, by Dr. Addison, result in the assertion that "the quantities of the urine, of the chloride of sodium, of the urea, phosphoric and sulphuric acids, excreted during the course of a maniacal paroxysm, occurring in acute mania, epilepsy, general paralysis, melancholia, or dementia, are less than the amounts excreted in an equal time during health."[1]

The *course* of mania is not often regularly progressive; there are generally remissions, and sometimes complete intermissions, or even so-called lucid intervals. The attacks may return at regular or irregular intervals, and thus constitute a periodic or recurrent mania; or attacks of melancholia may alternate with them, and give rise to what the French have described as *folie circulaire*, or *folie à double forme*. The duration of an attack of mania may be for hours or months, and recovery may be sudden or gradual. There can be no question of the occasional occurrence of a short maniacal fury, a *furor transitorius*, lasting for a few hours or days, usually associated with vivid hallucinations, and comparable to an attack of epilepsy,[2] and it is interesting to observe that these attacks are sometimes preceded by a strange anomalous sensation rising, like an epileptic aura, from some part of the body to the brain. When recovery takes place, it is usually within the year; it is rare after two years; and, indeed, the longer the disease lasts the worse is the prognosis, which is always unfavorable in recurrent mania and in mania alternating with melancholia. Recovery not taking place, the disease passes into chronic mania, or into dementia, or ends fatally. Death may be due to exhaustion, or to some intercurrent disease, such as pleurisy or pneumonia. When maniacal exhaustion proves fatal, the end may be very sudden and unexpected, so as to leave in the mind an anxious feeling of doubt whether a more energetic treatment might not have prevented death, or, if energetic treatment has been employed, whether that has not had something to do with hastening the fatal issue.

I have described the general features of the typical form of acute mania, but we

[1] Report of the Devon County Asylum for 1864.—Dr. Clouston has recently made some careful researches respecting the temperature of the insane. He finds the evening temperature of every form of insanity to be higher than the evening temperature of health, and excitement to be almost always attended by an increased temperature. (Journ. Ment. Science, April, 1868.)

[2] On the Delirium of Acute Insanity during the Decline of Acute Diseases; Med.-Chir. Trans. vol. xlviii.

[1] On the Urine of the Insane, by A. Addison, M.D.; British and Foreign Med.-Chir. Review, 1865.

[2] Ueber Mania Transitoria, von Dr. L. Meyer. Virchow's Archiv, Band viii. Die Lehre von der Mania Transitoria, von Dr. R. Krafft-Ebing. Erlangen, 1865.

meet with several varieties in practice. There is a form of very acute mania, which might properly be called an *acute delirious mania* or *acute maniacal delirium*. It is characterized by intense excitement, great restlessness and jactitation, entire incoherence, there not being the coherence of distinct and enduring delusion, and by only the briefest flash of momentary consciousness of what is going on around, or by apparent unconsciousness, except so far as fragments of impressions are caught up, whirled and lost in the agitation of delirium. It runs a rapid course, very often to exhaustion and death, the pulse becoming quick and feeble, the skin hot, and the excitement and restlessness continuing to the last.

[What is called by American writers upon psychopathology "Bell's Disease," from its especially careful study by Dr. Luther V. Bell, is thus described by Dr. Curwen, of the Penna. State Hospital for the Insane, at Harrisburg:[1] "In this there is excessive restlessness, incessant loquacity, the most remarkable incoherence of thought and expression (tireless babbling as it is expressed by the French), the pulse is rapid, weak, and very compressible, so as almost to be stopped, the skin is cool and also the scalp, and as a general rule dry, except after violent exertion, and often even then, the tongue and mouth very dry from the incessant talking, and the attention can scarcely be attracted long enough to obtain an answer to any question. In ordinary cases of acute mania, or in inflammatory disorders, the attention may be arrested so long as to obtain answers to questions, or to change the current of thought, but in this it seems as if the individual was so impelled to give utterance to the words that crowd his mind that he had not time to stop for any purpose. The resemblance of this condition to the symptoms of acute meningitis or cerebritis as laid down in the books will mislead any one who will not give earnest heed to its peculiar diagnostic symptoms, the freedom from all feverish heat, the peculiar weakness and softness of the pulse, and the physical state of depression of the whole system."—H.]

Puerperal Mania comes on within one month of parturition, and like the Insanity of pregnancy, occurs most often in primiparæ. It is of an acute and incoherent character, marked by noisy restlessness, sleeplessness, tearing of clothes, hallucinations, and in some cases by great salacity, which is probably the direct mental effect of the irritation of the generative organs. Suicide may be attempted in an excited purposeless way. The bodily symptoms, contradicting the violence of the mental excitement, indicate feebleness; the features are pinched, the skin is pale, cold, and clammy, and the pulse is quick, small, and irritable. Recovery takes place in three out of four cases of puerperal mania, usually in a few weeks; the patient, after the acute symptoms have subsided, sinking into a temporary state of confusion and feebleness of mind, and then waking up as from a dream.

Recurrent Mania reminds us of nothing so much as epilepsy in the regularity of its recurrence, in the uniformity of the symptoms of the attack, each being almost an exact image of the other, in its comparatively short duration, and in the temporary recovery. The patient becomes elated, hilarious, talkative, passing soon into a state of acute, noisy, and self-conscious mania, which may last for two or three weeks or longer, and then sinking into a brief stage of more or less depression or confusion of mind, whence he awakens to calmness and lucidity. In vain we flatter ourselves with the hope of recovery: after an interval of perfect lucidity, of varying duration in different cases, the attack recurs, goes through the same stages, and ends in the same way, only to be followed by other attacks, until at last the mind is weakened and there are no longer lucid intervals. Could we stop the attacks, mental power might still be regained by degrees, but we cannot; all the resources of our art fail to touch them, and I know no other form of Insanity which, while having so much the air of being curable, thus far defies all efforts to stay its course.

When the acute symptoms of mania have subsided, and the disease has become chronic, it presents most varied characters, according to its cause and the degree of mental degeneration. When there is considerable intellectual power apart from the delusions, as there usually is when the disease has been produced by moral causes, then the case may properly fall under monomania, or partial derangement of the faculties; when there is great loss of mental power together with delusions, as there often is when the disease has followed acute mania or a physical cause, then it may properly fall into one or other of the groups of dementia. On the one hand, then, chronic mania runs insensibly into monomania; on the other, into dementia. It is remarkable in some cases how much intellectual power may coexist with extravagant delusions: a person who fancies that not an event in Europe happens which has not some hidden relation to him, who detects a plot against himself in the meeting of a cabinet, or in the journey of an emperor to his country palace, may yet have an exact

[1] Transactions of Penna. State Medical Society, 1869.—H.]

knowledge of all his affairs, and be capable of giving a good opinion with regard to them. But what such a person cannot be depended on to do is to control his conduct.

The form of mental derangement produced by self-abuse—the *Insanity of masturbation*—furnishes a good example of a chronic mania in which there are no acute symptoms, the onset of the disease being most gradual. The patient becomes offensively egotistic; he is full of self-feeling and self-conceit; insensible to the claims of others upon him and of his duties to them; interested only in hypochondriacally watching his morbid sensations and feelings. His mental energy is sapped, and though he has extravagant pretensions, and often speaks of great projects engendered of his conceit, he never works systematically for any aim, but exhibits an incredible vacillation of conduct, and spends his days in indolent and self-suspicious self-brooding. His relatives he thinks hostile to him, because they do not show the interest in his sufferings which he craves, nor yield sufficiently to his pretensions. As matters get worse, the general suspicion of the hostility of people takes more definite form, and delusions spring up that persons speak offensively of him, or watch him in the street, or comment upon what passes in his mind, or play tricks upon him by electricity or mesmerism, or in some other mysterious way. Still he professes the most exalted moral or religious aims. A later and worse stage is one of moody self-absorption and of extreme loss of mental power. He is silent, or if he converses he discovers delusions of a suspicious or obscene character, the perverted sexual passion still giving the color to his thoughts. He dies at the last a miserable wreck. This is a form of insanity which has certainly its special exciting cause and its characteristic features; nevertheless, self-abuse seldom, if ever, produces it without the co-operation of the insane neurosis.

Monomania; Partial Mania; Partial Insanity; Delusional Insanity.—The exalted self-feeling which in acute mania uttered itself chiefly in turbulent action gets embodied in a fixed delusion, or in a group of delusions, which fails not still to testify the overweening self-esteem. As in melancholia the feeling of oppression of self was condensed into a delusion of being possessed with a devil, or otherwise afflicted, so here the exaggerated self-feeling is clothed in a corresponding delusion of power or grandeur, and the personality of the patient is transformed accordingly: he would fain have us believe that he has resolved the most abstruse problems of science; that he has devised an infallible scheme for reforming the world; that he is king, prophet, or divine. Monomania is, then, a partial ideational insanity, with overweening estimate of self, and fixed delusion or delusions upon one subject or a few subjects, apart from which the patient reasons tolerably correctly. Pathologically it represents a systematization of the morbid action in the supreme cerebral centres—the establishment of a definite type of morbid nutrition in them. Having regard to the mode of origin of the delusion, the deep hold which the manner of its genesis proves it to have on the individual nature, it will be seen how erroneous it is to speak of the mind as sound apart from the delusion. As in melancholia so here, there is a fundamental affective disorder incapacitating the individual from a just appreciation of those things that really affect the self, that touch to the quick those genuine feelings revealing his innermost nature and instigating his conduct; and he is liable at any time to outbreaks of fury, which, like the delusion, are but expressions of the deep-rooted derangement. The mind is not unsound upon one point, but an unsound mind expresses itself in a particular morbid action. Patients thus suffering often seem calm and harmless enough under the regular discipline of an asylum; but if they are exposed to the excitement of ordinary life, seriously crossed in some project, or subjected to the stress of adverse events, they are liable to outbursts of uncontrollable rage or of true mania; so that one who may have been hitherto only interested by their harmless delusions, will be horrified at the utter madness which they exhibit.

The particular delusions of the monomaniac differ according to his occupation in life and the degree of his culture, and are frequently colored by the events, social, religious, or political, of the epoch: Esquirol boasted that he could write the history of the French Revolution from the character of the Insanity which accompanied its different phases. Hallucinations and illusions frequently accompany the delusions which they appear sometimes to generate and always to strengthen. The behavior of the patient accords with the character of his delusions: one makes sweeping plans and projects, enters upon vast speculations, and sometimes goes through an immense amount of patient and systematic work in perfecting some marvellous scientific invention; another reveals in gait and manner the exalted character of his delusion; and to a third, ordinary language does not suffice to express the magnificence of his ideas, and he invents new and mysterious signs, which, unintelligible to every one else, have wonderful significance for him.

The *course* of monomania, once established, is very seldom towards recovery; for as it is rarely primary, it represents a

further degeneration or more advanced morbid action than mania or melancholia, upon which it usually follows. Even when it is primary, the outlook is not much more favorable, for it is then commonly secondary to some fundamental vice of character. Certainly recovery may take place, and the patient awake to sense as out of a dream; and in some rare instances it has taken place after years, especially under the influence of the revolution in the system produced by some intercurrent disease or at the climacteric period. When recovery does not occur, the disease becomes more chronic, lasting as such, or passes into actual dementia; the more the exaggerated self-feeling which inspires the delusion wanes, and the more this, losing its inspiration, becomes a mere form of words, the nearer the case gets toward incoherent dementia. As a general rule, it may be said that recovery does not take place when a fixed delusion has lasted for more than half a year.

Dementia.—It is the natural termination of mental degeneration, whether going on in the individual or through generations; and it is accordingly in the great majority of cases chronic, and secondary to some other form of mental disease. Dementia may, however, be both acute and primary, and is then not always distinguished from melancholia with stupor.

Acute dementia sometimes occurs after an attempt at strangulation, after certain acute diseases, and after a series of severe epileptic fits; and in one case under my observation a masked epilepsy seemed to take this form. As a primary disease, it sometimes follows a sudden and severe moral shock, and is now and then met with in young men and women, obscurely connected apparently with the state of the sexual functions. Dr. Skae describes a sexual or post-connubial mania taking the form of acute dementia, met with both in the male and female sex, but more frequently in the latter, and connected, he believes, with the effect produced on the nervous system by sexual intercourse. With these exceptions, dementia is a chronic and secondary disease presenting every possible variety in the degree of mental decay.

After the disappearance of a severe attack of acute mania, the effects of the shock are oftentimes visible in a certain condition of mental weakness without actual intellectual disorder: the force of character seems to have been sapped, and the finer moral and æsthetic feelings, which are, as it were, the bloom of culture, are abolished; the physiognomy has lost its highest expression, and the individual produces the impression of a certain childishness. This is one end of the scale of degeneration; but at the other extremity mental power is almost obliterated, the acquisitions of the past being completely razed out, and no interest in the present possible, and the patient leads a mere vegetative life. Between these two extremes every sort of transition is met with in practice, so that it is impossible here to do more than indicate certain prominent types.

Most of the permanent residents in asylums consist of those who, after mania, monomania, and melancholia, have subsided into a chronic state of more or less feebleness and incoherence of mind—the *crazy* people, who represent the wrecks of these forms of mental disease. Some there are who exhibit a few striking delusions which seem to be automatically expressed; the strong self-feeling which underlies or inspires these in monomania has faded away, and they are no longer full of self-assertion, nor eager, earnest, and consistent in carrying out their plans. The old paths of associations are broken up, and memory is almost abolished; all liveliness of feeling is gone, and there is little or no interest in what is going on around; and the only momentary excitement which occurs is when fixed delusions are attacked. It is remarkable, however, how even in these cases the excitement of a fever will sometimes restore temporarily the functions of the mind which seemed to have gone forever. The countenance no longer expresses any fixed passion; there is a want of harmony, or, as it were, a dislocation of its features, and the most which it manifests is the shivered expression of a passion or the shattered wreck of a smile. There is a corresponding imbecility on the motor side: some can certainly continue their former occupation, or can do a little simple manual work, but there is no systematic correspondence of action to delusions, and there is not unfrequently a useless and busy industry in gathering stones, pieces of paper, and sticks. Strange propensities of all kinds are exhibited, as, for example, to stand or crouch in a particular corner, to walk backwards and forwards for a certain distance on a particular slip of ground, or to fantastically ornament the person with feathers or flowers. The mood may be of surly depression, or of more or less exaltation. Hallucinations and illusions of the extremest kind are frequent, and tend to sustain the delusions: one woman has in her belly the whole tribe of the apostles, prophets, and martyrs; another lovingly nurses as her child a lump of wood decked in rags; a third person, whose singular movements seem unaccountable, is busy spinning threads out of sunbeams; while a fourth continues the most violent move-

ments of his arms in order to prevent the motion of the universe or of his own blood from coming to a stand. The bodily health is usually good, the patient frequently getting stout as the active symptoms of mania or melancholia subside into the calm of dementia. This group represents for the most part dementia following on monomania.

In another group of cases there is greater external activity, with a more general incoherence or craziness. There are no fixed delusions, although there is evidence in the patient's incoherent expressions, or in his senseless, parrot-like repetition of certain words, of the wreck of such as existed in the maniacal stage. The senses are open to the reception of impressions, but these do not seem to be further fashioned into ideas. There is sometimes entire indifference to surroundings, together with great insensibility to pain; or there are short outbreaks of incoherent passion or fury; or there may be desperate and unaccountable homicidal violence. The predominant mood is different in different cases: some are gay, happy, and prone to laugh and chatter; others are gloomy, weep, and display the mimicry of sorrow; while others again are malicious, spiteful, and addicted to a purposeless mischief with a monkey-like cunning and persistence. The loss of memory is marked: some have entirely forgotten their former life and their own names, while others, who perhaps forget instantly the last thing said, can reproduce the distant past with fidelity. In the movements of some there is marked feebleness, or the indication of commencing paralysis; while others are restless, agitated, and run about with ceaseless activity. The bodily health is usually good: they sleep well, and eat well, often gluttonously and without discrimination, and are sometimes prone to get fat, until an attack of excitement and agitation, to which some are periodically liable, reduces them. The physiognomy is blank and expressionless, especially so when the patient is addressed; it is also prematurely aged.

Lastly, there is a group of demented patients, in whom the mind is almost extinguished; who must be fed, clothed, and cared for; who manifest little or no sensibility; whose only utterance is a grunt or a cry; and whose only movements are to rub their heads or hands. Their existence is little more than organic, and if not carried off by pneumonia, tubercle, or some other disease, as they frequently are, they die from effusion on the brain, serous or hemorrhagic, or from atrophy of the brain, or from the effects of accident, to which, through their apathetic helplessness, they are much exposed.

Senile dementia is characterized by weakness of mind, inability to grasp the present, and great loss of memory, especially of recent events; the patient talking of events long past as if they had just happened, perhaps believing himself to be in daily intercourse with persons who have long been dead, and confounding his present life, the events of which are almost immediately forgotten, with his past life.

The *course* of secondary dementia is from bad to worse: it is impossible that recovery should take place, although the condition and habits of a patient may be much improved by proper care. Those who suffer from acute primary dementia get well generally; but, of course, senile dementia, though primary, is beyond the reach of remedy. Death may be due to effusion on the brain, or to atrophy of it, or it is produced by accidental disease, as tubercle or pneumonia. Dementia is the only form of Insanity in which the average temperature is below that of health.

Moral Insanity.—Under this unfortunate name,[1] Dr. Prichard described cases of real mental disorder, in which, without hallucination, illusion, or delusion, the derangement is exhibited in a perverted state of those mental faculties which are called the active and moral powers—the feelings, affections, propensities, and conduct. He never meant that a vicious act or crime, however extreme, should be deemed proof of moral insanity; for he expressly insists on tracing the disorder to certain recognizable causes of disease. "There is often," he says, "a strong hereditary tendency to Insanity; the individual has previously suffered from an attack of madness of a decided character; there has been some great moral shock, as a loss of fortune; or there has been some severe physical shock, as an attack of paralysis or epilepsy, or some febrile or inflammatory disorder, which has produced a susceptible change in the habitual state of the constitution. In all these cases, there has been an alteration in the temper and habits."[2] In reality, however this moral insanity is no special form of disease, but a variety of that mental derangement already described as *affective* or *pathetic;* and briefly to enumerate the varieties of this affective form of derangement, all which were confounded by Pinel under *mania sine delirio*, will best exhibit the nature and relations of moral insanity.

(*a*) There are attacks of derangement in which the moral or affective alienation is very great, and in which the intellect

[1 *Impulsive* Insanity is reasonably preferred by some writers.—H.]

2 A Treatise on Insanity and other Disorders affecting the Mind. By J. C. Pritchard, M.D., F.R.S.

is only secondarily affected through the moral perversion, the patient reasoning very well from the premises of his perverted feeling; he has no delusion unless his whole manner of thought in reference to self be called a delusion. These attacks are often associated with epilepsy, which they may immediately precede, as they sometimes precede an outbreak of mania; or they may occur at periodical intervals for months before actual epilepsy, and sometimes take the place of the true epileptic seizure afterwards; or again, the epileptic fits may cease entirely, and be followed by such attacks of profound moral derangement, occurring at uncertain periods, and perhaps passing into dementia. It is important to bear in mind that when associated with the epileptic neurosis they represent a condition in which vivid hallucinations and irresistible impulses of a desperate kind, homicidal or suicidal, are apt to arise instantaneously; that they, in truth, embrace the most dangerous forms of the so-called impulsive insanity—the *monomania instinctive* of Esquirol.

(*b*) There is the melancholic depression of the affective life already described—simple melancholia, in which the anguish rises to such a pitch as to issue in an explosion of convulsive violence, homicidal or suicidal, no fixed delusion being present. Some of the cases of so-called impulsive insanity are examples of this form of disease.

(*c*) The moral insanity proper of Prichard (*monomanie raissonnante*, Esquirol) occurs in most instances as the result of hereditary taint, aggravated or not by unfavorable conditions of life. It is a more advanced stage of degeneration than that which has been described as the insane temperament, but it does not reach actual intellectual derangement: the moral feeling being the highest acquisition of human culture in the course of development through the ages, its loss is one of the earliest effects of degeneration. Moreover, it will always be necessary to consider the social condition of any one suspected to have moral insanity, inasmuch as it is in the loss of the social feeling by reason of disease that the alienation essentially consists. If a person in a good position, possessed of the feelings that belong to a certain social state, does, after a cause known to be capable of producing every kind of Insanity, undergo a great change of character, lose all good feelings, and from being truthful, temperate, and considerate, become a shameless liar, shamelessly vicious and brutally perverse, then it is impossible not to see the effects of the disease. Friends and relatives may remonstrate with such a one, and punishment may be allowed to have its due course; but in the end both friends and every one who has to do with him must confess that he is the victim of disease—that his proper place is not the prison, but the asylum. Such moral alienation may occur after previous attacks of Insanity, after acute fevers, after some form of brain disease, or after injury to the head. After an attack or two of melancholia with suicidal tendency, from which recovery has taken place, the patient is perhaps attacked with genuine moral insanity, which ultimately passes into intellectual disorder and dementia. Or there has been more or less congenital moral defect, and maniacal exacerbations of actual moral insanity, without positive intellectual disorder, take place, dementia following after a time; these outbreaks may occur at puberty, or at the menstrual periods. A moral insanity again is in some instances the first stage of mental degeneration that is produced by self-abuse or sexual excesses: it now and then occurs in consequence of a severe moral shock, as the forerunner of more marked Insanity; and it not unfrequently precedes general paralysis. If the evidence from its own nature and causation were insufficient, the simple fact that it is often the forerunner of the severest mental disease, might suffice to teach the right interpretation of moral insanity.

(*d*) There are certain beings who are truly moral imbeciles, the original defect being due, as in idiocy, to some cause acting either before birth, or during the first years of life. With such moral defect there is often associated more or less intellectual imbecility, though not always plainly so; it is remarkable indeed what an acute intellect may sometimes coexist with an entire absence of the moral sense. Some of the notorious gaol-birds amongst the criminal classes belong to this group; and in higher social spheres there are now and then met with unhappy creatures who, from their earliest years, have been addicted to lying and stealing, or every sort of vicious act—who have been expelled from school after school, the hopeless pupils of many masters, and who finally end in an asylum. They are instinctively vicious or criminal, exhibit a complete moral insensibility, and commonly masturbate; and they sometimes decline into mania and dementia.

Here, then, may conveniently be summed up in groups, according to their most prominent symptoms, the various forms of Insanity described, idiocy and general paralysis, which yet remain to be described, being added:—

I. Affective or Pathetic Insanity.

1. *Maniacal perversion of the affective life. Mania sine delirio.*
2. *Melancholic depression, or Simple Melancholia.*
3. *Moral alienation proper;* approaching which, but not reaching the degree of positive insanity, is *the insane temperament, or the neurosis spasmodica.*

II. Ideational Insanity.

1. *General:*
   a. *Mania* { *Acute.* / *Chronic.* / *Recurrent.*
   b. *Acute Melancholia.*
2. *Partial:*
   a. *Monomania.*
   b. *Melancholia, Lypemania.*
3. *Dementia* { *Primary.* / *Secondary.*
4. *Idiocy.*
5. *General Paralysis.*

In making use of this or any other provisional classification of symptoms, it should be clearly understood that the forms of Insanity are not actual pathological entities, but different degrees or kinds of the degeneration of the mental organization—in other words, of deviation from healthy mental life: and they are consequently sometimes found intermixed, replacing one another, or manifested in successive order in the same individual. There is a strong propensity not only to make divisions in knowledge where there are none in nature, and then to impose the divisions upon nature, making the reality thus conformable to the idea, but to go farther than that and to convert the generalizations from observation into positive entities, and then to permit these creations to tyrannize over the thoughts. A typical case of madness might be described as one in which the disorder, commencing in emotional disturbance and eccentricities of actions—in derangement of the affective life, passes thence into melancholia or mania, and finally, by a further declension, into dementia. This also is the natural course of mental degeneration proceeding unchecked through generations. The necessity of describing different forms of Insanity under different names should never lead to a neglect of the real relations which they have to one another as different stages of deviation from that mental life which we agree to regard as ideal or typical.

Idiocy.—It is an arrest of mental development by reason of some defect of cerebral development which is either congenital or occurs soon after birth. It will not appear strange that such defect is not always detectable when we reflect that the development of the brain as the ministering organ of the mental life really takes place after birth, and that an arrest thereof would take place within the recesses of the intimate activity of nervous element to which our senses have not gained access—where the subtlety of nature yet far exceeds the subtlety of human research. Marked imperfections of the brain are undoubtedly often met with in idiots. It is sometimes abnormally small, the general arrest of growth being due either to some condition of defective bodily nutrition, or to a premature ossification of the sutures of the skull and a consequent prevention of the growth which normally takes place actively during the first years of life. While it may be that there is no other defect than the abnormal smallness, it happens much more frequently that there are other anomalies, as hydrocephalus, unequal size of the hemispheres, and deficient development of the convolutions. All degrees of unequal size of the hemispheres have been met with, from that slight difference which is natural, to that extreme degree where a whole hemisphere has been replaced by a meshwork filled with fluid. Again, there is scarce a particular part of the brain which has not occasionally been found wanting: the corpus callosum may be defective or absent; there may be a deficient development of the anterior lobes, and a simplicity of the convolutions, such as belong to the lower animals; or the posterior lobes may not extend far enough back to cover the cerebellum, as normally they do not in some monkeys, and in all the animals below them. Chronic hydrocephalus, apparently primary in some cases, but in many others secondary to the atrophy or defect of brain, is frequent in idiots, and sometimes makes them large-headed; the serous fluid may exist in large quantity within the ventricles without being fatal, and death ultimately occur suddenly from a slight increase of it.[1] Sclerosis of the brain-substance often accompanies atrophy, or defect of development.

The irregularities of the skull in idiocy have been much studied of late. When the development of the brain is simply arrested, the growth of the bone may be arrested also, and then the skull is *microcephalic.* More often it would appear that owing to some constitutional defect of nutrition the arrest of the growth of the bone is primary, and a premature closure of the sutures takes place, whence follows a narrowing or shortening of the

[1] On Serous Effusion from the Membranes and into the Ventricles of the Brain, by John Sims, M.D.; Med.-Chir. Trans. vol. xix.—Clinical Notes on Chronic Hydrocephalus in the Adult, by S. Wilks, M.D.; Journ. Ment. Science, January, 1865.

skull. Compensating enlargements thereupon take place in some cases, the growth of the brain being in the direction of least resistance, and increasing the cranial deformity though making the mischief less. According to the suture prematurely closed, and to the amount and character of the compensating enlargement, will be the degree and kind of the deformity, of which many kinds have been described. Virchow has investigated with great care what he calls the tribasilar synostosis, which, occurring at the base of the skull, is the anatomical condition of the skull of Cretinism.

The causes of idiocy are sometimes traceable to parents. Frequent intermarriages in families lead to a degeneration that manifests itself in deaf mutism, albinoism, and idiocy; parental intemperance and excess, according to Dr. Howe,[1] occupy a high place as causes; and the natural term of Insanity proceeding unchecked through generations is, as Morel[2] has shown, sterile idiocy. During fœtal life great fright or mental agitation in the mother, or irregularities and excesses on her part, may lead to mental defect in the child. But perhaps the most frequent causes of an arrest of mental development are those which operate after birth up to the third or fourth year: they are epilepsy, the acute exanthemata, perhaps syphilis, and certainly conditions of bad nutrition, such as are produced by overcrowding, dirt, and want. Cretinism is an endemic idiocy arising out of unknown territorial conditions.

The extremest idiots are destitute of any intelligence whatever; they are apathetic, torpid beings, having a human semblance, whom it is necessary to feed, to move, to clothe, to take care of in every particular; who can attend to nothing, and remember nothing; who cannot speak a word; who grunt, make unintelligible sounds, and are unquiet if their appetites are not satisfied, or mechanically continue some automatic movement of hand, head, or body. The senses are almost invariably defective or wanting, the sensibility of the skin being commonly very imperfect, the hearing feeble, and smell and taste so deficient or perverted that the most acrid or filthy matters are eaten with indifference. The muscular development shares in the general defect; there are cramps of the limbs, contractions or paralysis of certain muscles, and epileptic convulsions. In Scotland there were, five years ago, 2236 imbeciles and idiots, of whom 43 were paralytic, 46 hemiplegic, 10 paraplegic, 17 choreic, and not less than 207 epileptic.[1] In less extreme cases there is evident want of power over the muscles; the walk is staggering and uncertain, the eye rolls vacantly, strabismus is common, the speech is defective, and there is slavering at the mouth. Sexual power is absent in the worst cases, and notwithstanding the self-abuse practised by some idiots, feebleness is in all cases more common than excess.

At the other end of the scale stand the so-called imbeciles, the highest of whom are only a little lower than those simple-minded people not deemed imbecile. The difference again between imbecility and idiocy is only one of degree, so that it is impossible to define it. In all sorts and conditions of idiocy two principal types may be broadly distinguished: one embracing the torpid and apathetic beings, who have usually some bodily deformity, and who give feeble signs of life; the other, those lively and excited beings who, rarely observably deformed, are unquiet and restlessly respondent to a rapid succession of impressions, who shriek, laugh, weep, gesticulate, clap their hands, get into mischief, and sometimes pass by a turbulent declension into true maniacal fury. Even the quiet idiots are occasionally liable to sudden attacks of fury, in which they bite, shriek, scratch, kick, beat their heads against the walls, and in other ways act viciously enough. Special talents or aptitudes of a remarkable kind, for remembering, for drawing, or for music, which seem quite inconsistent with the general character of their intelligence, are sometimes exhibited by idiots whose disease is of hereditary origin.

Esquirol divided idiots into three classes, according to the condition of speech. The first division included those who could use words and short phrases; the second, those who could only utter monosyllables and certain cries; the third, those who had neither speech nor monosyllable. Dr. Hack Tuke[2] proposes a physiological division of them into, first, those who exhibit only reflex or excitomotor movements; secondly, those whose acts are sensorimotor and ideomotor; and thirdly, those who manifest volition. It is a division which, not perhaps practically available, serves to mark the different degrees of degeneracy. By Griesinger, idiots are grouped in certain types: 1. Well-formed children in whom the mental development, which remains at the lowest grade, is the only apparent de-

[1] Report on the Causes of Idiocy.

[2] Traité des Degénérescences physiques, intellectuelles et morales, de l'Espèce humaine, par Dr. B. A. Morel, 1857.

[1] The Psychology of Idiocy; Journ. Ment. Science, April, 1865.—Epileptics: their Mental Condition, by W. A. F. Browne, Commissioner in Lunacy for Scotland; Journ. Ment. Science, October, 1865.

[2] Manual of Psychological Medicine, by Dr. Bucknill and Dr. D. Hack Tuke.

fect, the defect not being due to any hereditary, but to some accidental cause of degeneration. 2. The cases in which both bodily and mental development have been palpably arrested: these are the dwarfs in mind and body. 3. The Cretin, or basilar-synostotic type of idiocy or imbecility. Cretinism generally manifests itself a few months after birth, and is frequently associated with bodily deformity and goitre; and it is supposed to be due to some miasmatic influence primarily affecting the growth of the bones of the skull. It is most common amongst the mountains of Switzerland, but is met with sometimes in badly-drained places, and now and then sporadically. 4. The Aztec type, consisting of the true microcephalic idiots. 5. The theroid idiots, who have a sort of resemblance to some animal. Pinel, for example, gives a striking account of an idiot who was very like a sheep in habits and manner; and some idiots irresistibly bring to mind the monkey. Still, however degraded an idiot may be, he never really reverts to an animal type; for he represents a new and morbid variety, which but for the fostering care of higher beings, would speedily be extinguished.

Though idiots can never reach a normal development, their condition in many cases may be much improved by persevering training. The faculties which they do possess may thus be brought out in a remarkable manner, and they be made automatically skilful in certain tasks. When epilepsy has coexisted with the idiocy, and has afterwards disappeared, marked improvements may take place. Idiots very seldom attain old age; they are "old in their youth, and die ere middle age," apparently from lack of vitality. In some cases, the disease of brain—atrophy or hydrocephalus—directly leads to death.

General Paralysis.—It is a form of Insanity, first described by Bayle and Calmeil, which is characterized by a progressive diminution of mental power, and by a paralysis which gradually increases and invades the whole muscular system. It is far more frequent amongst men than women, and its most frequent cause is intemperance of some kind. Two of the best marked examples of this disease which I have seen, occurred, however, in teetotallers, who never had been given to alcoholic excess; but in both there was hereditary taint, and in both there was reason to suspect enervating, though marital, sexual excess.

Much discussion has taken place as to whether the mental symptoms precede the paralytic phenomena, or whether the latter first appear—whether the Insanity is primary, or whether, as Baillarger holds, the paralysis is the primary and main affection, the Insanity secondary and accessory. There can be no question in the minds of those who, unbiassed by any theory, simply observe cases, that the mental disorder does sometimes appear simultaneously with the motor disorder; that far more frequently, indeed most frequently of all, the mental symptoms are observed some time before there is any trace of paralysis; but that in some few cases the paralytic phenomena do precede by a short period the mental symptoms. In fifty-one cases out of eighty-six observed carefully by Parchappe, he found the paralysis and mental disorder to be simultaneous; in twenty-seven cases the paralysis was subsequent, and in eight the precedence was undetermined. Leidesdorf has related one case in which the earliest symptoms were spinal; and one or two similar cases have been recorded.[1]

The motor symptoms are first witnessed in the tongue, which has to execute the most delicate and complex movements with so much precision, and especially in the articulation of words abounding in consonants, where the most complex coordination is required. When the patient speaks earnestly, he does not articulate exactly, and there is a certain pause or indecision detectable in his utterance, as if there was some difficulty in bringing out the word; in some cases the speech is slower, more deliberate, with a strong accentuation of and a lingering on the syllables, as if the patient were speaking with great consideration. When the tongue is put out, which is done with some difficulty, there is a fibrillar quivering or trembling of its muscles, but it is not pulled to one side. There is a tremulousness, also, in the muscles of expression when put in action—especially in those of the lips, which quiver as in one just about to burst into tears. These phenomena, which are of fatal omen, may not be apparent at first, when the patient is calm and collected; but if he has had a sleepless night, or if he is much excited from any cause, then they become evident. An inequality in the size of the pupils is often an early symptom, but it is not a characteristic one; it is sometimes present in other forms of Insanity, and it is not always present in general paralysis. A transitory squint is observed occasionally at the commencement of the disease, and at a later period a slight ptosis of the upper eyelid. As the disease increases, the patient's walk becomes affected: the feet are not properly raised and firmly put down on the ground; he easily stumbles at a step or on uneven

[1] Beiträge zur Diagnostik der Geisteskrankheiten. Von Dr. M. Leidesdorf.

ground, and if asked suddenly to turn round when walking straight forward, he will stagger like a drunken man. He may nevertheless be very active in walking, and commonly thinks himself wonderfully well and strong. Precise coordination of movement, such as is necessary for writing, sewing, and like acquired automatic acts, is lost. The disease still advancing, the articulation becomes less distinct, the walk more and more tottering; the knees fail: the patient frequently tumbles, and finally cannot get up again without help. At last the primary automatic or reflex movements fail; the pupils become dilated, but unequal in size; the sphincters lose their power, and the patient may be choked by a lump of food getting into his larynx. Sometimes there are transitory contractions of arm or leg, and a grinding or gnashing of the teeth is not uncommon. The contractility of muscles to the electric stimulus is retained.

Cutaneous sensibility is usually diminished in the early stages, and at a later period it is sometimes lost. Yet there are occasionally transitory conditions of extreme hyperæsthesia, so that the patient shrieks out in great agony, or the slightest touch produces reflex movements or even convulsions. The muscular sense is especially affected, so that the sufferer, deprived of power of executing all complex and delicate movements, deems himself not less skilful than when at his best state, or, quite paralyzed, believes himself to have a giant's strength. The special senses are commonly unaffected until near the end, when smell and taste are diminished or lost, and vision fails. Sometimes, however, the patient has vivid hallucinations in the night: there were glorious visions of angels descending from heaven on ladders of gold in one patient under my care, and an agonizing vision of his own wife in the act of adultery rendered another frantic for a time. A great increase of sexual desire and an excited display of it are not unfrequent at the beginning of the disease, but there is not corresponding power; and what power there may be is soon quite lost.

The mental derangement is commonly marked by an exaggerated feeling of personal power and importance. After a brief stage of melancholic depression there is a notable change in the character, manifest in different ways: in one there is great mental excitement, and he is joyously and actively busy with wide-sweeping projects and speculations, indifferent to stern realities, and in all ways eager and ready to accomplish the impossible: in another there is a lack of former energy, and he is painfully troubled about little things, dull and confused in his thoughts, and demented in behavior; while another exhibits unwonted perversities of feeling and conduct, such as mightily astonish his friends; he breaks out into sexual excesses quite foreign to his usual sober character, or orders numerous articles of jewelry for which he can never pay, or even steals what strikes his fancy. Begin as it may, the mental disorder, when unchecked, generally issues in incoherence and extravagant delusions as to personal power and grandeur: the miserable sufferer who can scarce support his tottering body avers that he has the might and vigor of Hercules; while industriously hoarding up pieces of rag, paper, or glass as articles of value, he will sign a check for countless millions, or make an easy present of New York; maintaining that he can command a king to do his pleasure, in the same breath he prays piteously to be allowed to go to his own humble home; or, with sexual power extinct, boasts exultantly that a princess shall be his wife and princes be born of his loins. An extreme loss of memory is in striking contrast with the semblance of exaltation; the patient forgets entirely how long he has been in confinement, or denies angrily that he has a wife, though recognizing her gladly when she visits him. In some cases the delusions are of a terrific character and accompanied with great emotional depression; and a day of melancholic depression may now and then intervene in the course of the exalted form. There is a class of patients who present in physiognomy and habit of body a mixture of stupidity and the deepest depression, and exhibit sad delusions of as extreme a character as the delusions of grandeur: they think themselves bodily transformed in whole or in part; that their body has been immensely enlarged, that their eyes cannot see, their ears cannot hear; that their throat is sealed up. Dr. Clouston has pointed out that there is an intimate relationship between this form of general paralysis with depressions and tuberculosis. In another variety, of rare occurrence, the mental disorder consists in a regular decline of intelligence—a gradually increasing stupidity from the first. Attacks of great excitement and blind violence frequently occur during the progress of the disease. During them L. Meyer has observed the temperature of the head to be raised, and after them the mental decay is found to have increased. As the disease approaches its end—the end of life—the dementia is extreme, and the face becomes an expressionless mask across which now and then flickers the broken ripple of a smile, or it is fixed in a ghastly, sardonic grin; but even in the last stage of mental disorganization, when the capability of a distinct delusion is

gone, the muttered words are oftentimes about golden carriages and millions of money.

The *course* of general paralysis is towards death, though not steadily so. Under proper treatment a great improvement takes place in the early stages, and the disease seems to be arrested. Some have thought that actual recovery does now and then take place; and certainly there have been in exceptional cases intermissions of such a length that the disease has lasted for ten years. On the whole, however, it is irregularly progressive, its duration being usually from a few months to about three years. It hardly ever occurs before the age of thirty. In the more advanced stages sudden attacks of loss of consciousness, with epileptiform convulsions, are not unfrequent, after which the paralysis and mental decay are both found to have increased. Dr. Saunders has observed that the temperature of the body in general paralysis is generally one or two degrees below the average, but that it rises during the excesses of maniacal excitement, falling again as calmness returns. During the so-called congestive attacks, again, where there is complete coma or epileptiform convulsion, there is generally a considerable rise of temperature: in one case the temperature was for some time 98°, but it rose an hour after one of these attacks to 105°, and next day to 106°, the patient dying in thirty-six hours from the commencement of the attack.[1] Dr. Clouston has found that in general paralysis there may be a difference of 5·8° in the same individual in different stages of the disease. In the last miserable stage of all, when life flickers before extinction, large sloughing bedsores form, notwithstanding the best care, and diarrhœa or pneumonia hastens the long-expected ending.

Diagnosis.—The diagnosis of Insanity is as difficult in some cases as it is easy in others. Acute mania cannot well be confounded with any other disease, and the only doubtful question in regard to it will be in the event of an impostor attempting to simulate it. He must be a clever impostor, however, who can simulate the wild restless eye, the quick fragmentary associations of ideas, the rapid capricious movements, and the volubility of utterance of acute mania, so as to deceive an experienced observer; nor can he, however skilful, pass days without sleep, and even weeks with only a few hours' sleep, maintaining a constant activity the while, as the maniac does. The skin in acute mania is dry and harsh, or clammy, but the skin of a pretender who tries to keep up the muscular agitation will hardly fail to be hot and sweating. *Meningitis* will be known from mania by its own positive symptoms,—by the premonitory rigors, when they occur, the cephalalgia, the fever, the contracted pupil, and the intolerance of light; by a muscular activity, paroxysmal, not continuous, and by frequent spasms or convulsions; by the acute severity of the delirium and the vivid illusions of the senses; and by its rapid progress either to recovery or death. *Delirium tremens* will also be distinguished by its characteristic symptoms—the muscular tremors, the peculiar fearful illusions and hallucinations, the cold skin, feeble pulse, and white and tremulous tongue. It must not be forgotten, however, that persons who have a strong predisposition to Insanity, or who have been insane, or who have suffered at some time from a severe injury to the head, do sometimes after an alcoholic debauch become truly maniacal for a time. In this condition, which may be of short duration, dangerous hallucinations sometimes arise, and the sufferer may perpetrate crime, not knowing afterwards what he has done: of this fact a searching investigation of instances can leave no doubt. The result again of continued intemperance, or of a long debauch, is sometimes to produce, not a delirium tremens, but a true mania, marked by active and violent delirium.

Chronic mania is the most likely form of Insanity to be feigned, and if feigned with the skill of Hamlet, the very elect may be deceived. A vulgar impostor will, however, generally "o'erstep the modesty of nature," and overact the part in the extravagance of what he says, and in the absurdity or violence of what he does, while he will almost of a certainty fall short of his part in the emotional expression of the maniacal countenance. Having the popular notion that a lunatic is widely different from a sane person, he will entirely fail to understand the character which he wishes to represent, so that an experienced person may detect his ignorance; and believing that he must make a great display in order to produce an adequate impression, he will, like a bad actor, exaggerate and rant, so that any one with insight, though without special experience, may discover his incompetency. He will pretend that he cannot remember the simplest things, that he cannot reckon correctly, and will act extravagantly, and answer stupidly or wrongly, when a real lunatic would act calmly and answer intelligently or rightly; he will moreover show no irritation or anger at the suspicion of his sanity. It may not be amiss to suggest incidentally in his hearing some symptoms which he ought to exhibit, and to take notice whether he subsequently adopts the hint.

[1] Report of the Devon County Asylum for 1864.

If he refuses to converse, feigning apathetic dementia with dirty habits, which is perhaps the form of insanity most often feigned, the diagnosis becomes more difficult, and a long observation may be necessary in order to establish it. It is truly astonishing how long an impostor will sometimes persist: one man, whose story Dr. Bucknill tells, kept up the pretence of Insanity for more than two years, and then broke down in his part. The two important considerations to be kept in view are, first, the existence of any motive for feigning, and secondly, the consistency or inconsistency of the symptoms with a definite form of mental disease, Perhaps it would not be far from the truth to say that he who can feign madness so completely as to deceive an experienced observer, is not far from being the character which he represents; for unless there be real madness beneath the feigned phenomena, there will be some want of coherence in them as a whole, and an incongruity with any recognized form of mental disease.

The discovery of chronic mania or monomania where it does exist, but where the patient is suspicious and strives to hide it, may be a very difficult task. There is generally some sign of the disease in the countenance and bearing of the patient: "The principal characteristic in some is," says Dr. Bucknill, "a peculiar want of harmony in the expression of the features: in others, the fixed expression of some intense emotion is remarkable—of defiant pride, of sullen obstinacy, of smirking vanity, or of leering sensuality." The demeanor may be defiant, sullen, restless, or absorbed, and the clothing untidy and neglected: in both demeanor and dress there are sometimes peculiarities which, when bottomed, open up a secret mine of madness. To detect any delusion the patient should be examined carefully on all matters intimately touching himself, anything peculiar or notable in his expressions or any obscure references, being watched for, noted in the mind, and subsequently quietly followed up. If he seems to pass hastily over, or to avoid, some subject, he should be unobtrusively but steadily pressed upon it; and if he declines to speak of the matter, or gets angry, the refusal or irritation is alike significant. All this should be done in as quiet and amiable a manner as possible, so as to avoid giving unnecessary offence, and to make it a difficult matter for the patient to take offence and break off the interview. It is an intellectual contest between a sound and an unsound intellect, in which the weakness of the latter is compensated by its acting on the defensive, the superiority of the former lessened by its acting on the offensive. Heinroth has affirmed, what is popularly thought, hat though the insane may often *conceal* their delusions, they cannot *deny* them. This is by no means true of all cases; some will deny their delusions with as much energy as Peter showed when he denied the dangerous truth, or will even labor to explain them away as jokes. When all else fails, it may be worth while grievously to offend the patient's self-love, and to make him extremely angry, and in the fury of passion he will sometimes, notwithstanding his suspicion, reveal the hidden delusion. Failure will be rare with the expert who likes to be persistent and patient enough. In doubtful cases, it will always be well to get the patient to write, for it is truly surprising what extravagant delusions may be exhibited in a letter by one who manages to conceal them in conversation. Careful research should of course be made into the previous history, in order to discover whether there is hereditary taint, and what degree of it; whether there has been any previous attack of Insanity, and whether there has been any observed change of feelings, character, and habits, especially after some efficient cause of Insanity. At the outset a patient sometimes has a suspicion that he may be thought mad, and is very earnest and vehement in accounting for his morbid feelings, and at great pains to convince those around him that he is not mad.[1]

Melancholia is not usually difficult to recognize, as patients in most cases do not care to conceal their painful delusions; still there are some who do not only conceal but deny them. A patient afflicted with homicidal and suicidal impulse, and intensely miserable in consequence, will positively conceal and deny the morbid impulse, in order to throw those around off their guard and the better to effect his purpose; and more than one such, foolishly removed from under control by ignorant but well-meaning friends, has afterwards committed suicide or homicide, or both. Another, who cannot entirely conceal his disease, will even attribute his depression to the confinement which he is undergoing, and asseverate most earnestly that he will be quite well at home; this intense eagerness to be delivered from control being truly the surest sign that he is not fit to be from under control. In all such cases it is necessary to watch patiently from day to day; for it will sometimes happen that a delusion, denied on one day, is predominant on another, and it is very apt to become so, if the patient does not see a prospect of release through his simulation of sanity.

[1] There are some excellent observations by Dr. Bucknill in the Manual of Psychological Medicine, on the mode of conducting the examination of an insane person, p. 310.

It may be important to distinguish between the hypochondriac and the melancholic, as the former, committing a murder, would certainly be hanged, the latter probably not. The hypochondriac refers his sufferings to some organic disturbance or disease, in which there may be more or less reality; he displays an exaggerated sensibility in regard to all his organic processes, or to some one of them in particular, so that he has either many delusions respecting his health, or his whole habit of thought respecting it is perverted; he is fond of talking of his sufferings, and of consulting medical men; he evinces a great love of life, and no disposition to commit suicide; his intellect is sound, and his feelings are not perverted, apart from questions touching his health. The melancholic, on the other hand, refers his sufferings to some groundless extraneous cause, either operating from without, or having taken possession of body or soul, or both, so that he has frequently a single and fixed delusion; his anguish is a mental anguish, and he asserts that medicine can do him no good; he is often suicidal; his affective life is profoundly implicated, and he is incapacitated from intellectual activity, though there may be no marked intellectual derangement apart from the delusion. It must not be overlooked, however, that hypochondriasis may pass into true melancholia, as well as coexist with it, and that a true hypochondriacal melancholy may rise to such a pitch as to render the individual irresponsible for his actions.

Melancholia with stupor may easily be confounded with acute dementia; nor is it always possible to distinguish them at the outset. The expression of the melancholic is that of one astonied, or as if fixed in a painful trance—the mind veiled, as it were, by a great cloud let down between it and the external world. The patient stands or sits in one place, or moves slowly to and fro; he often offers a passive resistance to being moved, or to being fed; sometimes he exhibits a strong tendency to suicide, and, now and then, a temporary excitement; on recovery, he remembers his suffering as a painful dream, or as a strange and fearful trance, during which he was partly conscious of things around, but unable to express himself. In dementia, the countenance is expressionless; there is no resistance to being moved, or to food; the patient is not suicidal; the bodily functions are less affected; on recovery, there is no memory of the attack.

In certain criminal or civil trials, it may be necessary to distinguish between eccentricity and Insanity. There is a great gap between them: the truly eccentric man has a strong individuality, but little vanity; he has broad and original views, and great moral courage; he is emancipated from vulgar prejudice, and heeds not much the world's blame or censure; he differs from the majority perhaps because he is in advance of the habits and superstitions to which it is in bondage; and he is not at all likely to become insane. But there is a weak affectation of eccentricity which is very apt to end in Insanity: with it are infected certain feeble-minded beings, often badly bred or badly trained, who are empty of any true individuality, but inflated with an excessive vanity; who have a small intellect which they use in the service of their passions; who do silly and eccentric things, not unconsciously as the spontaneous expression of their nature, but out of a morbid craving to attract attention; who represent a condition of mental derangement that is the forerunner of Insanity; who, when they are not given up to sexual excesses, are often masturbators.

In the diagnosis of so-called moral insanity, it is necessary to look for a sufficient cause of mental disease from which the vicious or violent acts may be logically traced through a train of symptoms, such as marked change of character, feelings, and habits. Neither vice nor crime, however extreme, is proof of Insanity. To be so, it must be proved through a chain of morbid symptoms to flow not from passion, but from disease; and attention should not, therefore, be entirely occupied by it, but should carefully traverse the whole affective life, in order to reveal the perversion of nature detectable in a case of real moral Insanity, and the connection of the morbid change with an efficient cause of disease. A man may get into the police-court for stealing, in whom one may perceive instantly the earliest symptoms of general paralysis; another may commit murder, apparently without motive, or from a very inadequate motive, in whom a melancholic anguish has risen to a convulsive explosion; and a third may perpetrate violence in a state of affective derangement, which skilled observation recognizes to be premonitory of, or vicarious of, an attack of epilepsy.

When it is a question of the existence of an irresistible impulse in any case of homicidal or other kind of violence, it is very necessary to keep in view the possibility of epilepsy, either in the form of epileptic vertigo or in its convulsive form. When an epileptic person has done a murder without discoverable motive, without advantage to himself or any one else, without premeditation, without malice, openly, it is almost certain that he has been driven to the crime by an irresistible impulse.

General paralysis is easily recognized after it has passed its earliest stage. It is not always easy of diagnosis before

the physical signs appear; and yet a man may at this stage get into trouble—get into the police-court, or get married foolishly—entirely by reason of the disease. It is necessary to weigh carefully the character of the act, whether it is anywise explicable, or motiveless and quite unaccountable; to mark well the state of the patient's articulation under excitement or after a sleepless night; to note his happy and elated mood of mind; and to attend to the great exaggeration and general extravagance of his conversation on all matters concerning himself, even when there is no fixed and positive delusion. General paralytics, in the early stage, speak so extravagantly and absurdly regarding things which they have seen, or events in which they have been concerned, that an inexperienced person might be apt to put down the whole as a delusion. It is needful to bear in mind that there may be some foundation of fact in what they say of themselves—that they do not at first so much invent as outrageously exaggerate. It is needful also to remember the alternations of calmness and apparent sanity which occur in the early course of the disease. The main points to direct attention to are, first, any indications that there may be of altered speech and of loss of memory and mental power; and, secondly, the light in which the patient regards these symptoms when they are pointed out to him. If they are positively present, and there is on his part an entire unconsciousness of them, or if he laughs at and makes light of them, as is often the case, affirming that he was never better in his life, then it is almost certain that he is the victim of incipient general paralysis.

Pathology.—Though it may be that there are no morbid changes detectable in the brain of one who has died raving mad, it is none the less certain that, with change of energy, there is a correlative change in the nervous substratum. Nothing is yet known of the intimate constitution of nervous element, or of the mode of its functional action; and it is beyond question that the difference in its condition may be the difference between life and death, without any appreciable physical or chemical change. As the means of research improve, however, the instances of Insanity in which morbid changes are not found are more and more rare; and those who have most studied the matter are those who are most certain and speak most confidently of the invariable existence of such changes. When a morbid poison acts with its greatest intensity, there are notably fewer traces of structural alteration than where its action has been less intense and more prolonged; and, in like manner, appreciable organic changes in Insanity may be justly expected only when the degeneration has been extreme and long continued—in chronic mania, in chronic dementia, in general paralysis, and epileptic Insanity. Where this has been the case, morbid changes are seldom looked for in vain.

Investigations into the electrical properties of nerves, and into the phenomena of conduction by them, have not only rendered conceivable the existence of important, though undetectable, molecular changes among their ultimate elements, but have proved the necessity of dismissing all metaphysical conceptions of nervous function, and of making positive research into the physical and chemical conditions which, whatever its nature, determine its manifestations. So far from conduction by nerve being due to the instantaneous passage of some imponderable principle, it depends upon a modification of its molecular constitution, for which a certain time is essential; for it has been shown that a definite period of time, varying in different persons and at different periods in the same person, is necessary for the propagation of a stimulus from the peripheric ending of a nerve to its central ending in the brain; and when the stimulus has reached the brain, a certain time must elapse before the will can transmit a message to the muscles so as to produce motion. No investigation has yet been made of the time-rate of activity of the cerebral centres, but there is assuredly a considerable variation in the time in which the same mental functions are performed by different persons, or by the same person at different times. "The mind in health," says Locke, "will boggle and stand still, and one cannot get it a step forward, and at another time it will press forward, and there is no holding it in." Appreciable and variable, then, is the time-rate of thought, and the measure probably of that intimate molecular activity which is the condition of its manifestations. That such molecular activity does take place, the "waste" after function proves; the chemical reaction of nerve becomes acid after activity, owing probably to the formation of lactic acid in the retrograde metamorphosis; and the increase of phosphates in the urine, and the bodily exhaustion after great mental work, are only to be explained by supposing an idea to be accompanied by a correlative change in the nerve-cells. Here, indeed, is a region of most delicate activity, which, like that of thermal oscillations, or of undulations of light, is yet impenetrable to sense; and so far from its being improbable that undetected morbid changes may exist in Insanity, so far from its being wonderful that morbid appearances are sometimes not found, the wonder truly is that they should have been expected always. Where the sub-

tlety of nature so much exceeds the subtlety of human investigation, to conclude from the non-appearance the non-existence of change, would be analogous to the blind man asserting that there are no colors, the deaf man that there are no sounds.

Not only have erroneous ideas been entertained respecting the kind of organic change that might suffice to give rise to Insanity, but the nervous element itself, as a living individual entity with intrinsic properties, has been commonly ignored; the main stress having been laid upon the bloodvessels, as if they were the primary agents in exciting and keeping up the mental disorder. The truth is, that the first step in Insanity often is, as it is in inflammation, a direct change in the individual elements of tissue, the change in the bloodvessels being secondary. The experiments of Lister[1] have proved that in the case of mechanical or chemical injury to some tissue, the individual elements are directly injured, and that a determination of blood, a dilatation of the vessels, and an adhesion of the corpuscles follow the local mischief; and it is easy to conceive that damage to the nervous element of the brain, however caused—whether from overwork or emotional anxiety, or from poison in the blood, or by direct injury or reflex irritation—may in like manner declare itself in disordered function, and be accompanied or followed by vascular disturbance. The nervous element is brought to a lower state of life, and manifests its deviation or degeneration from its normal kind by an abnormal or degenerate energy, while the disturbance of circulation takes place as a coincident or sequent effect of a common cause. Where this is hereditary taint, there is innate vice or defect in the constitution of nerve element, and it will accordingly break down more easily under adverse stress. The effects of strychnia may serve to illustrate the presumed course of events: when a dog is poisoned with strychnia, there may be no morbid appearances; but if there be any, they are such as congestion of the spinal cord, aneurismal dilatation of the capillaries, and perhaps small effusions of blood into the gray matter. The congestion is plainly a secondary result of the intense morbid activity of the directly poisoned nervous element. Here, then, is the abstract and brief chronicle of the order of events in many cases of Insanity. Transfer the convulsive action from the spinal cells to the cortical cells of the hemispheres, the result is a violent and acute mania in which the acute determination of blood is certainly not the cause, if it be not the consequence, of the degenerate function. In what is called *mania transitoria*, the patient falls suddenly into a violent fury, in which he raves and often exhibits destructive impulses; his face is flushed, his head hot, and there is plainly an active determination of blood to the brain; and in a short time the fury subsides, and the man is himself again, scarce conscious, or quite unconscious, of what has happened. Was the rush of blood the primary and active agent in the production of the fury? Was it not rather secondary to the intense morbid or degenerate action of the nervous centre; the attack representing a sort of epilepsy, and the congestion taking place not otherwise than as it takes place in the spinal cord poisoned by strychnia? So in chronic insanity, the congestion discoverable may not be the cause, but the effect and evidence of the morbid action of nervous element. And in those cases of Insanity in which there is no special morbid appearance after death, though there has been fixed delusion during life, it is because the definite morbid action which does exist takes place in that innermost region of activity of individual element to which our senses have not yet gained access. Only by fixing attention on the individual elements will a just conception be formed of the mode of that degeneration which reveals itself in mental disorder, but at present is not otherwise revealed; and only thus will the morbid appearances that are met with receive their right interpretation.

It would be one-sided and mischievous, in another way, to overlook the fact that disturbance of circulation, of extraneous origin, may directly favor and even produce Insanity. Having regard to the extreme susceptibility of nervous element, and the abundance of the supply of blood to it, there can be no question that the quantity and quality of the blood play a weighty part in the pathology of Insanity.

Quantity and Quality of the Blood.—Since the time of Hippocrates, it has been known that similar symptoms are produced by too much and by too little blood in the brain. In that continued active relation between the blood and the nervous element, whereby due reparative material is brought and waste matter carried away, it amounts to much the same thing whether, through stasis of the blood, the refuse is not carried away, nor the supply brought to the spot where it is wanted, or whether the like result ensues by reason of a defective blood or a deficient circulation. Now, although temporary irregularities in the cerebral circulation may, and often do, pass away without leaving behind any abiding ill effects, yet when they recur frequently, and become more lasting, their disappearance is

[1] On the Early Stages of Inflammation, by Joseph Lister, F.R.S.; vol. xxxi. Philosophical Transactions, 1858.

by no means the disappearance of the entire evil; they are efficient to initiate a degeneration, which thenceforth continues of itself and leads to permanent mental derangement. Once the *habit* of a definite morbid action is fixed in a part, it continues almost as naturally as, under better auspices, the normal physiological action.

A vitiated state of blood, by reason of matters bred in it, or introduced from without, may act perniciously on the supreme cerebral cells. The rapid recovery which takes place after moderate doses of alcohol, opium, Indian hemp, seems to show that the damage they inflict is transitory; but it admits of no question, that when nervous element is repeatedly exposed to their poisonous agency, it acquires a disposition to a degenerate function. The intense gloom produced by the presence of bile in the blood, and the extreme irritability produced by some urinary constituent in the blood of a gouty patient, serve to show what effects upon the supreme nervous centres may be due to the non-evacuation of the waste matters of the tissues. When, furthermore, it is remembered that the blood is itself a living, developing fluid; that, "burnished with a living splendor," it circulates rapidly through the body, supplying the material for the nutrition of the various tissues, receiving the waste matter of their activity, and carrying it to those parts where it may either be appropriated and so removed by nutrition, or eliminated by secretion—it is plain that multitudinous changes are continually taking place in its constitution and composition, that its existence is a continued metastasis. There is the widest possibility, then, of abnormal changes in some of the manifold processes of its complex life and function, such as may generate products injurious or fatal to the life of nervous element. Poverty of blood undoubtedly plays a weighty part in Insanity, as in other nervous diseases; and there is, in the effects of the viruses of acute fevers, ample evidence that morbid poisons, bred in the organism, or entering it from without, may act in the most baneful manner on the nervous centres. In some cases of malignant typhus, and in the putrid infection after surgical operations, the virus generated is directly fatal to the life of nerve element; and when it acts with less intensity, it gives rise to the delirium of fever, and predisposes probably to the Insanity following acute fevers.

Reflex Action, or Sympathy.—The supreme cerebral centres may—like other nervous centres—suffer secondarily from morbid irritation in some other part of the body; though why they should do so at one time and not at another, we know not any more than why epilepsy should be caused by an eccentric irritation at one time and not another. That they do so, many recorded instances clearly testify.[1] When a chronic insanity is brought about in this way, the delusion has sometimes a relation to the primary morbid cause; the secondary derangement of the cerebral centres testifying to the special effect of the particular diseased organ—as, for example, when a woman, with uterine or ovarian disease, believes she is with child by supernatural means, or, with morbid irritation of the sexual organs, has salacious delusions. There is the most perfect harmony, the most intimate connection or sympathy, between the different organs of the body, as the expression of its organic life—a unity of the organism beneath consciousness; and the brain is quite aware that the body has a liver or a stomach, and feels the effects of disorder in any one of the organs, without declaring it directly in consciousness. This unconscious, but not unimportant, cerebral activity, which is the expression of the organic sympathies of the brain, receives its most striking illustrations in the influence on the mind of the development of the sexual organs at puberty, and in their subsequent influence on dreams; and it is of great weight in the production of morbid mental phenomena. A just appreciation of its importance will not fail to teach the lesson which a true conception of the organism as an individual whole, formed of differentiated parts harmoniously co-ordinated, teaches also, that every organic motion, visible or invisible, sensible or insensible, ministrant to the highest aim or to the humblest function, does not pass issueless, but has its due effect upon the whole, and is felt throughout the most complex recesses of the mental life.[2]

The primary morbid centre, which gives rise to secondary derangement by a reflex or sympathetic action, may not be in a distant organ; it may be in the brain itself. A tumor, abscess, or local softening does not interfere with the mental processes at one time, while it produces the gravest disorder of them at another; and it is not uncommon in abscess of the brain for the mental symptoms, when there are any, entirely to disappear for a time, and then to return suddenly in all

[1] References to such cases may be found in Griesinger's work. There is a remarkable case also cited by Dr. Brown-Séquard in his Lectures on the Physiology and Pathology of the Nervous System. 1860.

[2] "Man is all symmetrie,
Full of proportion one limb to another,
And to all the world besides.
Each part calls the furthest brother;
For head with foot hath private amity,
And both with moon and tides."
GEORGE HERBERT.

their gravity, the derangement or abolition having been due to a sympathetic or reflex action.

Here, then, should be distinguished the different kinds of disorder of the cerebral centres to which a morbid cause may give rise. The sudden appearance and equally sudden disappearance of extreme mental derangement prove that it is functional; it being impossible to conceive the existence of serious organic change in such case. Although, then, the functional disorder necessarily implies a molecular change of some kind in the nervous element, the change may well be one affecting the polar molecules, such as the researches of Du Bois Reymond have proved may rapidly appear and rapidly disappear. At any rate the induction, by definite experiments, of recognizable transitory changes, in the physical constitution and the function of nerve, warrants the belief in similar modifications producible by morbid causes which are not artificial, but which are just as abnormal as if they were artificial. The modification of molecular constitution, which vanishes at first with the removal of the cause, will not fail, if too great or too prolonged, to degenerate into actual nutritive change and structural disease, just as an emotion which alters a secretion temporarily may, when long enduring, lead to nutritive change in the secreting organ.

Excessive functional activity.—The display of function being the waste of matter, it is plain that if there be not due intervals of periodical rest, degeneration of nervous element must take place as surely as when directly injured by morbid poison, or mechanical irritant, or as surely as a fuelless fire must go out. It is sleep which thus knits up the ravelled structure of nervous element; for during sleep, organic assimilation restores as statical or "latent" the power which has been expended in function, or made "actual" in energy. Sleeplessness is, accordingly, one of the troubles following intense mental anxiety, or too great mental activity, and forerunning Insanity: that which should heal the breach is rendered impossible by the extent of the breach. Like Hamlet, according to Polonius, the individual falls into a sadness, thence into a watch, then into a lightness, and by this declension into the madness wherein he finally raves. To provoke repose in him is the prime condition of restoration; the power of its "closing the eye of anguish," and healing "the great breach in the abused nature" of nervous element.

[It requires some courage to question, for a moment, the almost unanimous conclusion of psycopaths at the present day, that Insanity *always* results from a physical lesion or disorder of the brain. But some cases occur, which (in the absence of a fatal result to allow of actual inspection of cerebral conditions) may, with the greatest probability, admit of a different explanation. Accepting the fitness of the French term, *aliéné*, any one may be held to be insane who, from any cause, becomes so "beside himself," so estranged from accordance in his judgment, apprehensions, motives, and actions with the realities of the world around him, as to be incapable of what all recognize as sane conduct and conversation. As with manifestly physically produced Insanity, there may be a close limitation of *delusions* and *consequent actions*, to one subject, or group of subjects, in persons who have become subjected to gross and injurious errors of opinion, so as to "believe a lie." Cases of Insanity produced by spiritualism may be named as of this kind. Those so affected continue, sometimes, for years, in a state of general health, almost incompatible with any marked disease of the brain; and yet obviously much astray in their judgment of facts and conclusions, and often led to do very strange actions in consequence. Superstitions of various kinds may account for similar mental alienations; as in the instance of a man who took the life of his child, in 1879, in one of the Eastern States, under the belief that he was divinely commanded to do so; his wife and some fellow-believers also justifying his conduct. One of the most distinguished American chemists presented, some time since, a sad instance of spiritualistic derangement in his latter years; and, more recently, a case perhaps equally remarkable has occurred, in a man in the prime of life, a Superintendent of Public Education in one of the largest cities in America. If, in every such case, it be insisted that there must be a causative lesion or disorder of the brain, such a statement is to be accepted rather as a matter of pathological theory or dogma, than of necessary inference from the facts observed. In some instances, coming under the knowledge of the present writer, the opinion above implied was first suggested by the history of the mental aberration, and afterwards confirmed by the restoration of those affected after a considerable period, simply through their being enabled to find the clue which led them out of the labyrinth of delusion in which their reason was astray. Certainly the diagnosis of brain lesion, in such cases, would have been difficult to make out, on any other ground than that of an *axiom* in psycho-pathology.—H.]

Thus much from a pathological point of view concerning the causation of Insanity: it now remains to enumerate the morbid appearances that have been met with in the brain and its membranes.

Morbid Anatomy.—The broad result of investigation is, that the morbid changes most constantly met with are such as affect the surface of the brain and the membranes immediately covering it; and of these changes, those in the layers of the cortical substance are the principal and essential. The signs of more or less inflammation of the membranes, especially milky opacities of the arachnoid, are common enough in the bodies of those who have not died insane. But there would appear to be some hindrance to inflammation spreading easily to adjacent parts that are of different structure, whereas, when they are of the same structure; it passes readily from element to element of the same kind, as it were by an infection; the intercostal muscles are scarcely affected in acute pleurisy, the muscular walls of the intestines scarcely affected in peritonitis, and inflammation takes place in the membranes of the brain, without seriously implicating the cortical layers. If these are involved, there can be no question that the mind suffers. Deaths in the acute stage of Insanity are not usual; but if an opportunity presents itself of examining the brain at this early stage, the morbid appearances are those of acute hyperæmia—namely, great injection of the pia mater, with spots of ecchymosis, and more or less discoloration and softening of the cortical layers, which may be separated easily with the handle of a scalpel from the white substance beneath; the discoloration being in red streaks or stains, with spots of extravasated blood, and the softening of a violet or pinkish hue; the puncta vasculosa of the white substance are also increased. There are no observable differences between the morbid appearances met with in acute mania and in acute melancholia; and though such fact ill agrees with their different symptoms, it is not entirely singular, forasmuch as alcohol makes one man lively and another melancholic. A differential pathology would involve the knowledge of what constitutes individual disposition or temperament. It must be confessed that in both mania and melancholia morbid changes are sometimes wanting.

The instances of chronic insanity in which no morbid lesions appear are rare: the longer the disease has lasted, the more evident they usually are. There is mostly some degree of thickening or opacity of the arachnoid, which may form a white opaque layer through which the convolutions are scarce visible; and many of the more advanced cases exhibit some degree of atrophy of the brain, especially of the convolutions—these appearing shrunk, pale, and anæmic, or as if some were wanting and replaced by an effusion of clear serum into the subarachnoid space. The atrophic change may be, according to Dr. Wilks, a simple degeneration, or a degeneration associated with the changes resulting from chronic inflammation. The pia mater is at times adherent to the surface of the brain, so that there is some difficulty in stripping it off without bringing portions of gray matter away with it. This adhesion is not peculiar to general paralysis, as some have thought, though most often met with in it; for it is now and then found in other forms of chronic insanity, particularly those following epilepsy and drunkenness. The ependyma of the ventricles is thickened, and sometimes covered with fine granulations, such as have been described also by L. Meyer on the arachnoid and inner surface of the dura mater.[1] Dr. Wilks has seen a minutely granular condition of the lining of the ventricles, in a case of acute mania; he has often seen it in epilepsy—once, when the granules were as large as peas, and "the whole surface of the ventricles had very much the appearance of an ice-plant." In some cases, the exudation is in flattened scales or patches.[2]

The morbid changes most frequent in general paralysis, though in rare instances there are scarcely any detectable, are great œdema of the membranes, adhesion of the pia mater to the gray substance beneath, local discoloration or softening of the cortical layers, or superficial induration of them. More or less atrophy of the whole brain, and particularly of the convolutions, is common, and is accompanied with greater firmness of its substance, enlargement of the ventricles, and serous effusion into them. Diffuse pachymeningitis, effusion of blood into the membranes, or rather into the layers of exudation (Virchow, Rokitansky), and degeneration, atheromatous and calcareous, of the arteries, are not unfrequently met with. Though these changes are more common in general paralysis than in any other form of Insanity, they are by no means peculiar to it, nor are they constant in it; in some cases the evidence of meningitis is most marked, in others that of atrophy.[3]

A diffuse albumino-fibroid exudation of low form, glueing the membranes to the surface of the brain, has been declared by some to be characteristic of syphilitic insanity. Instead of being diffused, the *gum-like* exudation, or *syphiloma*, as it has

[1] Virchow's Archiv, B. xvii. s. 209.

[2] Clinical Notes on Atrophy of the Brain, by S. Wilks, M.D.; Journ. Ment. Science, October, 1864.

[3] A compact account of the morbid changes in general paralysis, with exhaustive references, will be found in a paper by Dr. E. Salomon on the Pathological Elements of General Paresis; Journ. Ment. Science, October, 1862.

been called, may be circumscribed so as to form a tumor, and press into the substance of the brain, causing softening immediately around it; or again, it may be met with as a diffuse infiltration or a tumor within the brain, the membranes being unaffected. At the outset it consists of an exuberant growth of connective tissue, which afterwards undergoes more or less fatty degeneration; and it certainly has not any character by which it can be distinguished as a specific product.[1]

Researches have been made into the absolute weight and specific gravity of the brain in Insanity, but they have not been sufficiently exact.[2] Dr. Skae and Dr. Boyd found the absolute weight to be slightly increased in the insane, the increase being greatest in mania, and least in general paralysis. The specific gravity is also increased, the lowest specific gravity, which is still above the average, occurring in dementia, the highest in epilepsy. Dr. Bucknill observed that the mode of death influenced the results, and found also that the increase of specific gravity was due, in some cases, to a deposit of an inert albuminous matter amongst the proper nervous elements, and the consequent shrinking of these,—a condition seemingly not unlike that since described by Prof. Albers as *parenchymatous infarction of the brain*, and met with after typhus, in some cases of old Insanity, and in imbecile children, especially those of a scrofulous habit of body. A fibrinous or albumino-fibroid exudation would appear to be a not uncommon result of the degeneration of extreme Insanity; it is the condition probably of the increased consistency, or *sclerosis*, which is the final result of atrophy; and it is comparable with the product of what is described as chronic inflammation in other organs, as the liver and spleen. A similar exudation is the cause of the so-called hypertrophy of the brain from which children sometimes die, and which is now and then met with in single cases of Insanity and epilepsy.

The microscope has of late years added something to our knowledge of the nature of the morbid changes in Insanity. The most constant result has been to establish a rank or exuberant growth of connective tissue, and a coincident or sequent decay or destruction of the proper nervous elements, in Insanity of long standing, and especially in general paralysis. The researches of Rokitansky and Wedl reveal a more or less diseased state of the capillaries of the cortical substance in general paralysis; these exhibit aneurismal dilatations, and tortuosities varying from a single twist to a more complex twisting and even to little knots of varicose vessels.[1] Round the capillaries, small arteries, and veins there is a hyaline deposit of embryonic connective tissue beset with oblong nuclei; this afterwards becoming more and more fibrous, so that the vessel may look like a piece of connective tissue, in which granules of fat or calcareous matter ultimately appear. Other products of the retrograde metamorphosis, such as amyloid corpuscles and colloid corpuscles, are also found in connection with the hypertrophied tissue, which, whether called inflammatory or not, is itself essentially the result of a vital degeneration. The degeneration appears to be of two kinds: first, there is a defective nutrition, a retrograde nutritive process, whereby the vitality not reaching the height necessary for the production of the proper elements of the structure, there are engendered from the germinal nuclei elements of a lower kind—connective tissue instead of a nerve; and, secondly, there is a retrograde metamorphosis of the formed elements of the part—a colloid, fatty, or calcareous degeneration. Be this as it may, there are at any rate three principal stages of the degenerative process: (1), a morbid change in the vessels, whereby there must be a great hindrance to regular nutrition; (2), atrophy of nerve element, either in consequence of the hindrance to nutrition (Rindfleisch[2]), or from the rank growth of connective tissue (Rokitansky); (3), the increase and subsequent retrograde metamorphosis of connective tissue. Recently it has been asserted by Dr. Tigges, that there is, even at an early stage, an increase of nuclei in the ganglionic cells; the numerous scattered nuclei, usually

[1] Des Affections Nerveuses Syphilitiques, par Dr. Léon Gros et E. Lancereaux, 1861. Ueber die Natur der Constitutionell-syphilitischen Affectionen, von Rudolf Virchow; Archiv, B. xv. Das Syphilom, oder Die Constitutionell-syphilitische Neubildung, von E. Wagner; Archiv der Heilkunde, 1863. Ueber Constitutionelle Syphilis des Gehirns, von Dr. Ludwig Meyer; Zeitschrift f. Psychiatrie, 1861. Des Affections Nerveuses Syphilitiques; M. Zambaco. Wiener Medizinal-Halle Zeitschrift, February, 1864; Dr. Leidesdorf. Zeitschrift f. Psychiatrie, 1863; Dr. Westphal.

[2] Dr. Bastian's recent researches seem to prove that the specific gravity of different parts of the gray matter differs considerably in health. "On the Specific Gravity of the Human Brain;" Journ. Ment. Science, January, 1866.

[1] Ueber Bindegewebeswucherung im Nervensysteme; Rokitansky, 1857. Wedl, Beiträge zur Pathologie der Blutgefässe; Wien, 1859.

[2] Histologisches Detail zu den grauen Degeneration von Gehirn und Rückenmark. Von Dr. E. Rindfleisch. Virchow's Archiv, B. vi.

thought to belong to connective tissue, he considers to have escaped from ganglionic cells at a later stage of their inflammatory degeneration.[1] Such changes are not peculiar, as some have said, to general paralysis; like changes have been described by Wedl[2] in the brains of three congenital idiots, and have been met with in dementia following on long-continued Insanity, and in tabes dorsalis; and there can be little doubt that the morbid product in syphilitic dementia is of a similar nature.

Briefly summed up, then, the kinds of degeneration met with in the brain, after Insanity, are as follow: 1. There is in acute insanity an acute hyperæmia, or the early stage of *inflammatory degeneration*. 2. There is that degeneration which consists in the increase of connective tissue, and in the atrophy of the nervous elements, and which might be called *connective tissue degeneration*. Whether called sub-inflammatory or not, is not of much moment, so long as we keep in mind the true relations of organic element to the supply of blood, and the degenerate nature of inflammation, whether acute or chronic. 3. *Fatty degeneration* takes place not only in the small vessels, as in atheroma, and in the new morbid products, but also in the broken-up nervous elements, and even in the nerve-cells. 4. The *amyloid degeneration* is undoubtedly pathological. Wedl holds that the amyloid corpuscles should be ranked along with the so-called colloid bodies, and viewed as the result of an increased exudation that may take place without hyperæmia. Rindfleisch, on the other hand, believes that he has traced their production from the nucleated connective-tissue corpuscles; while some, like Rokitansky, maintain that the ganglionic cells themselves are converted into colloid bodies. 5. *Pigmentary degeneration* is sometimes met with. In senile atrophy the ganglionic cells are sometimes richly filled with brown pigment molecules; and, in one case of dementia, where there was partial paralysis of the tongue, Schroeder van der Kolk found the cells forming the nuclei of the hypoglossal nerves in such a state of blackish-brown degeneration that he at first mistook them for little points of blood, but they were filled with granular, dark-brown pigment. Dr. Lockhart Clarke has observed similar pigmentary degeneration of the cells in general paralysis. It is worthy of remark, that cases of pigmentary degeneration of the retina are often found to occur in the same family, to be accompanied with general defective development, mental and bodily, occasionally with mutism and Cretinism, and to occur in those who, like albinos, are sometimes the degenerate offspring of marriages of consanguinity.[1] 6. *Calcareous degeneration* is common enough in the hypertrophied connective tissue and in the small bloodvessels; and it has been observed in the ganglionic cells themselves. Erlenmeyer met with calcified cells in the optic commissure of a monomaniac; Förster figures calcified cells found in the gray substance of the lumbar enlargement of a boy whose lower extremities were paralyzed; Heschl found what he calls ossified cells in the brain of a patient who died melancholic; and Wilks believes certain bodies, found in the brain of a general paralytic, in whom the small arteries were calcified, to have been ganglionic cells that had undergone calcareous degeneration.

Those who duly weigh the pathological import of the kinds of degeneration enumerated, who reflect on the great gap which there is between a calcareous granule and a nerve-cell in the economy of nature, or between a connective-tissue corpuscle and a nerve-cell in the histological scale of life, must admit that the difference is not less great than that between dementia and sound mental action, and will scarce venture to assert that the morbid appearances throw no light whatever upon the nature of Insanity. Even the comparatively slight signs of hyperæmia are of weighty significance, if their true relations are recognized, if they are viewed as results and evidence of that degeneration of individual nervous element, of which the mental disorder is also result and evidence, if they and the Insanity are viewed as, what they often are, concomitant effects of a common cause.

PROGNOSIS.—Two questions at once present themselves: the first, whether the disease directly endangers life; the second, and perhaps more solemn one, whether there is any prospect of recovery. Respecting the first question, it may be said in general terms that Insanity does certainly reduce the mean duration of life, and much more so in its recent acute forms than in its more chronic forms. Of all forms, general paralysis is the most fatal, other varieties not being, as a rule,

[1] Zeitschrift für Psychiatrie, B. xx. In Virchow's Archiv, 1865, Dr. Franz Meschede has striven to prove that hyperæmia and parenchymatous swelling of the inner layer of the cortical substance are the beginning, and fatty degeneration the end, of the organic changes in general paralysis.

[2] Histologische Untersuchungen über Hirntheile dreier Salzburger Idioten. Von Prof. C. Wedl; Medizinische Jahrbücher der K. K. Gesellschaft der Aerzte in Wien. 1863. Heft 2 und 3.

[1] On Retinitis Pigmentosa, by J. Laurence; Ophthalmic Review, No. 5, April, 1865. The observations of Prof. Graefe and Liebreich are quoted.

directly dangerous to life. Still, certain cases of acute mania and acute melancholia do terminate suddenly in death, owing probably to exhaustion, and it may be utterly impossible to say beforehand whether they are going to do so or not. When the temperature of the body rises several degrees above the natural standard, the prognosis is bad; and any indication of motor paralysis, or any kind of hybrid epileptiform convulsion, in the course of the disease, is of evil omen, while an attack of genuine epilepsy, unfavorable as regards recovery, is not so as regards life. A long-enduring refusal of food may sometimes end fatally, both in mania and melancholia.

What prospect there is of recovery in a particular case will depend greatly upon the cause of the attack, upon its form, and upon its duration. The more recent the outbreak, the better the chance of recovery, the expectation of which from proper treatment adopted within three months from the commencement is about four to one, while it is less than one to four after twelve months' duration of the disease. Certainly there do occur instances in which patients recover after being insane for years, but they are exceptional. When the stage of secondary dementia, incoherent or apathetic, has been reached, all hope of recovery is gone. Primary dementia is generally curable.

Of the acute primary forms of mental disease, melancholia is more curable than mania, although some have thought otherwise, deceived probably by the experience of an asylum into which simple cases of melancholy do not usually come. Next to melancholia acute mania is most curable; when attacks of melancholia and mania alternate, the prognosis is very unfavorable. A day of great depression and weeping intervening in the course of acute mania, is of good omen. When the maniacal fury is subsiding, the prospect is good if the patient is sad and depressed, begins to inquire about his family, and to show other signs of a return to his former feelings; it is bad if the feelings remain unmoved, and the intellect is calm in its disorder—if, in other words, there is evidence of the organization of disorder. Even the disappearance of intellectual disorder is not a certain sign of recovery, unless there is a return to the old healthy feelings, and the patient recognizes that he has been mad. A periodical recurrence of attacks of Insanity, with long intermissions, is of decidedly unfavorable augury; the attacks commonly become longer, the intermissions briefer, and the outlook gets more and more gloomy.

Monomania is far less curable than mania, the fixed delusions marking the establishment of a definite type of morbid action of a chronic nature. Recovery does sometimes take place under the influence of systematic moral discipline, or from a great shock to the system, whether emotional or produced by some intercurrent disease. In melancholia, where there is a fixed delusion that the cause of misery is in some external agency, the prognosis is unfavorable; but it is more favorable in the melancholic who attributes his affliction to his own imaginary backslidings. In like manner the homicidal melancholic, who believes himself the victim of persecution, seldom recovers; the suicidal melancholic, who is not so in consequence of any definite delusion, frequently does recover, especially after some serious and almost successful suicidal attempt.

In moral insanity the prognosis is bad; for these symptoms usually mark the tyranny of a bad organization. General paralysis may be pronounced incurable.

When Insanity has been slowly developed, the prognosis is more unfavorable than when it has been of sudden origin—this probably being a part of the larger truth, that when Insanity is slowly developed it is produced by the egoistic passion of some peculiarity of character, as pride, ambition, avarice; but when it is suddenly caused, it is by the shock of an altruistic emotion, as, for example, grief about others. For a like reason a frequent alternation of active symptoms is more hopeful than a steady persistence in a particular group of quiet symptoms. Hereditary insanity is generally deemed most unfavorable, but recent researches prove that it is not so much so during a first attack, although the disease is more liable to recur than when not of hereditary origin. In the acute mania sometimes produced by drunkenness we may reasonably look for recovery, but by no means so in those cases in which a continued intemperance has resulted in mental weakness, loss of memory, and loss of energy of character. When Insanity has been caused by habits of self-abuse or by sexual excesses, the prognosis is unfavorable in all but the earliest stages. If religious excitement purely has been the cause of an outbreak, the prognosis is most favorable; but it is necessary to bear in mind that a form of religious insanity is the vicarious satisfaction of unsatisfied sexual love, that more or less nymphomania is oftentimes associated with it, and that the outlook then is hopelessly bad. When disease of brain, or injury of the head, or epilepsy, has been the cause, the derangement is practically incurable; but where it occurs during the decline of some acute disease, it is generally soon curable. The prognosis is bad in Insanity after sunstroke; nevertheless Dr. Skae mentions one case under his care in which

recovery took place, being one of the very few recoveries on record from Insanity produced by this cause. The prognosis is favorable in hysterical insanity; as it is also in puerperal mania, in the insanity of pregnancy, of lactation, and of the climacteric change. A decidedly bad symptom is a fixed hallucination, as is also a complete preservation of bodily health with persistence of mental disorder; when there is palpable bodily disorder, as digestive disturbance, anæmia, menstrual irregularity, there is good hope that with the restoration of bodily health the mind may be restored also.

When Insanity has followed the suppression of an eruption or an accustomed discharge, the prognosis is favorable; when associated with phthisis, it is unfavorable as regards recovery and as regards life.

The most favorable age for recovery is youth, the probability of it diminishing with the advance of age, and few recovering after fifty; as many as 86 per cent. of males and 91 per cent. of females, attacked with mania under twenty years of age, recovered at the Somerset Asylum. This proportion will appear the more favorable when we reflect that children under ten or twelve years of age do not recover from mental derangement, it being often dependent upon defective organization, and associated with epilepsy. The recoveries amongst women exceed those amongst men, by reason probably of the frequency and fatality of general paralysis among men.

The general conclusion of Dr. Thurnam from his careful statistics is that, "as regards the recoveries established during any considerable period — say twenty years—a proportion of much less than 40 per cent. of the admissions is under ordinary circumstances to be regarded as a low proportion, and one much exceeding 45 per cent. as a high proportion." The liability to recurrence of the Insanity after recovery from the first attack cannot, he thinks, be estimated at less than 50 per cent., or as one in every two cases discharged recovered. On the whole, then, he holds that, of ten persons attacked, five recover and five die sooner or later. Of the five recoveries, not more than two remain well during the rest of their lives; the others have subsequent attacks, it may be after long intervals, during which at least two of them die.[1]

THERAPEUTICS.—The treatment of Insanity is moral and medical, the two methods properly being combined. It must again be *individual*, as the case is; for the varieties of the insane character demand different moral means, as the varieties of causes call for different medical treatment. It is necessary to penetrate the individual character, with the design of influencing it beneficially, and carefully to investigate the concurrence of conditions that have issued in Insanity, with the object of removing them. Herein lies the chief difficulty of treatment; in no other disease are there so much concealment and so much misrepresentation, witting and unwitting, on the part of friends. It is before all things necessary again that treatment should be begun early, before the habit of a definite morbid action has been fixed; but, though early, it must not be rashly vigorous and energetic, with the aim of effecting any sudden revolution, but rather patient and systematic, in the hope of a gradual change for the better. Whilst in other diseases time is reckoned by hours and days, it must in Insanity be reckoned by weeks and months.

Moral Treatment.—To remove the patient from the midst of those circumstances under which Insanity has been produced, must be the first aim of treatment. There is extreme difficulty in treating satisfactorily an insane person in his own house, amongst his own kindred, where he has been accustomed to exercise authority, or to exact attention, and where he continually finds new occasions for outbreaks of anger or fresh food for his delusions. An entire change in the surroundings will

[1] In an elaborate paper on Vital Statistics and Observations, in the Journal of Mental Science, January, 1865, Dr. Boyd records the statistics of the Somerset Asylum for many years:—

	Of 1000 Males.	Of 1000 Females.
Recovered	252	276
Relieved	55	79
Not improved	47	35
Died	324	258
Remaining	192	223
	870	871
Re-admissions	130	129
	1000	1000

The cause of death in 539 cases out of 2000—295 males and 244 females—in which post-mortem examinations were made, were as follows:—

	Males.	Females.
Diseases of Respiratory organs in	148	104
" Nervous system	112	73
" Digestive organs	18	41
" Vascular system	11	18
" Genito-urinary	2	1
" Locomotor organs	1	3
" Fevers	0	2
" Accidents	3	2
	295	244

The diseases of the respiratory organs which proved fatal were principally pneumonia and phthisis.

sometimes of itself lead to recovery; if the patient is melancholic, he no longer receives the impressions of those whom, having most loved when well, he now most mistrusts, or concerning whom he grieves that his affections are so much changed; if he is maniacal, he is not specially irritated by the opposition of those whose acquiescence he has been accustomed to, or encouraged by their submission to his whims and their indulgence of his follies. Travelling may be recommended in the early stages, in order to secure change of place and scene; or if the patient cannot travel, he may be removed from his own home to another residence, and systematically treated there. If the pecuniary means do not admit of the adoption of either of these expedients, or if the patient is furious, or desperately suicidal, or persistently refuses food, it will be necessary to send him to a suitable asylum. It must be borne in mind that an insane person cannot, from the very nature of his disease, have his own way, and that to allow him to do so would be directly to aggravate his disease. To put him under restraint in some way, to exercise for him that control which he cannot exercise for himself, is indispensable so long as there is a hope of cure: to let him distinctly understand that this is legally done will of itself have a beneficial effect. The melancholic who finds himself in an asylum finds a real grief to alternate with or perhaps to take the place of his fancied affliction; and the maniacal patient, feeling his wild spirit of exultation to be rudely checked by the influence of a systematic control, can scarce fail to have more sober reflections aroused. It is extremely objectionable except in an emergency to entrap a patient into an asylum; there should be no deception about the matter, if possible, but all should be done openly and firmly, in the spirit in which an act of obedience is inculcated upon a child, and in any case inflexibly insisted upon.

The patient having been removed from those influences which have contributed to the production of the disease and tend to keep it up, and having been made to recognize from without a control which he cannot exercise from within, it remains to strive persistently and patiently by every inducement to arouse him from his self-brooding or self-exaltation, and to engage his attention in matters external—to make him step out of himself. This is best done by engaging him earnestly in some occupation or in a variety of amusements; and this will be more easily done now that the surroundings are so entirely changed. The activity of the morbid thoughts and feelings subsiding in the new relations and under new impressions, more healthy feelings may be gradually awakened; and the activity of healthy thought and feeling will not fail in its turn further to favor the decay of morbid feeling. It is not by arguing against a delusion or directly contradicting it that any good will be done: it were almost as well to argue against the east wind or a convulsion; but by engaging the mind in other things, and substituting a healthy energy for the morbid energy, this will be most likely to abate and finally die out. But though it is of little use talking against a delusion, it is desirable to avoid agreeing with it: by quiet dissent or a mild expression of incredulity when it is mentioned, the patient should be made to understand clearly that he is in a minority of one. It is most necessary to avoid any harsh word or act, a tone of ridicule, or a look of disgust; an angry speech will often be remembered when the frenzy has passed off, and will leave a sore feeling in the patient's mind. It is a mischievous and silly practice too to speak to insane persons as if they were babies: they are often more sensitive to such impertinences than sane persons, while they seldom fail to be influenced beneficially by a sincere, sensible, and sympathetic address.

Medical Treatment.—There is no specific agent in the treatment of any form of Insanity. A truly scientific treatment will be grounded on the removal of those bodily conditions which may appear to have acted as causes of the disease and on the general improvement of nutrition. An attack of melancholia occurring in a gouty subject, and perhaps taking the place of an attack of gout, is sometimes cured by the treatment proper for gout; and there are cases on record in which acute mental derangement has affected persons living in a malarious district in regular tertian or quartan attacks, and has been cured by quinine. The morbid sensations, so common in Insanity, should not be overlooked, as they often arise from some real bodily derangement, and tend to keep up the delusion. Now, bodily disease is not always easily detected in the insane; for the usual symptoms are very much masked, and they, like animals, make no intelligent complaint. "Insanity," says Dr. Bucknill, "is not confined to the brain, and when it is confirmed, a man becomes a lunatic to his finger ends." It is necessary, therefore, to examine carefully into the state of the different bodily functions and to pay particular attention to the physical signs of disease: there may be no cough, no expectoration, when the thermometer or the stethoscope reveals advancing phthisis.

General bloodletting is now rarely, if ever, used; even in the most acute and seemingly sthenic insanity it is not simply useless, but it is positively pernicious. Violent symptoms may abate for a time,

but the disease is very apt to become chronic and to pass rapidly into dementia. Local abstraction of blood by leeches or by cupping may be useful where there appears to be great determination of blood to the brain; by withdrawing blood from the overloaded vessels the opportunity of rest is afforded to the struggling and suffering nervous element. It may be well to add here that in Insanity it is not judicious but mischievous to shave the patient's head, to confine him to bed, to shut out the light, and to treat him in all ways as if he were suffering from an acute inflammation of the brain or its membranes; on the contrary, in almost all cases it is necessary to enjoin abundant exercise in the open air.

The continued application of cold to the head by means of a douche pipe, or by pouring cold water upon it, while the patient lies in a warm bath, is often successful in calming excitement and in procuring sleep in acute insanity. The warm bath alone, taken for about half an hour, has a soothing effect, and may induce sleep; and its efficacy has been supposed to be wonderfully increased by the addition of several handfuls of mustard, so as to produce a general redness of the surface of the body. Brierre de Boismont professes to get very good results from employing the warm bath for eight or ten hours at a time; and Leidesdorf has used for three or four hours, and in many cases with marked calming effect, a bath constructed by Prof. Hebra, in which patients may be kept night and day at a definite temperature. Such a use of the bath must obviously be avoided where the pulse is feeble and where there is anything like commencing paralysis, and it is of no avail in cases of chronic insanity. The prolonged use of the shower bath and of the cold bath, at one time much in fashion, is now justly abandoned. The shower bath or cold douche may certainly be usefully employed in certain cases of melancholia, where reaction does not fail to take place after it, and in cases of chronic insanity, with the purpose of rousing the patient and giving tone to the system; but its use should never be continued for more than three minutes, and it should not be with the aim of producing any special effect, but on general principles of improving the bodily health. Pouring water on the head from a hand shower bath, or a common garden watering-pot, or sponging the forehead and face with cold water, is refreshing and grateful to the patient suffering from acute excitement, and has sometimes a good effect beyond what might be expected from a measure so simple. The virtues of the Turkish bath were at one time much vaunted by its advocates, but an exact discrimination of the cases in which it is useful yet remains to be made. Packing in a wet sheet has sometimes a beneficial effect, and is commonly rather grateful to the patient.

Counter-irritants are not much used now in Insanity. Schroeder van der Kolk, however, put much faith in the application of strong tartar-emetic ointment, or of a blister to the shaven scalp; and Dr. Bucknill has found it useful to rub croton oil into the scalp in the passage from acute to chronic insanity or dementia, and in chronic melancholy with delusion. Blisters to the nape of the neck appear to have little other effect than to increase mental irritation; and the benefit of setons and issues in the neck is very problematical.

After errors of digestion and secretion have been duly attended to, the diet of the insane should be good; and it will be desirable in most chronic cases, and in many acute cases, to allow a moderate use of wine. There can be little doubt that an attack of Insanity might sometimes be warded off by a generous diet and free use of wine at a sufficiently early stage. It is at any rate a truth worthy of all acceptation, that energetic antiphlogistic treatment in the course of Insanity is energetic mischief. Leeches may be applied to the head, and a patient may be kept on low diet, in order to subdue maniacal excitement, without any other result than an increase of the excitement with the increase of exhaustion; and the most active purges may be given, and given in vain, to overcome an obstinate constipation, — when brandy and beef-tea, reducing exhaustion, will subdue excitement, and a simple enema will produce full action of the bowels. [*Per contra*, the following is from Dr. Forbes Winslow:[1] "In Insanity, when the symptoms are acute, the patient young and plethoric, the habitual secretions suppressed, the head hot and painful, the eyes intolerant of light, the conjunctiva injected, the pupils contracted, the pulse rapid and hard, and the paroxysm sudden and rapid in its development, local bleeding will often arrest the progress of the cerebral mischief, greatly facilitate the operation of the other remedies, and ultimately promote recovery. In proportion as the symptoms of ordinary insanity approach those of phrenitis, or meningitis, shall we be justified in the use of depletion. Although it is only occasionally, in instances presenting peculiar characteristic features, cases occurring in the higher ranks of life, where the patient has been in the habit of living *above par*, and is of a sanguineous temperament, that we are justified in having recourse to general depletion, there is a class of recent cases

[1 Obscure Diseases of the Brain and Mind, 2d American edition, p. 424.]

presenting themselves in the asylums for the insane, both public and private, in the treatment of which we should be guilty of culpable and cruel negligence, if we were to omit to relieve the cerebral symptoms by means of the *local* abstraction of blood." —H.] Active purgation, once so much favored, is now quite eschewed in all forms of Insanity. The bowels may generally be regulated by dietetic means; and if a purge is needed, a dose of aloes, rhubarb, or castor oil will answer every purpose—a moderate dose of the last often succeeding where the most drastic purgatives fail. The state of the bowels ought, however, to be carefully attended to, for melancholia has sometimes been produced by habital constipation, and is always made worse by it. A mixture containing sulphate of magnesia, sulphate of iron, with a little quinine and sulphuric acid, will be found very useful. It will, of course, be necessary to be guided by the bodily state of the patient, and by the history of the causation of the disease, as to whether wine is given or not in the most acute stage; it may be desirable in cases of a sthenic type to do nothing but wait patiently, only preventing the patient from doing harm to himself or others, until the fury of the storm has passed, and then to give support.

Amongst drugs useful in Insanity, opium undoubtedly occupies the first place. It is especially useful in that state of mental hyperæsthesia which so often precedes the outbreak of Insanity: when the mental tone is so changed that almost every impression is painful, then opium, freely administered, has virtues which can scarce be exaggerated. It is useful, again, in cases of simple melancholia, when it should be given in doses of one, or even two, grains twice a day, and continued steadily for weeks, notwithstanding an apparent want of success at first. In these cases it does not usually produce constipation; but if it does, then each dose may be combined with a grain of extract of aloes or a quarter of a grain of podophyllin. Where there is fixed delusion of some standing, it is not of much use, except as an occasional expedient for procuring sleep.

As opium agrees better with persons of melancholic than with those of sanguine temperament, so it appears to be more useful in melancholic than in maniacal forms of Insanity. The early writers on Insanity condemned the use of opium in mania, because of the exaggeration of the mental symptoms which they observed in some cases after sleep occasioned by it. Certainly there are cases of acute mania in which, after two or three hours of such sleep, the patient seems only to have gained new vigor for a fresh start in frenzy, and in which it is impossible to help feeling that the drug has done harm rather than good. But there are, on the other hand, cases in which there can be no reasonable doubt of the benefit conferred by opium. These are the cases in which there is no heat or congestion of the head, but where the face is pale, the pulse weak, and where a restless activity and incoherence are accompanied with want of sleep. In the mania caused by intemperance, in the mania or delirium of nervous exhaustion, and in puerperal mania, opium is beneficial; but it is of little use in melancholia with stupor, in idiopathic sthenic mania, or in the attacks of excitement that occur in the course of chronic mania and general paralysis.

In cases of great excitement, maniacal or melancholic, where opium does no good, large doses of digitalis sometimes produce the best effects, especially where there is general excitement of the circulation. The excitement abates, and the pulse, falling in frequency, may be kept for a long time at a standard below the average. In the attacks of excitement which occur in the course of general paralysis, the effects of digitalis are excellent; a single dose of two drachms, or continued doses of half a drachm every three or four hours, being safely given. Hydrocyanic acid may be usefully combined with it; and some entertain a high opinion of the good effects of this acid when given by itself.

The subcutaneous injection of morphia is a valuable expedient to have recourse to, where there is a refusal to take medicine, and it usually operates much more effectually than opium taken by the mouth. Not more than a quarter of a grain should be injected to commence with, and the quantity may subsequently be increased, if necessary. It will be well to have in mind, that neither opium by the mouth, nor morphia hypodermically injected, will quench the fury of acute mania, and that successive doses of opium or successive injections of morphia, followed by brief snatches of fitful sleep, have been followed also by fatal collapse.

It is not yet possible to speak positively and definitely of the virtues of chloral as a remedy in Insanity. So far as my experience reaches, it is that, given in doses of 30 grains, repeated in an hour or two if necessary, it will produce sleep in most cases of Insanity, but that permanent good follows the sleep only in those asthenic cases in which opium does good. There can be no question that we have in this drug a useful hypnotic, but, it is another question whether hypnotics will stay the course of acute Insanity.

Hyoscyamus is useful also in some cases where opium does not agree, but it should be given in doses of not less than a drachm to begin with. Tartar emetic will often

calm for a time the most furious maniac, through the prostration which it produces, but it does no permanent good, and its employment for such purpose is rather a relic of the old system of quieting a patient by some violent means or other, short of actually killing him. If mercury be ever useful, and not mischievous, in the treatment of Insanity, it is when given in small doses of the perchloride, in cases that are becoming chronic, or where there is a suspicion of syphilis. To administer mercury systematically in general paralysis, as has been done, is as unaccountable in theory as it is undoubtedly pernicious in practice.

In hysterical mania, in epileptic mania, and in mania connected with sexual or uterine excitement, I have never seen any good from the use of opium. In all these forms of mania, though least markedly in epileptic mania, I have, however, seen benefit from the employment of bromide of potassium, with or without tincture of henbane. In one case, that of a widow of sanguine temperament and active habits, who suffered from acute chattering mania, connected seemingly with uterine excitement, recovery took place within a fortnight under the use of bromide of potassium and henbane. Exactly a year afterwards she had an exactly similar attack, when the same treatment was successful in a short time.

In all those cases of Insanity in which tonics seem to be demanded by the state of the bodily health—and they are the majority of cases, at one period or other of their course—iron and quinine may be given; and one of the best ways of giving them is in a mixture containing quinine, the tincture of the sesquichloride of iron, and chloric ether. In some cases it happens that an uncontrollable diarrhœa sets in and carries the patient off, nothing availing to check it: acetate of lead, with opium, and enemata of starch and laudanum, are most likely to be useful.

When Insanity has become chronic, or when fixed delusions are established, there is small hope of special benefit from drugs. The general health being duly attended to, a systematic moral treatment will be best adapted to restore health of mind. Where there is persistent refusal of food, it must never be allowed to continue so far as to endanger the bodily health; and if persuasion entirely fail, then the stomach pump must be used to administer food, or this may be given by a tube passed through the nostril. Those who are suicidal should be carefully watched at all times, and especially so on getting up in the morning, when the thoughts are gloomy, and the desperate impulse is apt to surprise and overpower them. The monomaniac, who has delusions that he is watched continually, or otherwise persecuted, must always be deemed dangerous to others; for at any time he may become so impatient of his sufferings as to make a fatal attack upon his fancied persecutor. Those who suffer from moral Insanity are often very troublesome to deal with satisfactorily; but it will be worth while always to remember that one unequal to the responsibilities and duties of the social position in which he was born may not on that account be unequal to the relations of a much lower social stratum. It is not because a person insists on ruining himself that it is justifiable to deprive him of liberty by sending him to an asylum.

In all forms of Insanity, it is most necessary to look beyond the mental symptoms, whether these be symptoms of excitement or of depression, to examine closely the physical symptoms co-existing with the mental perversion, and to direct the medical treatment to the nature of the bodily disease which will sometimes be found to be at the root of the whole disorder. Neither opium, nor henbane, nor digitalis, nor any other drug, will act as a specific in any kind of mental derangement; and it is vain to hope and a folly to attempt to get rid of the disease by merely stifling its prominent symptoms. A rational method of treatment must be based on a careful inquiry into the patient's previous history and into the origin of his disease, and on a faithful study of all the symptoms, bodily and mental, which it presents.

[As connected with the general principles of management of Insanity, the subject of *restraint* may be here alluded to. Reacting from the barbarous measures common until near the end of the last century, when Pinel, in France, led the movement for their abolition, the tendency in England of late years has been toward the prohibition of all bodily confinement, except that of secure apartments and the vigilance of trained attendants. Non-restraint has thus, especially by Drs. Hill and Conolly, been made an absolute rule. In America this idea has not been accepted without qualification by the best alienists. All admit that a *minimum* of restraint is the aim to be constantly held in view. But men of large experience—Dr. Isaac Ray, for example—still hold that cases occur, although exceptionally, in which the temporary use of bodily restraint is a measure not only of safety, but of real advantage to the patient. At the same time, American hospitals for the insane compare favorably with those abroad in their provision of arrangements tending to keep out of sight and use, as far as possible, everything which abridges the liberty and ease of the patient; "moral treatment" being maintained by the aid of liberal resources for mental occupation and diversion, of wholesome kinds.—H.]

HYPOCHONDRIASIS.

By William Withey Gull, M.D., D.C.L., F.R.S., and Francis Edmund Anstie, M.D., F.R.C.P.

Definition.—A disease of the nervous system, of unknown and possibly varying seat. It is markedly hereditary, being one of the transformed neuroses which descend from a parent stock strongly tainted with insanity. Its principal feature is mental depression, occurring without apparently adequate cause, and taking the shape, either from the first, or very soon, of a conviction in the patient's mind that he is the victim of serious bodily disease. The sufferer's belief in this disease is so firm, that he describes minutely the symptoms which, as he fancies, indicate its existence. But he may place the imaginary malady in almost any organ of the body, and he usually describes some symptoms which are anomalous, or even incredible. Finally, Hypochondriasis may be evoked by a real organic disease acting as an irritant to an hereditarily predisposed nervous system: in this case, the anomalous nervous symptoms may mask, and even conceal, the occurrence of serious changes in some viscus.

Nomenclature.—The same name has been applied to the disease since the days of Hippocrates: it has always been known as "Hypochondriasis," or the "hypochondriac disorder," and sometimes as the "vapors," or the "spleen;" but these last two synonyms are, in fact, mere explanations of the ancient hypothesis which was expressed in the word Hypochondriasis. This hypothesis it is really important to say a few words about, since the vulgar conception of the disease still, though unconsciously, cherishes it; although our improved knowledge of the relation of the nervous system to the organism generally has now made it an anachronism.

The words ὑποχονδριακὸν πάθος, applied by Hippocrates and Galen to the disease, imply a belief that the viscera behind the xiphoid cartilage, and below the diaphragm,[1] were its seats; and Galen very distinctly says that *black bile* is its cause. It is worth while to recall for a moment the physiological ideas which Galen, with great ingenuity, had compounded from the speculations of Plato on the one hand, and Hippocrates on the other. According to Galen, the functions of the human body were maintained by three πνεύματα (whence, remotely, our "vapors"). The lowest of these was the πνεῦμα φυσικόν, and developed the *natural force* in the liver; the second was the πνεῦμα ζωτικόν, which elaborated the *vital force* in the heart; and the third and highest was the πνεῦμα ψυχικόν, which developed the *animal* or *soul force* in the brain.[1] Any one who has been curious enough to investigate these questions will see at once that the lowest or "natural" force of Galen is the counterpart of that lowest kind of *mortal soul* which[2] Plato represented as residing in the abdominal organs, and chiefly in the liver, and as having to do with the baser animal passions and the supply of the needs of vegetable life. The ancient tendency to view every source of functional activity as an entity—almost a personality—made it quite consistent for the long succession of Galenist physicians to endow the liver-force with a quasi-consciousness and perception, and even with voluntary activity, though of a low kind; and, on the other hand, the Paracelsian and Helmontian doctrine of the *Archæus* rather added to than changed anything in the extraordinary power over the general organism which was attributed to the abdominal organs. Then the absence of any accurate knowledge of the functions of a central nervous system, the recipient of sensory impressions, and the originator of motor acts, induced men to localize in the various organs the source of the functional disturbances which appeared to be manifested therein. The vagaries of hypochondriacal sensation were therefore, in the ancient view, the perturbations of the natural force generated in the liver, spleen, and pyloric part of the stomach. It is to be remarked, moreover, that Hypochondriasis was very generally confounded with hysteria (to which it doubtless has a relation), quite down to the present cen-

[1] The viscera of the hypochondria, to which the ancients attached such importance, seem to have been especially the liver, the *pyloric part* of the stomach, the omentum, the mesentery, and the spleen. The stomach considered as a whole they regarded rather lightly.

[1] De Loc. Affect. v. De Usu Part v. De Usu Resp. 163, 164.

[2] Timæus, Ed. Stallbaum, §§ 69, 70.

tury. There is nothing surprising in this. The flatulence which is frequently a striking symptom both in hysteria and Hypochondriasis was, for the ancients, a commotion of the natural spirits which resided in the abdomen.

Of the long list of authors who have treated of Hypochondriasis, since the days of Galen, there is scarcely one who viewed the disease in any essentially different light from that in which Galen regarded it, until we come to Thomas Willis, the great investigator of the nervous system. It is easy to see what were the common ideas on the subject at the time by consulting that curious book, Burton's "Anatomy of Melancholy," which was published a few years after Willis's death, and which represented the knowledge which a learned and clever layman might pick up without knowing, or without demeaning himself to notice, the writings of a contemporary countryman. Burton says that the general view of authors represents the hypochondriac or windy melancholy as arising "from the bowels, liver, spleen, or membrane called *mesenterium*," and quotes Laurentius as dividing it into three kinds, the *hepatic*, the *splenetic*, and the *mesaraick*.

Willis,[1] on the other hand, placed Hypochondriasis amongst the diseases proper to the *diathesis spasmodica;* he made it an affection of the nervous system, but so far conformed to the old ideas as to attribute its ultimate causation to impurity of the splenic blood. In the next century, Flemying[2] ventured a more distinct opinion, that the brain was the part primarily affected; and Cullen[3] and Robert Whytt[4] (especially the former) placed great stress on the share which the nervous system has in the production of the disease. The next prominent step was taken by Georget[5] (1819), who protested against the view (at that time still commonly prevailing) of the abdominal origin of Hypochondriasis, and the practice of powerful purgation, &c., which was based upon it. But the most complete and effective attack on the old view was made by J. Falret,[6] in 1822. This author dwelt strongly on the hereditary character of the disease, and the great frequency with which it is immediately excited by stress of intellectual labor, or by moral and emotional causes. The view of Falret was perhaps carried to excess in limiting the primary seat of the disease so strictly to the brain; but it has prevailed, and Hypochondriasis is now commonly placed among the varieties of Insanity. Griesinger, for instance, in his admirable treatise on mental diseases,[1] makes Hypochondriasis a variety of melancholia, which is his first class of "mental diseases characterized by depression;" and Leidesdorf[2] adopts the same view. It will be seen that the view which we hold differs in some degree from this; but there can no longer be any doubt that the true seat of the disease is in the nervous centres.

History.—The history of a hypochondriac patient is that of his nervous system under the two aspects of its congenital form and the influences—of nutrition, education, and emotion—to which it has been subjected. So far, it is not different from the story of the sufferer from any other form of nervous disease. But Hypochondriasis is distinguished by this—that a more important part is played by the congenital disposition of the nervous system, and a less important part by the physiological and spiritual influences which have been brought to bear on it, than is the case with the majority of nervous diseases. It comes nearest, in this respect, to insanity on the one hand, and to epilepsy and neuralgia on the other. It is the almost *inevitable* inheritance of a certain percentage of the descendants of any individual who may be very strongly tainted with insanity. On this subject we shall say no more till we can discuss more fully the etiology of this singular disease.

Symptoms.—This phrase is pre-eminently appropriate to the phenomena of Hypochondriasis. Of physical signs we have almost none to guide us; and this is in perfect agreement with the position which this disorder holds in the category of diseases generally. All is in the region of symptoms. For the most part, too, the symptoms are subjective only: still there are features which the experienced physician can detect, and which can hardly be simulated by a malingerer.

The most important external feature of Hypochondriasis is this: that without any sufficient reason for such conduct, and without any signs of intellectual insanity,

[1] T. Willis, Opera Omnia, 4to. Genevæ, 1676. The whole treatise, De Morbis Convulsivis, and that on Hysteria and Hypochondriasis (in reply to the strictures of Nat. Highmore), are astonishing efforts of genius, and will well repay perusal in the present day.

[2] Neuropathia; sive de Morb. Hypochond. et Hyster. Ebor. 1744.

[3] Clinical Lectures. London, 1777, pp. 39—57.

[4] Observations on the Causes, Nature, and Cure of the Disorders called Nervous, Hypochondriacal, &c. London, 1777.

[5] De la Phys. du Syst. Nerv. Paris, 1819.

[6] De l'Hypochondrie et du Suicide. Paris, 1822.

[1] Die Path. und Therap. der psych. Krankheiten. 2d edit. Stuttgart, 1861.

[2] Path. und Therap. der psych. Krankheiten. Erlangen, 1860.

the patient is observed to concentrate his attention on some particular organ of his body, and to fancy that it is seriously diseased. This concentration of attention is often preceded and accompanied by notable depression or variability of his spirits, with a tendency, on the whole, to depression: this is not always the case, however, for there is sometimes no antecedent symptom connected with the general mental state. In many instances the patient's first sufferings take the form of what he himself considers dyspepsia, but which is in fact little more than flatulence, from the formation of large collections of gas in the stomach and bowels. Along with this flatulence, there are sometimes appearances which give a superficial color to the idea of a primary stomach derangement: the tongue, for instance, is often pasted and coated, and there may be foul breath; the appetite is not unfrequently ravenous, capricious, or well-nigh lost; there is generally obstinate constipation; in rare cases there are even attacks of vomiting.

More commonly there is an antecedent mental change, the character of which it is at first difficult to seize, and which forms one of the grounds for the modern practice of including Hypochondriasis in the varieties of actual insanity. Before any local symptoms have declared themselves, the patient has already become changed in his disposition; in most cases, perhaps, the change is simply in the direction of despondency or vague alarm, for which he can give no reason. It is remarked by alienists that the mental condition, even thus early, is characterized above all things by an expansion of the *self-feeling*, a pre-occupation of the patient with his own condition, to the exclusion of other interests and affections. This is true; but it appears to us that the self-concentration is more like that of a person in the preliminary stage of an acute inflammation or fever, the nature of which is not yet declared, than the egotistic alteration of character which seems to lie at the basis of insanity, and which probably depends directly upon minute changes taking place in the cortical substance of the brain. It is a real bodily sensation (though at first indescribable) which enchains the sufferer's attention; and before long this vague feeling is exchanged for a positive localized sense of uneasiness or actual pain.

Sometimes the early mental state is one not merely or chiefly of despondency, but characterized by suspiciousness and irritability of temper, with quick changes from high spirits and loquacity to moody silence. In any case, after a time, the patient not only exhibits in his aspect and conduct the general uneasiness from which he suffers, but begins to complain of definite subjective symptoms. Probably the most common of these is pain of a gnawing or burning character, or else a sense of great though vague uneasiness at the pit of the stomach. But in fact any part of the peripheral distribution of the sensory nerves may be the apparent seat of painful sensation; and besides this there is often a generally heightened sensibility of the skin. Both the active pain and the heightened sensibility of (uncomplicated) Hypochondriasis are subjective, and resemble the similar phenomena which are so common in hysterical women, in vanishing when the patient's attention is powerfully diverted from them. The painful sensations of which hypochondriacs complain are very acute; and their severity concentrates the attention of the sufferer exclusively upon them, increasing the apparent egotism of his disposition. But it may here be remarked, that the heightened self-feeling of Hypochondriasis does not partake of the despondency of true melancholia, still less of the character of other forms of insane egotism. The patient (as observed by Leidesdorf), though depressed in mind, not only wishes to get rid of his malady, but has great faith that he will do so: a faith which suffers repeated shocks, indeed, from the non-success of particular remedies, but quickly revives in favor of some new mode of treatment. The eagerness with which he pursues the means of cure is the true cause of the limitation of his thoughts.

Next to pseudo-dyspeptic symptoms, and the occurrence of pains or anomalous feelings at the pit of the stomach, the most common morbid sensations in Hypochondriasis, are, probably, formication of the skin, and *burning pains* in the course of particular superficial nerves. It is noteworthy that, so far as we are aware, the nerve-pains most frequently assume the burning type, rather than the lancinating, throbbing, or aching forms which neuralgia more commonly takes. A common occurrence is the sudden shifting of the pains, or the sense of formication from one part of the body to another, or their sudden extension from a small area which they first affected over almost the whole surface of the body. It is important to distinguish, from these pains, those which occur in the early stages of locomotor ataxy: and, in fact, this may be difficult in some instances, for the ataxic pains also are singularly shifting. However, the pains of ataxy are confined so strictly to the *limbs* (usually to the feet, thighs, and nates), that this of itself constitutes a difference from hypochondriacal pain. Another very frequent subjective symptom is the feeling of pain or great but indescribable uneasiness deep in the heart, or the lungs, or the liver, the bladder, or

the rectum. The development of the subjective symptoms is very often seriously influenced by the fact that the patient is driven by his misery to consult medical books, or to pester his medical friends with questions bearing on his sufferings: his defective knowledge and distorted fancy leads him to apply, *à tort et à travers*, the scraps of information which he picks up, and to imagine, successively, that he has discovered in himself the signs of one, two, or half a dozen serious organic diseases. Attention being thus directed to particular organs, the subjective symptoms naturally increase and multiply, and the emotional excitement produced also frequently sets up severe functional disturbance, such as flushings of the face, abdominal pulsation, palpitation of the heart, partial suppression of bile and jaundice, or bilious diarrhœa; symptoms which still further confirm the sufferer in the belief that he is laboring under serious organic disease. A very common delusion is the belief that there is fatal heart-disease; and a scarcely less frequent one is the persuasion of the patient that he is impotent from spermatorrhœa: this last is of course greatly fostered by reading pseudo-medical treatises. In the case of patients whose family is strongly tainted with insanity, the anomalous sensations often assume a type which approaches to hallucination or illusion (as where there is the belief that a serpent is writhing about in the entrails, &c. &c.), or the judgment becomes affected to such a degree that the patient entertains preposterous delusions (as that he is made of glass, and in danger every moment of being broken, that he is being magnetized, that people are conspiring to poison him, &c.). The delusions sometimes are confined, at any rate for a time, to one or two organs, but are nevertheless so extravagant that it would really seem no paradox to say that the patient has a mad stomach, a mad liver, or a mad bladder, while on all other matters his intellect remains healthy, and often usually acute. It is probable that *any* portion of the nervous centres may be functionally disordered in Hypochondriasis, and thus give rise to disturbances of this kind in the organs with which they are related. But on the subject of the Protean symptoms of Hypochondriasis it is really unnecessary to enlarge further, and we may refer those who desire to read a truthful and highly picturesque description of them to the pages of Burton,[1] to say nothing of more modern writers.

Diagnosis.—The diagnosis of Hypochondriasis, from maladies superficially resembling it, is proverbially beset with difficulties, and the practitioner can only gain confidence in his decision on the more doubtful cases by means of long experience. Nevertheless, the general principles on which his judgment must be formed are not very difficult to state.

If the anomalous character of a patient's subjective symptoms point in the direction of Hypochondriasis, the very first subject of inquiry should be the family history. A well-defined history of insanity in the race would at once indicate the probability that the patient's sensations were partly illusory, and not referable to their apparent site. On the other hand, a decided history of the absence of insanity and of the other severe neuroses from the family during two or three previous generations would still more strongly suggest that the case was *not* one of Hypochondriasis. The next point for investigation would be the mode of commencement of the illness. A history of the primary occurrence of severe bodily symptoms, whether in the shape of pyrexia, of disturbance of hepatic or gastric functions, or of pain in the course of nerves, is unfavorable to the diagnosis of Hypochondriasis, unless these phenomena were preceded or accompanied by psychical changes such as have been already described. Even a more chronic development of capricious pains, of formication of the skin, of flatulence, palpitations, and the like, is not specially indicative of Hypochondriasis, unless there is unusual anxiety on the patient's part, and an egotistic tendency to dwell on his sufferings. A great deal may be gathered from considerations of age and sex. Hypochondriasis is pre-eminently a disease of adult and middle life; it is hardly ever seen before puberty, and it very rarely makes its first appearance after the age of fifty. It is greatly more common among men than among women; in the latter sex it appears to be replaced, for the most part, by hysteria. Still Hypochondriasis may occur in women, and the question of diagnosis from hysteria, in such a case, becomes important, and may be very difficult. Beyond all other circumstances which favor the probability of Hypochondriasis is the fact of a strong hereditary taint of insanity. The age at which the symptoms commence is important: thus the first appearance of hysterical phenomena nearly always takes place between the ages of fourteen and thirty, or else at the grand climacteric; and has very commonly a marked relation to those changes in the nervous system which correspond with the changes of the sexual apparatus; whereas the development of Hypochondriasis is especially associated with the circumstances of middle life—in the rich and idle with the *tedium vitæ* of a

[1] Anatomy of Melancholy, pp. 270—274.

purposeless existence; in the poor and anxious with the cares of a family, and perhaps with the added misery of a conscious failure in efforts to support relations and dependents. Severe moral and emotional shocks may be followed either by hypochondriacal or hysterical disorder; but the latter is the infinitely more probable result in women who are not descended of markedly insane families, and especially in women who lead busy lives.

One of the most important questions in diagnosis is the decision whether, if Hypochondriasis be present, there is not at the same time some organic visceral disease; for it sometimes happens that the first sign of the occurrence of such disease is an outbreak of hypochondriacal symptoms, the patient being hereditarily predisposed to the latter disorder, and his nervous system excited to morbid action by the irritation of the new organic processes which are going on. Of the diseases which have been known to produce such an effect, structural changes of the liver, and, next to them, structural changes of the stomach, are probably the most frequent examples; and, after these, aneurisms of the great vessels, valvular diseases of the heart, angina pectoris. It is unnecessary here, even if there were space, for us to go into the characteristic symptoms of these organic diseases. The first feature which may lead the physician to suspect the existence of organic visceral disease, in the midst of symptoms which he feels sure are hypochondriacal, is the persistence of some one complaint by the patient—*e. g.*, of pain in a particular locality: especially if this be combined, always in the same order, with other symptoms that belong to the suspected organic disorder, and with which the patient is not likely to be acquainted so as to be mentally influenced to reproduce them. Thus if, along with a fixed complaint of pain in the præcordia increased by swallowing, there is the regular occurrence of regurgitation of some of the food very shortly after deglutition, it is a case for inquiry as to the possible existence of cancerous or other stricture of the cardiac end of the stomach, &c. It is needless to say that physical signs, when they are present, are the most valuable helps in discerning organic disease which is masked by Hypochondriasis; yet even here there is need of caution. For instance, the occurrence of hardness and tumidity in the epigastrium or the hypochondrium, in such a form as closely to imitate a scirrhous tumor (even on repeated examinations), may be produced by spasmodic contraction of one or both recti: in such a case the administration of chloroform would at once dismiss the suspicion by dissolving the "phantom" tumor. A circumstance which is always of doubtful interpretation is the occurrence of wasting, especially if combined with jaundice. If this takes place with rapidity, it can hardly be owing to hypochondriacal disturbance of digestion and assimilation, but is probably due either to the generally depraving effect of cancerous or tubercular taint, or to direct interference with nutrition from the mechanical effects of ulcer, stricture, or tumor, upon some of the chylopoietic viscera. The combination of insidious and unsuspected malarial poisoning with hypochondriacal tendencies may produce formidable difficulties in diagnosis, which can only be overcome by careful study of the patient's past history, sometimes by the discovery of enlarged spleen, and above all by the effects of anti-periodic medicines. Another variety of blood-poisoning, which in hypochondriacal patients may be somewhat masked, is chronic alcoholism; but it has been shown in another article in the present volume, that the symptoms of the latter complaint are, after all, tolerably distinct and recognizable from their peculiar grouping, and even in a hypochondriac they may be generally identified.

A more serious difficulty in diagnosis than any which has yet been mentioned is the distinction between certain forms of Hypochondriasis and true melancholia. Given a patient with a decided family history of insanity, with a mental condition marked by a strong tendency to dwell on complaints of bodily misery, and with dyspepsia and flatulence, it may be very difficult to say whether or not the case will pass into true melancholic insanity. The following case will give a good idea of the occasional uncertainty. A postman, aged forty-three, a widower, was much overworked, and especially harassed by having to sort the letters in the morning before he started on his beat, a task which had to be hurriedly done, and hence gave him much anxiety lest he should make mistakes. He applied for relief from dyspepsia and flatulence and bilious diarrhœa, but at the same time complained that his spirits were dreadfully low, that he had thoughts of suicide, and that he believed he had "something alive in his inside." A simple tonic mixture of mineral acid and bark, combined with the moral influence of encouraging assurances from the physician, did this patient so much good that he soon seemed perfectly well, and remained so for some months. He then got married again, and his marriage apparently embarrassed his means, though not seriously; but his despondency now returned in the form of a belief that he and his family would have to go to the workhouse (of which there was really not the least danger), and the impulse to suicide again became very urgent. At the same time his dyspepsia

and bilious diarrhœa returned. He applied for medical relief, was ordered the same treatment as before, and was encouraged to hope for a speedy cure; but the very next day he attempted suicide by completely severing the whole of his genitals from his body with a razor. He was taken to St. George's Hospital, and with great difficulty kept alive while the wound healed. Six weeks after his discharge from the hospital he appeared before his former attendant, looking fat and fresh-colored, but more despondent than ever: indeed plainly insane. He was then lost sight of, but there could be little doubt that he would get worse, and, if not carefully watched, would commit suicide.

Such a case as the above has little to separate it from Hypochondriasis except the one important feature of the early occurrence of *suicidal despondency;* but this feature would probably be sufficient justification for a decided diagnosis. It is only where the hypochondriac patient has been exhausted by a long continuance of his sufferings and rendered hopeless by the failure of a thousand attempts at cure, that he turns his thoughts to self-destruction, and by that time he may be considered really insane. Indeed, the hypochondriac proper regards the idea of suicide with the utmost repulsion and disgust.

There is no serious difficulty in distinguishing simple Hypochondriasis from the other forms of insanity.

Prognosis.—The prognosis of Hypochondriasis varies extremely, not so much with the apparent severity of the symptoms as with the circumstances under which they arose, the length of time during which they have already persisted, and above all the degree to which the patient's family has been tainted with insanity. But in general it may be said that the younger the patient, the shorter the time during which he has suffered, the less that the nutrition of the body has deteriorated, and, above all, the less of decided family taint of insanity that can be traced, the more hopeful is the aspect of the case; and *vice versâ*.

Etiology.—The "causes of Hypochondriasis" is an expression even more singularly unhappy than the average instances of a phraseology of causation applied to those circumstances which precede the outward and visible development of functional disorders. To commence with those influences which have a conventional title to be called "exciting" causes: it is undoubtedly true that in a considerable number of cases the train of disastrous events has seemed to be fired by the moral collapse consequent on over-exhausting labor, or bitter disappointment of cherished hopes, or on the sudden revelation to the mind of an idle man that he is a mere burden on the face of the earth. Again, it is commonly said that reading or conversation on medical topics often frightens laymen, and, more rarely, even doctors, into a nervous and hypochondriacal frame of mind. There is, doubtless, something to be attributed to such influences, but the most thoughtless person could hardly fail to be struck, on reflection, with the surprising infrequency of Hypochondriasis in comparison with the ubiquitous operation of such influences as grief, fatigue, the sense of shameful failure, the habit of miserable and heart-wearying idleness, and the practice by the laity of reading medical treatises. If we turn to the events which would be conventionally spoken of as "predisposing" causes, we are scarcely likely to be more satisfied with the appropriateness of the term "cause;" though we come upon facts of far greater practical value than those which have just been mentioned. The fact of hereditary insane taint, for instance, is an antecedent which is observed in an immense number of cases, if not in all. The preponderance of males among hypochondriacs is equally unmistakable; and so is the fact that the bulk of cases occur in persons in the prime and vigor of life. It is also an undoubted fact that the average level of intellect in hypochondriacs is not below but rather above the general standard; and that their bodily health has often been excellent up to the moment when the nervous symptoms made their appearance. But instead of saying that these circumstances are "causes" of the disease, it will be convenient to say that they are *conditions* of its occurrence in the following degree and manner: A certain number of the descendants of a family strongly tainted with insanity will invariably be born with peculiarities of the nervous system: these peculiarities do not, probably, consist of recognizable structural faults, but rather of tendencies of one or more (perhaps scattered) portions of the central system to change interstitially in a certain morbid direction, at particular crises of life, through which healthy organisms pass unharmed. Arrived at such crises the nervous system will surmount them, or will succumb, according to the absence or the presence of certain external disadvantages. If the morbid change occurs, it will not affect the machinery of the intellectual and reflective faculties chiefly, perhaps not at all; its force will be spent mainly on that portion of the nervous apparatus which performs the function of translating to the mind the perceptions of sensitive nerves at the periphery: but it is not impossible that even the primary morbid action is occasionally developed in nervous

centres which govern secretion and other functions of organic life; and that the dyspepsia, and other functional disorders of viscera, may in these cases be the *direct* result of a central disease, instead of reflex phenomena dependent upon the condition of consciousness, as is probably the case in many instances. In the latter stages of the malady there can be no doubt that the mental depression reacts with great force upon the machinery of organic life, disordering secretions and rhythmic motions very extensively.

The Pathology of Hypochondriasis, in the strict sense of the word, does not exist, for there are no anatomical or physiological facts upon which it can be based. Morbid anatomy has revealed absolutely nothing which in the slightest degree explains the occurrence of the disease, and the physiology of the symptoms is to the last degree obscure and uncertain in its interpretation. It is only in those cases which develop into true insanity, more especially those which pass into dementia, that the brain exhibits any notable changes; and these alterations, when they occur, are no proper part of Hypochondriasis. It is neither impossible nor unlikely that the improved modern methods of examining the nervous centres, if they could be applied to the central ganglia of certain visceral nerves (and especially to the nucleus of the vagus), might detect appreciable changes even in the early stages of the disease. But the opportunities for carefully examining the nervous system of patients in the early periods of Hypochondriasis can rarely be obtained, and it is probable enough that the question as to the pre-occurrence or not of structural changes will never be thoroughly cleared up.

The Treatment of Hypochondriasis consists of the use of moral and constitutional remedies and of remedies for symptoms.

It is obvious that the first duty of the physician is to encourage the hypochondriac to forget his woes; but nothing is so difficult in practice, and that for the best of reasons. It is a fallacy to suppose that the sufferings of the patient are unreal; on the contrary, they are most vividly real, and it is impossible that he should forget them till they cease. Yet the mind has a reflex influence upon the bodily disorder, which may be as effective for good as for evil; and this fact may be taken advantage of. The key to the moral treatment is the breaking down of the patient's morbid self-concentration, and this object may be achieved to some extent in many cases by a change in the course of his daily life. The class of patients with whom this may be most readily carried out are those in whom the constitutional tendency to Hypochondriasis is aggravated by the *ennui* of an idle life: for these an active career or pursuit of almost any kind is an immense gain; only the new occupation should be one which forces them to mix with the world. The isolated activity of the student is no real diversion from the fancies of Hypochondriasis, as the case of Dr. Johnson, and of many other famous intellectual workers, abundantly proves. It is needless to say that all actively depressing influences should be removed, such as immoderate venereal indulgence, of whatever kind, or alcoholic intemperance. On the other hand, the influence of new emotions which tend to lift the patient out of himself can scarcely fail to be beneficial; and it would be a real good-fortune to a hypochondriac if he could fall in love in a natural and healthy manner, or if he could interest himself warmly in philanthropic schemes or other plans of public usefulness. And, above all, something like a police supervision should be exercised as regards his studies, in order that he may be rigorously kept from the perusal of medical or other books which might remind him of his miseries; for though we do not believe that these things can create Hypochondriasis, yet they can certainly prevent its cure. It is well understood, however, that no good can be effected by simply laughing at his narrative of suffering, or bantering him on his fancifulness; on the contrary, it is necessary for the physician to be interested and to believe in the reality of his painful sensations. If the patient once thinks that the doctor is taking pains to get at the secret of his troubles, he will be inclined to accept the first word of encouragement the latter throws out; and the reflected influence of reviving hope will be certain to assist recovery.

The *constitutional* treatment is to be directed towards improving the general nutrition; and the task here is partly that of aiding the primary process of digestion of food, and partly that of rendering more active the processes of decomposition and exchange in the tissues generally. The hypochondriac either has a deficient, a capricious, or a ravenous appetite, but in any case the primary function of digestion is almost always markedly impaired if the disease has lasted for any length of time; and when this depends on a want of tone mainly, or a condition of irritation of the stomach (such as is indicated by a coated tongue with a red or strawberry tip), the use of vegetable bitters and mineral acids will often do great good. Defective secondary assimilation, which will be especially indicated by the condition of the urine, is generally much benefited by the use of cod-liver oil for a

rather prolonged period, if the remedy can only be tolerated by the stomach. In cases where the oil cannot be borne, cream, butter, or some other form of fat, will often agree, and may be made the first stage to inducing the stomach to retain the cod-liver oil. Nor is it by any means only in cases where there is general emaciation that the administration of fat does good; it is probable that the nutrition of the nervous tissues is directly improved by this treatment in many instances. The administration of iron is doubtless of great use to some anæmic patients, and sea-bathing frequently appears to exercise a very beneficial influence: but the first of these remedies is generally most efficacious when taken in the form of the chalybeate waters of some foreign spa; and there is good reason to doubt whether both mineral waters and sea-bathing do not owe most of their apparent power to the moral influences of travel and change of scene and mode of life. The more specific nervous tonics, such as strychnia, quinine, or phosphorus, seem to exercise but a doubtful and accidental influence.

The treatment of symptoms is a thing to be eschewed in Hypochondriasis, with certain special exceptions. While, however, it is desirable to avoid concentrating the patient's attention on parts which are the apparent seat of mere morbid sensations, it is important to relieve him of the distress caused by real (though mere functional) disorders of the digestive system. Decided acidity of the stomach should be counteracted by the use of antidotes, of which none is more efficacious than magnesia ponderosa, in ten-grain doses thrice daily, or Brandish's solution of potash, ten minims three times a day, with gentian or cascarilla. The excessive or too long continued use of alkalies is, of course, to be avoided. The distressing flatulence, which is often one of the earliest, and also one of the most annoying symptoms, is greatly relieved by creasote (one drop in a pill twice or thrice a day), or the infusion of valerian. Alcoholic tinctures should be very cautiously employed, if at all; for there is a real danger of the patient coming to appreciate the comforting sensations given by the spirit so highly, that he gradually takes to drink: this is especially true in the case of hypochondriacal women, as it notoriously is in hysteria. We may add that it is particularly likely to occur in patients exhausted by masturbation, or other venereal indulgence. The constipation, which is frequently so obstinate and troublesome, must be remedied, if it be anyhow possible, without the use of drugs; for it is most dangerous to stimulate the patient's love of self-doctoring in the direction of the habitual use of purgatives. The prescription of fruit, green vegetables, &c., as articles of daily food, is a far more desirable mode of accomplishing our object: and the habitual practice of active bodily exercise is a powerful aid to the same end.

The question of the *quantum et quale* of physical exercise which may be beneficial in Hypochondriasis forms a fitting subject with which to conclude our remarks on treatment, since this is a remedy which directs itself alike to the moral, the constitutional, and the symptomatic condition of the hypochondriac. The only rule, however, which it is possible to lay down for our guidance in this matter, is the direction to employ physical exercise in such a manner and to such an amount as shall fully exercise the muscles without ever producing severe fatigue, and shall also be amusing to the patient. It is a very dangerous error to carry exercise to the fatigue point; a short continuance of such malpractice will usually suffice to produce a profound deterioration of the vigor of the nervous system, and an aggravation of the hypochondriacal fancies.

HYSTERIA.

By J. Russell Reynolds, M.D., F.R.S.

The most characteristic feature of Hysteria has been held to be a particular form of convulsion, which will be described hereafter in detail; but there are many phenomena, some falling far short of convulsion, even when they resemble it in kind, and others differing widely from it in their form, and these have been termed "hysterical." If, therefore, by the word "Hysteria" is intended a definite disease, the term "hysterical" should be employed with a no less definite aim; but it has un-

fortunately been sometimes used to denote either a mere variety of the "nervous temperament," a mood or disposition of the health of both body and mind, or sometimes a vague condition of disordered function which cannot be conveniently placed elsewhere. The employment of the word "hysterical" may sometimes be found indicative of the state of the mind of the practitioner rather than of that of the patient's health. It simply conveys a doubt as to what is the matter, but expresses a prevailing conviction that it is nothing very serious as to life, and that it might culminate in an attack of convulsions of the kind called "hysteric."

The vast preponderance of Hysteria in the female sex has given rise to its name, and to a theory as to its essential nature and mode of production. It has, however, and especially of late years, been so distinctly shown that Hysteria may exist among men, that the older nomenclature, although retained on account of its practical utility, is virtually exploded as to its etymology. Hysteria is not necessarily associated with disease or derangement of the generative organs of either sex: such association may and does very commonly exist; but the true nature of the malady may be overlooked if regard be paid exclusively to that particular relation.

The hysterical condition may exist for many years, and yet be unattended by any distinct convulsive paroxysm. The latter never exists without the former. There are at the present time numberless individuals whose lives are, to themselves and their friends, the sources of more or less constant misery, from the fact of their being distinctly and definitely hysterical, but in whose history there has never occurred a single attack of characteristic convulsion. We cannot, therefore, draw the line of definition so as to include the paroxysm and exclude all beside; but must, on the other hand, regard as essential that which is constantly present, and recognize as the disease Hysteria a peculiar condition of the nervous system of which the paroxysm is only one, although a highly important feature. The mental state of the hysteric patient is more constantly and as characteristically altered as is the condition of the muscular system. There is a defective or perverted will; an increased activity of emotion, and sometimes of thought; an altered and augmented general sensibility; an exaggeration of all forms of involuntary motility—ideational, emotional, sensational, and reflex; and usually some distinct perversion of the physical health. It is almost impossible to frame an accurate "definition" of the disease; and it seems to me more desirable to make the above general statement than to attempt a less detailed description for the purpose of giving it the form of a definition.

NATURAL HISTORY.—I. CAUSES.—*Sex.* Doubtless the most frequently predisposing cause is that condition of the nervous system which is more or less characteristic of the female sex. Hysteric women are met with daily; hysteric men and boys are of comparatively rare occurrence. When Hysteria is found in either a man or a boy, it is to be observed that such person is, either mentally or morally, of feminine constitution, or that he has been overworked mentally, exposed to much emotional disturbance, or greatly reduced in physical power. It is well known that men have frequently become hysterical as the result of some severe accident, such as a fall from a horse or a collision in a railway train. The predisposition to Hysteria does not exist in the fact of an individual having the organs of one or the other sex, but in the possession of a nervous state—an habitual, constitutional, or induced relation between the several elements of mental, moral, and physical life—which is common to, but not always possessed by women; unnatural to, but sometimes exhibited by men. Some women are as little likely to become hysterical as some men are to fall pregnant; they are of masculine build, both mentally and bodily, and their existence and their predispositions to disease furnish another proof of the truth of the general proposition, that it is in the nervous endowments, and not in the nature of the reproductive apparatus, that the special predisposition lies.

Age.—In the female sex, Hysteria usually commences at or about the time of puberty, *i. e.*, between twelve and eighteen years of age; but when once developed, the symptoms may remain throughout life. At the climacteric period Hysteria may become developed in a previously healthy person;—this is, however, comparatively speaking, rare; but examples of extreme Hysteria may be met with after the climacteric period has been passed—and that without unusual discomfort—in some women who were hysterical in earlier life, but whose hysterical symptoms were then less pronounced. Of 351 cases analyzed by Landouzy,[1] the ages at commencement were distributed as follows:—

From 10 years	to	15 years	.	48	cases.
" 16	"	20	"	.	105	"
" 21	"	25	"	.	80	"
" 26	"	30	"	.	40	"
" 31	"	35	"	.	38	"
" 36	"	40	"	.	15	"
Above 40 years of age	.	.	.	25	"
				351	

Boys sometimes exhibit hysteric symptoms at puberty, but the most marked

[1] Traité complet de l'Hystérie, Paris, 1846, p. 184.

cases of the disease that I have seen in males have been at a more advanced age, viz. from thirty-five to fifty and upwards. It will be seen therefore that Hysteria may occur at any age; and it must be remembered that many of the symptoms of senile decay, and many of those which accompany chronic degenerative disease of the brain in middle life, are often called "hysterical," because they correspond very exactly with those which, when they occur in earlier years, are described, and without hesitation, by that word. As decaying life passes on to a "second infancy," we see the signs of a "second childishness:" but often, before either of these terms would be strictly applicable, we may observe something that resembles, in its nervous characters, the period of commencing puberty; for often the first sad signal of faltering power is either undue emotional disturbance or deficient control of its display. We call these states "hysterical," and may perhaps sometimes use that word very loosely; but the resemblances are real, and may help us to appreciate the pathology of the disease "Hysteria."

Temperament.—If by nervous temperament be meant simply an hysterical condition, it is unnecessary, and would be wrong, to speak of the one as a predisponent to the other. The two expressions are sometimes used interchangeably; but this is a great mistake: the truly nervous temperament implies no disproportion, *inter se*, of the several nervous endowments; all are alike active. There may be in those of nervous temperament a higher but a shorter life; an intense vitality, which burns itself out before its time: but the hysteric condition is essentially one of disproportion, and it is not encountered with any especial frequency in those who have previously exhibited the exaltation described. This latter is peculiar to some individuals, but is not necessarily morbid.

Sexual Condition.—Undoubtedly Hysteria is more common in the single than in the married, but it is not limited to the former, and it may exist to its highest degree in the latter. It is said that the wives of incompetent husbands, and barren women, as well as widows and old maids, are frequent victims of the hysteric malady; but statements such as these do not convey the whole truth in regard of the etiology of Hysteria, and it must be remembered that Hysteria is met with in those who are happily married, in pregnant women, and in nursing mothers.[1] From what has already been said in regard of the age at which the disease begins, it is obvious that age, rather than sexual condition, is the predisposing cause. Hysteria commences at a time of life when girls are, in this country at least, held to be too young for marriage. The fact of its existence, to a high degree at any rate, does not increase their social chances in this particular; they often remain single because they are hysteric, and this probably quite as often as that they become hysteric because they are single. Such a condition does not tend to improve itself, and the disappointment of being left alone may keep up and exaggerate the morbid state. The facts that there are to show that marriage has sometimes cured the malady are not so numerous, nor are they of such character, as to outweigh the evidence to be derived from the persistence, and even aggravation, of Hysteria after marriage. Moreover, it is well known that hysterical women who are married are often frigid, and averse to sexual intercourse, and that their aversion is not necessarily due to pain. It is comparatively easy to shrug the shoulders and utter innuendoes over a case which baffles treatment by well-directed regimen and medicine; but does it not sometimes happen that such hints are only a clumsy excuse for the failure of therapeutics? That marriage may be of use in Hysteria is not denied, but it is asserted that it may be so by other than its mere sexual relationships; there may be a number of circumstances which are changed by taking this step in life—annoyances may be removed, new purposes conferred, work given to do, and strong help rendered kindly for the doing it; and all these may concur to lift the hysteric woman out of her former self. If, then, we are regarding Hysteria etiologically, all these points should be considered, and the argument from the effects of marriage should not be based upon one element alone.

Sexual excesses are held to be sometimes productive of Hysteria; and doubtless they are so occasionally in men, but there is a deficiency of evidence to prove this in regard of women. According to Duchatalet, Hysteria does not exist with especial frequency in women of the town, and in those who are exposed to excesses of this kind.

Temperature, Climate, and Season.—There are many facts to show that warmth of climate and the seasons of spring and summer conduce to a production of the hysteric condition, but it has yet to be shown what is the element comprised under those terms which is of etiologic moment.

Occupation.—It is demonstrable that absence of employment, as it is commonly met with among the upper classes, favors the production of Hysteria in women; and it is equally clear that overwork, anxiety, and great "strain" upon the in-

[1] *Vide* Niemeyer, Handbuch, p. 356; Hasse, in Virchow's Handbuch, p. 212.

tellectual and moral faculties, lead to the development of Hysteria in man. It is, however, questionable whether the mere fact of occupation, or its kind, is either favorable or unfavorable to health, or to Hysteria. The unoccupied life of woman is one of exposure to numberless disturbing causes, as is also the over-occupied life of man. The woman, or the young girl, who has nothing to do—nothing serious to which her mind is turned—finds "time" to be egregiously tedious; and she has to choose between two evils, either that of "doing nothing" with it, or that of "getting rid of it" by utterly frivolous pursuits, the distraction of reading silly books and doing the sillier things that "society" prescribes. She thus brings upon herself all the petty annoyances of selfishness and wounded pride, and all the tease and turmoil of unreal and extravagant emotions. The man who is "overworked" finds, commonly, in that work itself, and in that which leads to it, much more than mere mental occupation, viz., anxiety, suspense, and worry, with their concomitants, loss of both appetite and rest; and it is these which, by the nature of their operation, develop the hysteric condition, rather than the amount of simple work which has to be undertaken or pursued. It is then, I believe, neither the presence nor absence of occupation, *per se*, which conduces to the development of Hysteria in either sex; but in the one sex the "nonsense" that takes the place of sober work, and, in the other, the emotional disturbance that attends upon over-exertion. These lead, under apparently dissimilar circumstances, but in a really analogous manner, to the production of the same result. That which is common to the two conditions is an excess of emotional disturbance; but in the one case it is the outcome of idleness, in the other it is often the cause and motive of overwork. Absence of occupation may give time and opportunity for the manufacture of feelings that are tormenting and unreal; over-exertion may be the means taken to relieve the anxiety and emotion which are already pressing. Either condition may be met with in either sex, but the common distribution of work and idleness in relation to Hysteria is that which is indicated above.

Menstruation.—It has already been said that Hysteria breaks out most frequently at or about the time of the commencement of puberty; but it has not yet been shown that it has any definite relation to the varying conditions of menstruation. In an individual already hysterical there is or may be more than usual disturbance at or near the monthly "periods," and this is exaggerated by any kind of irregularity which may exist. Menorrhagia, by reducing the vital power, through loss of blood; dysmenorrhœa, by affecting the same result through nervous exhaustion; or amenorrhœa, by its physically direct, and mentally and morally indirect influences, may, either of them, conduce to the increase, or even development, of the hysteric state: but it has yet to be shown, that either one of these is of itself sufficient to produce the disease. For it must be remembered that Hysteria may exist to its highest degree—it commonly has done so in the cases which have fallen under my own observation—in individuals who have presented no anomaly whatever in regard of the menstrual functions; and, yet further, that it is found in the male sex, which may be influenced much by sexual conditions, but which exhibits nothing analogous to those perturbations that have been mentioned. All that I can add to the above is, that I have found no one condition, either of excess, defect, or perversion of the menstrual function, so commonly or prevailingly associated with Hysteria as to give to it any special claim to consideration in the etiology of the disease. Some people become more or less hysterical about everything that is wrong in the performance of their functions: it is both obvious and notorious that the uterine functions are invested in the minds of women with an amount of importance that is more than their physiological due; and hence it is that menstrual derangements are regarded by them as exerting much more influence than those of the digestive or the excretive functions, and are therefore brought prominently forward in the statement of their cases. That they do exert this excessive influence may be due partly to their intrinsic nature, but partly also to the results of thought and feeling about them; and it is important to bear in mind, for etiologic purposes, this latter element in their mode of action.

Hereditary taint has not been shown to exert any marked influence in the development of Hysteria. It is well known that members of one family occasionally exhibit similar symptoms, but then it must be remembered that many conditions besides those of blood-relationship are ordinarily common to the individuals of one family; they may share equally in what is good or bad in respect of example, education, and surrounding circumstances. M. Briquet states that those who are born of hysterical parents are twelve times more liable than others to the outbreak of Hysteria. Very different numerical statements are made by others, and we must remember that besides the direct transmission of an hereditary taint there is much contingent upon having an hysterical mother.

The most frequent *determining cause* of an outbreak of hysteric symptoms is some

mental or moral disturbance; either a violent and unexpected commotion, or more commonly the occurrence of a trivial circumstance which takes the individual by surprise, overcomes the power of restraint, and gives evidence of what is often an ill-understood, but long concealed, annoyance or distress. Sometimes the determining cause is physical, *e. g.*, an accident, a loss of blood, or an attack of acute bodily illness, such as one of the exanthemata, pneumonia, or pleurisy. It then appears, and sometimes to the surprise of even the most intimate relatives, that there is a morbid condition of both mind and body which is difficult to describe, and often much more difficult to manage. There may be the extreme symptoms of the hysteric paroxysm, or some of the marked features of the hysteric state. These have now to be described.

II. Symptoms. — It is convenient to separate the paroxysmal symptoms from those which exist in the intervals of attack; and the former will be best understood by those who have become acquainted with the latter, for the hysteric state does much to explain the hysteric paroxysm.

It will be well to divide the interparoxysmal symptoms into several categories, describing separately the condition of the mental and emotional, the sensorial, motorial, and general health.

(*a*) Interparoxysmal Symptoms.— *Mental and Emotional State.*—The will is perverted and defective, while ideas and emotions exhibit excessive activity. The patient says that she cannot do this, or cannot bear that; and, while under the belief that these things are impossible, they are so. It is often obvious to others that no physical impossibility exists; but it must not be supposed, therefore, that the patient is pretending or "acting a part." For the time being it is often true that the hysteric patient states the fact. What she wants is motive, and this may be sometimes supplied by a sudden alarm, or by an accidental circumstance; but under ordinary conditions the motive is wanting, or is held in abeyance by some imperious idea or emotion. There is an exaggeration and perpetuation of what exists in all people under certain circumstances. Fear paralyzes the strong man, while sudden alarm may make the weak man strong. A prevailing idea may limit as well as induce movements which the will can neither counterfeit nor hinder. Let this be remembered, therefore, at the outset in describing the symptoms of Hysteria.

The patient asserts that she cannot control her thoughts, emotions, expressions, or general movements; that she cannot move this or the other limb; cannot open the eyes; cannot stand or walk; cannot relax the rigid spasm of the hand or of the leg; and what she says is true under the existing conditions. But often, under the influence of some unexpected idea, or emotion, or sensation, she does the very things that were said to be impossible.

A patient may be carried into the room, and may fall when left for a moment to herself; tell her to walk, and a wooden doll seems as capable of movement; but, under the stimulus of a wish that what she is saying should not be overheard, she walks to the open door and closes it. Certain ideas seem rampant in her mind; she cries about them, and gesticulates in the wildest manner: tell her to be silent, to keep them to herself, or to control her feelings, and you find them exaggerated, and she affirms that "all the world shall hear" what she has to say; but a gentle rap at the door, that may come from the hand of some one from whom she wishes to conceal her state, is sufficient in a moment to hush this stormy talk, to compose her face, to dry her eyes, and make her speak and smile with placid composure. Sometimes she speaks in a whisper only, and if asked to "exert herself," or "make an effort," so that some particular friend who is a little deaf may hear what she has to say, the only effect is that the whisper becomes quite inaudible, that she makes less sound than ever, and often none at all—she moves her lips, but not even the ghost of a sound is heard to pass them; and yet this self-same person may, when no attention is directed to the voice, speak loudly enough to be heard and understood in the adjoining room. The fact seems to be that the will can be called into exercise only by some one dominant idea or emotion; and that it is this which determines the varying phases of the mental state. Under its influence the hysteric patient may submit to pain, annoyance, and privation such as a healthy person would shrink from without concealment; and under its influence, also, she may be unable to do what any one else could accomplish without either difficulty or fatigue.

Ideation is often excessively active in regard of certain classes of thought; there is sometimes quite a preternaturally acute condition of intelligence in certain directions, *i. e.*, in those directions wherein lie the morbid notions which are at the foundation of the malady. Often the hysteric patient makes many mistakes, and attributes to people and circumstances motives and meanings which they do not possess; but very often she exhibits marvellous ingenuity in perceiving the ideas of others, and in unravelling the intention

of complicated conditions, when these have happened in any way, or to any degree, to have had relation to herself. Apart from these direct personal relationships the mind often is, or seems to be, a perfect blank; the patient is listless, apathetic, and dull; a most uninteresting specimen of humanity; becoming of value only when her vagaries afford curious illustrations of certain pathological laws. There is a prevailing belief in the importance of self, and the patient thinks that she differs from every other human being; that ordinary laws do not apply to her; that she is "not understood," as the phrase is; and that only some very *outré* or utterly novel mode of treatment can do her any good. She believes all this, and acts upon it with a pertinacity "worthy of a better cause," exhibiting as much energy of purpose in a wrong direction as would serve to cure her were it rightly ordered.

Emotion is commonly excessive in itself, and also in its expression. The patient is hurried from one extreme to the other with almost ludicrous rapidity; and often she walks, as it were, constantly upon that narrow line where tears and laughter meet. Laughter and sobbing not only alternate, but coexist; and often without any obvious and sufficient reason for either. There is sometimes listless indifference to everything of ordinary interest; sometimes absorption in some trivial object: often great restlessness and impatience, with extreme irritability of temper on any attempt being made at control, or any suggestion being offered of change.

These mental and emotional conditions are liable to much variation. Sometimes the patient exhibits them for a day or two, and then becomes like an ordinary mortal; sometimes they are persistent, and vary only in the degree of their intensity.

In Hysteria we occasionally meet with somnambulism, cataleptic conditions, and syncopal attacks, which latter sometimes pass into the state of "trance," or apparent death, of which several cases are on record. These are, however, among the rarer features of the malady.

Sensorial Condition.—An exaltation of sensibility generally may be the earliest, and sometimes the only sign of the hysterical condition. It may, and more commonly does, exist in association with other symptoms, or in the intervals of their recurrence. But sensibility may be altered in several distinct directions; there may be increased, painful, perverted, or diminished sensation, or there may be absolute though partial anæsthesia and analgesia.

Hyperæsthesia is very commonly confounded—nominally and perhaps theoretically—with painful sensibility or sensation; from which, however, it is quite distinct. The hysterical patient often exhibits true hyperæsthesia; she does actually see, hear, smell, and taste what would not be perceptible to those in health, and to herself at other times. The exaggeration of hearing power is that which is the most frequently observed; hysterical girls do sometimes seem to "hear through stone walls:" they detect the minutest change in odor or in taste, and exhibit an exalted keenness of sight. Their sense of touch is also sometimes preternaturally acute.

Painful sensation, or dysæsthesia, is almost always present to some degree. One patient cannot bear the light, another is distracted by the slightest sound, to a third all ordinary odors are intolerable, and to others certain tastes, or the contact of sundry innocent articles of clothing, are most highly objectionable. Pain on pressure of the skin is very common; and sometimes the symptom is universal, but more commonly it is limited in its distribution, *e. g.*, to the occiput or the spinal column. It is to be observed that the direction of attention to them always makes the painful sensations much more intense; the mere fact of asking a question about them may sometimes develop them. A middle-aged hysteric woman, whom I saw in hospital a few days ago, had been lying for weeks with her hand before her eyes "to keep out the light" of a dull London sky. Bringing a candle before her—the room being so dark from an accidental fog that I could not see the pupils—she shuddered, knit her brows, and held both hands between her and its feeble light. There was no undue contraction of the pupils, and when her mind was distracted to the condition of her front teeth—the light being still close to her eyes—the brows were relaxed, the hands removed, and there was no expression whatever of uneasiness. This is but one example of a large class of dysæsthesiæ which may be commonly observed in the hysteric.

A lady to whom I was speaking lately, in a tone by no means loud, exclaimed in a voice much noisier than mine, and putting her hands to her ears at the time, "Not so loud—not so loud;" but, a moment afterwards, she stirred the fire so vehemently, and made so much noise in the process, that it was positively annoying to myself, and this without appearing to give herself the least uncomfortable sensation. Sometimes there is obvious and distinct painfulness of sensation upon impressions of ordinary intensity, and this may be limited to one or another organ. Most commonly, however, the pain is not necessarily associated with the impression, is of variable kind and locality, and intermittent in its form.

Besides these alterations of sensibility, hysteric patients constantly complain of "pain," more or less spontaneous in its development. Such pain, wherever it

may be situated, usually requires several strong adjectives for its description, and the account given of it is sometimes tediously minute. I have heard one hysteric lady enumerate and detail nine different kinds of pain in her chest! Of these some were bearable, some "intolerable," others "agonizing;" and four or five of them usually appeared together, and were present at the moment of description—and yet the face was calm, and simply conveyed the expression of interest in the description.

The localities of pain are very numerous, but some are much more frequently complained of than are others. The favorite haunts of hysteric pain are the top of the head, the left mammary region, the hypogastric, and the sacral. Sometimes the coccyx, and often one of the joints of the limbs, is fixed upon; but I have rarely observed any definite pain in the reproductive organs. When one of the joints is painful, and there is much tenderness of the affected part, it will commonly be found that passive movements or even succussion of the limb may be borne without complaint, whereas even gentle pressure of the skin is described as "agonizing in the extreme."

There are other sensations of which much is heard that do not fall into any of the foregoing catagories. The processes of ordinary life, which in health are unfelt, become sensible, and sometimes painfully so, to the hysteric patient. She feels the want of breath, the action of the heart, the intestinal movements, the processes of micturition and defecation, and those of sexual intercourse to an exaggerated degree, and in a distressing manner. She feels "short of breath," although there is no actual acceleration of the respiratory movements; "palpitation," although the action of the heart is normal; rolling and rumbling movements, when such are not perceptible to the physician; and distress or difficulty in relieving either the bladder or the rectum, when there is no physical disturbance of such processes. Sexual intercourse is sometimes quite impossible from some morbid sensitiveness of the vulva or spasmodic action of the sphincter vaginæ, neither of which can be detected on examination by the medical attendant.

There is, further, the sensation termed "globus hystericus," which is often, but by no means universally present. It is not always of the same character. Sometimes it is the feeling of a "great lump" in the hypogastric region, rising through the epigastrium and chest, to the throat; but this is, so far as I have seen, extremely rare, and is more frequently met with in books than in practice. The commonest form of distress is that of a ball or lump in the throat; a something which the patient cannot swallow, and which she feels will "choke" her. Anything round the neck is intolerable; she feels as if "something were tight there," although there may be nothing present in the form of dress; she makes constant attempts to swallow, but the "lump will not move;" and these discomforts are exaggerated by sobbing efforts which very frequently attend them. Sometimes the patient puts her finger in her throat to cause vomiting, that she may "bring it up;" sometimes she drinks largely to "push it down;" but in spite of both classes of effort the lump remains. Children when about to cry know what the feeling is, and probably it is of the same nature in the adult.

Diminished sensibility, and even actual anæsthesia of certain localities, may also be found. Anæsthesia may exist over the whole or a large portion of the surface of the body, and may extend to the deeper tissues, to the muscles,[1] and even to the nerves of special sense. It most commonly follows an hysteric paroxysm, but it may be met with when no such seizure has taken place. Commonly it is limited to certain parts, such as the back of the hand or foot, but it may be paraplegic or hemiplegic in its distribution, or may be found along the course of one or more intercostal nerves, and in the mucous membrane of the nose or mouth. The temperature of anæsthetic limbs is sometimes reduced. It is a rare event in any locality, but it has existed in several cases that I have seen in the mucous membrane of the vulva and vagina; the anæsthesia in these instances having been absolute, and that in highly hysterical, married women, who had borne and were still bearing children. Probably, anæsthesia is the cause of occasional retention of the urine and of the fæces in hysterical patients, as either bladder or rectum may be found sometimes enormously distended without the patient being aware of any other discomfort than that occasioned by the swelling. Loss of the senses of sight, hearing, smell, and taste also sometimes occur as phases of hysteric anæsthesia. [Following Piorry, Macario and Gendrin in France, and Szokalsky in Germany, Briquet, Charcot, Westphal, and others have of late years made an extended study of Hysterical anæsthesia, and especially *hemianæsthesia*.[2] In this, the line of demarcation is usually perfect for the whole length of the body. Briquet asserts that the left side is affected in 70 cases, to 20 of the right. There may be *complete* anæsthesia, involving the sense of touch, of pain, and of temperature, together; or it may be incomplete. In

[1] See article, Muscular Anæsthesia.

[2] Charcot, Lectures on the Diseases of the Nervous System, Lecture X.]

such a case *analgesia* (insensibility to pain) may exist, and yet heat and cold may be perceived and discerned; or, instead, *thermo-anæsthesia*, insensibility to temperature, without perfect analgesia.

The anæsthetic side of the body is apt to be pale and cold; anæmic or ischæmic. Sometimes very little blood will flow, even from a wound. Charcot supposes that possibly the story that, in the "Convulsionnaires" of Saint Medard, no blood followed when they were struck with a sword, may meet with a partial explanation in this way.

Affections of the special senses not unfrequently accompany that of the surface of the anæsthetic side. I remember in my own practice the case of a girl who, for several months, was totally blind in one eye, and then, recovering sight upon that side, the other eye became blind; this, too, being followed by recovery.[1] Galezowski has called attention to Hysterical *achromatopsia*, color-blindness, as occurring upon one side; especially the left.

Charcot points out that hemianæsthesia is commonly attended by *ovarian hyperæsthesia of the opposite side*. If loss of muscular power, or contraction of the limbs occurs, it is upon the same side as the hemianæsthesia.

As a part of the medical history of Hysteria, it is proper to mention in this place some facts in connection with the recent use of *metalloscopy* and *metallotherapy*. Dr. Burq, a French physician, obtained, in 1876, the appointment by the Société de Biologie, of a commission, consisting of Drs. Charcot, Luys, and Dumontpallier, to examine into his asserted observations. These were to the effect that, in patients having hysterical anæsthesia, the internal use, and external application of certain metals, different for different patients, had a remarkable power in restoring sensibility. The commission not only confirmed these statements (at least concerning the external contact of metals), but also discovered that, when, in hemianæsthesia, the sensibility of the affected side was restored, a *transfer* of anæsthesia occurred, to the sound side. Regnard, Magnan, Westphal, and Adamkiewicz essentially confirmed these results; and Charcot exhibited, in August, 1878, at the Salpétrière, some of the most remarkable of them, to Profs. Virchow, Grainger Stewart, Liebreich, Ray Lankester, Broadbent, and other distinguished medical men.[2]

Besides simple metals, magnets and galvano-electric spirals have been found to have similar influences. Some of the observers are inclined to refer the action in all cases to the effects of galvanic currents; but no satisfactory explanation has yet been agreed upon. In reading such accounts at a distance, the most natural suggestion would seem to be, that, as the events described take place in hysterical patients, the elements of imagination and simulation may play a large part in their production. Yet the evidence of those who have witnessed, and in great degree, confirmed them, entitles them to attentive study and record, while awaiting the final decision of science in regard to them. Farther reference to some of the same facts will be made in the article on *Hystero-epilepsy* in this volume.—H.]

Motorial Condition.—In health there are different kinds of movements which the muscular system exhibits; some are voluntary, others depend upon idea, a third group upon emotion, a fourth upon sensation, and a fifth upon impressions which are not felt. There is, moreover, a certain relation between these which is characteristic of health, but either of them may be so altered as to disturb the balance of well-being, and constitute or exhibit either derangement or disease. In Hysteria the normal relation is perverted, and there is an excess of the involuntary motility, and a diminution of the volitional; the former overrides the latter: and not only so, but the particular elements of the former display their powers in an altered ratio; sensational movements are not in due relation to emotional, nor are the latter to those of idea. Generally speaking, the lower acquires the ascendency, and exhibits the most marked phenomena.

Involuntary muscular activity may be increased in regard of either the readiness with which it is called into exercise, or the force and persistence of its display. If these involuntary movements be placed in the following order—ideational, emotional, sensational, reflective, and organic—it will be found generally true that the increased readiness of action is found at its maximum in the first, whereas augmented force and persistence of action are observed most distinctly in the last; and that the two kinds of alteration diminish in passing from either one of these extremes towards the other.

Voluntary movements are sluggish, the patient often lying about on sofas, or in bed, and saying that she is quite incapable of doing this or that. Irrational movements are in excess; and, under the influence of morbid "notions," gestures may be assumed, and sundry physical efforts performed which transcend the powers of health. All the emotional movements are exaggerated; the individual laughs, cries, and makes grimaces of the most distressing kind, and manages often to do

[1 See a paper by Dr. G. C. Harlan, Phila. Med. and Surg. Reporter, August 12, 1876.]

[2 British Med. Journal, Oct. 12, 1878.]

that which she could not do under ordinary circumstances.

Sensational movements are in excess: the brows are knit and the eyelids contracted upon the approach of light; there is starting and tremor upon the occurrence of any sudden sound or jar; there is violent spasm upon the production of any pain; and this often without any real exaggeration or sensibility. Reflex motility is greatly increased and perverted, so that spasms arise from "irritations" which in health would be passed by unnoticed; and numberless movements, of momentary duration and varied locality, occur in relation to impressions which are quite inadequate to produce them in the normal state. Other muscular actions, tonic or persistent, exist in various parts of the body, which differ from the reflex in the fact of their permanence, and must be referred to some induced change in the nervous centres. Of these, histrionic spasm, cramps, and long-continued spasms in the limbs or elsewhere, afford the most frequent examples. These tonic spasms are not confined to the muscles of the limbs, they may affect the pharynx, the œsophagus, the stomach, or the intestines; causing difficulty of swallowing or breathing, vomiting movements, strange noises, eructations, hiccup, borborygmi, and griping pains.

But besides the alterations mentioned, there is another kind which is less common, but of more serious character, viz., paralysis. Loss of power usually occurs as the seque lof an hysteric paroxysm, but it may sometimes take place spontaneously

M. Landouzy[1] states that, in 47 cases of hysteric paralysis, the distribution of the symptoms was as follows:—

General paralysis of motion and sensation	3
" " of sensation	2
Complete left hemiplegia	8
Complete hemiplegia, side being doubtful	6
Paraplegia	9
Partial paralysis	19

Usually only one extremity is involved, sometimes only a part of the limb; but in many instances there may be what is roughly termed "hemiplegia." In the latter case the face and tongue are rarely affected, and the paralysis is incomplete; and the motion of the partly paralyzed leg is not like that of an ordinary hemiplegic individual. Dr. Todd says that the patient "drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no act of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks."[2] This is true of many cases. And there is a yet further condition which may be noticed, viz., the absence of any special paralysis of the extensor muscles of the toes. If an ordinary hemiplegic patient be made to walk, it is seen that on attempting to raise the foot from the ground the toes droop and the leg is circumducted; but the hysteric patient does what the healthy person cannot help doing, except by a strong effort of the will,—when making the attempt to walk, she causes an elevation of the great toe at the time of endeavoring to move the foot forwards. The paralytic patient looks at his feet, the hysteric patient looks at her observers. The electric irritability persists in the palsied limb, and its nutrition does not become affected; but there is sometimes diminished sensibility to the electric current; it is at other times normal, and in some cases notably increased. Occasionally the electric contractility and sensibility are both diminished or abolished. Paraplegia is a form of malady sometimes witnessed, and patients suffering in such manner may keep their beds, or leave them only to exhibit a most curious mode of progression, or a series of falls which are quite unlike those arising from organic lesion of the spinal cord. At this time I have under my care two cases of hysterical paraplegia, in which the symptoms are almost precisely similar. The limbs are well nourished, there is perfect electric contractility and sensibility; the patients when lying in bed can elevate their limbs, separately or together, to any height that is required; they can move all the toes, and cutaneous sensibility is intact: but if they attempt to walk, their legs appear to be no stronger than pieces of wet paper, and they tumble down and bruise themselves in various quarters. What, however, is peculiar in the attempt at walking is this, that no amount of help, such as a strong arm on either side, prevents the staggering and falling, but the patients tumble down to within a few inches of the ground, and then recover themselves without help. An ataxic patient would walk, comparatively speaking, well with such assistance; a really paraplegic patient could not so recover the upright position. Where paraplegia has been due to hysteria, I have not found loss of power over the expulsors or the sphincters of either rectum or bladder; although it often happens that the latter exists without the former. General paralysis is extremely rare, and is only imperfect in degree.

Vaso-Motor Condition.—Sometimes hysteric patients, after a slight rigor, exhibit a certain amount of fever, with headache, and mild delirium, which speedily pass away, or give place to paralysis of the kind described. An irregular distribution of temperature, or the fitful occurrence of sweating, of salivation, or of increase in

[1] Traité complet de l'Hystérie, p. 106.
[2] Clinical Lectures, p. 620.

some other secretion, afford further illustrations of disturbance in that portion of the nervous system which is related to the control of blood-supply.

The *General* symptoms in Hysteria, or those outside the nervous system, are not distinctive in their character. Usually the patient is not in robust health; there is some pallor, and failure of nutrition; or there may be a great tendency to deposit of adipose tissue. Sometimes there is a condition of habitual ill-health, or delicacy; digestion is impaired, or the uterine functions are irregular, or there is some constant, but more or less indefinite, grievance in the head, thorax, or abdomen. On the other hand, there are many cases of Hysteria in which the general health is good; the patient eats, drinks, sleeps, feels, and is "well."

Numbers complain of nausea, and eructations, or vomiting; but in many cases these symptoms have been entirely due to errors, and even absurdities of diet, and in not a few to excess of stimulants. It is by no means rare for hysterical people to "take to drinking." Alcohol relieves them for a time; is often recommended by medical advisers; the patients know its power to diminish their passing discomforts, and push, beyond all reasonable bounds, their recourse to its aid. Flatulence and borborygmi of the intestines are common enough; and so are palpitation of the heart, syncopal feelings, and dyspnœa—the last, however, without any notable change in the ratio of respiration to pulse. Large quantities of pale, limpid urine, of low specific gravity, are passed; but this is also true of epilepsy and of many other diseases of the nervous system. With regard to affections of the generative organs, there is great discrepancy of opinion. Landouzy states that in twenty-six, of twenty-seven cases, there was some abnormal condition;[1] and, further, that of sixty-seven cases of Hysteria, the symptoms coincided with material alterations of the generative apparatus in fifty-eight, and that in nineteen cases the Hysteria was cured after the removal of the genital affection:[2] but it is, so far as my experience extends, the exception and not the rule to find any definite malady, or indeed definite complaint, in this direction; while in a vast number of cases there has been absolute health in all particulars relating to the reproductive organs.[3]

When men have presented hysterical symptoms, there have always been, in my practice, considerable deterioration of the general health, an impaired nutrition, and a feeble circulation, with exhausted brain.

(*b*) Paroxysmal Symptoms.—The attacks of hysteric convulsion do not pass through stages that can be defined, like those of epilepsy, to which they sometimes bear a rude resemblance. They differ widely in degree of intensity, but have a general similitude *inter se*, and can rarely be mistaken in either their slighter or severer form. When slight, they are but an exaggeration of the interparoxysmal state; when severe, they have been confounded with certain grave diseases.

The following may be regarded as the description of a severe attack: A patient is talking vehemently, often unreasonably, and is agitated in manner; she is crying or laughing, or both, and perhaps apologizing for or lamenting her weakness; friends are either scolding or condoling, and sometimes there is a combination of both modes of domestic treatment; some real or imaginary grievance is uppermost in the mind and the conversation, and is not "met" or removed by the endeavors of the friends. Suddenly the patient gives a scream, or makes a spluttering noise, appears to lose voluntary power and self-control; she falls down with snorting breathing, and a quasi-tonic contraction of the muscles of the extremities and the trunk. She makes hideous grimaces and outrageous noises, throws her limbs about in a disorderly manner, utters incoherent sentences, adopts histrionic attitudes; complains of her throat and stomach, and breathing; appears exhausted, or faint, and sometimes stupefied; occasionally she seems to lose her consciousness, and then, after a fit of "crying," to be "herself again." The whole paroxysm may last for a few moments only, but more commonly it is of much longer duration; a number of absurd gesticulations and irregular convulsive movements lasting from a few minutes to three or four hours, after which the patient seems worn out, and falls asleep.

These points may be observed during the attack: There is rarely absolute or sudden loss of consciousness; the patient does not fall in such manner as to hurt herself, or tear her clothes; there is somebody near who shall see the phenomenon; hysteric paroxysms do not occur during sleep, or when the patient is alone; there is something artistic in the mode of their approach—the hysteric patient gathers her robe around her, and falls gracefully; she appears to the casual observer to be unconscious, but there is not real or ab-

[1] Traité, p. 171. [2] Op. cit. p. 174.

[3] Niemeyer says: "Unter den Krankheiten des Uterus sind es ausser dem Infarct namentlich die Geschwüre des Muttermundes und vor Allem die Knickungen der Gebärmutter, welche am Häufigsten zu H. führen, wahrend bei bosartigen Neubildungen und bei destructiven Prozessen hysterische Zufälle weit seltener sind."—*Handbuch*, 2er Band, p. 356.

solute loss of sense or of perception; there is not the hideous distortion of feature observed in epilepsy, nor is there the dilatation of the pupil; the eyelids may quiver, and the eyeballs may be turned up, but there is no divergent strabismus, nor is there the wide-open eye. Examined carefully, the physician may observe that the patient not only sees, but looks; the eyes are often definitely turned towards objects or persons standing near, and then rolled up again towards the forehead: there is no bitten tongue, although there may be much foaming and spluttering with the mouth: the breathing is tumultuous and noisy, but there is no such absolute arrest of respiration as to cause asphyxia; and the irregular movements and noises that accompany the labored breathing may often be seen to be occasioned by the lips. The attacks last for an indefinite time, are followed by much apparent exhaustion, but not by real stupor.

Wherever the attacks pass beyond the description here given, it is probable that something more than mere Hysteria exists, and that the case borders upon the much more severe ailment known as epilepsy. In a few patients the two diseases coexist, and then the attacks bear some of the characters common to the two elements; but in the vast majority of cases it is comparatively easy to distinguish between them.

Mr. R. B. Carter, in an interesting book,[1] has described the hysteric paroxysm under three phases, giving to them the terms "primary," "secondary," and "tertiary;" implying that in the first instance the attack is quite involuntary, and is the product of violent emotion; that in the second it is reproduced by association of ideas; and that in the third it is deliberately "got up" by the patient. There does not, however, appear to me to be sufficient reason for adopting these phrases, as I am sure that in many instances all that can be said of any of the tertiary paroxysms may be affirmed with equal accuracy of the very first attack.

Hysterical Mania sometimes appears after an attack, and its features resolve themselves into an exaggeration of the condition already described as the "hysteric state." The patient is unmanageable, sometimes mischievous, and very often highly abusive; but generally is merely loquacious, unreasonable, and demonstrative in regard of emotion, and the attack speedily subsides under judicious treatment. It, however, exhibits a great tendency to recur; and hysteric patients sometimes become, for a time, maniacal without going through a paroxysm of convulsion.

[1] On the Pathology and Treatment of Hysteria, p. 43.

Hypochondriacal symptoms are met with, and are by no means rare, in cases of Hysteria; but it is quite easy to separate the two diseases.

Pathology.—Anatomical investigation has failed to show the presence of any organic lesion which is either so constant or so prevalent in Hysteria, that it may be justly regarded as its cause. Pathological examination has been equally unsuccessful in its attempts to explain the disease by a reference to the disturbed function of any one set of organs. It is common to find some derangement of the digestive, the assimilative, or of the reproductive systems; but these may exist without Hysteria; and *vice versâ*, that disease may be present when those bodily functions are healthily performed. There is, however, one thing common to all cases of Hysteria, and that is a perturbed condition of the nervous system. The essential character of this morbid state is an exaggeration of involuntary motility, and a diminution of the power of the will; the emotional, sensational, and reflex movements are in excess, while the voluntary are defective. The outcome of such a condition is seen in the mode of life of the hysteric patient. The will is determined by anything rather than by judgment, while ideas, feelings, and fancies exert an undue influence. Sensations are often morbidly acute, are uncorrected by any careful discrimination, and thus they increase the evil. Reflex movements, which in health are under some control, are not only exaggerated in their individual intensity, as a part of the hysteric state, but, from the weakness of volition, are allowed to run such riot that they pass beyond all bounds of healthy influence.

It has already been shown that the hysteric condition is somewhat analogous to that of the earliest period of decay, whether that be the result of age or of degeneration from disease. Hysterical symptoms are common enough in softening of the brain, and also during the course of, or convalescence from, exhausting diseases. They may break out suddenly, from a loss of blood, of food, or of rest, and they may occur as the immediate sequel of some violent shock, mental, moral, or corporeal. Whatever weakens the individual generally may bring about this state of nervous disturbance, may alter the relations of the several nervous functions; and this is, I believe, the true pathology of Hysteria, a disease which is more closely associated with affections of the nervous system than with those of the generative organs, although it is well known that the latter may and do exert a marked influence upon the former.

The hysteric state is essentially one of mental perturbation; and it is brought

into existence, if not inherited, by those conditions which are the most active in producing disorder of the mind: in the male sex by worry, anxiety, over-work, late hours, accidental injuries, and dissipation; in the female sex by vexatious emotions, want of sympathy or success, disappointed and concealed affection, want of occupation, fear, and morbid conditions, or supposed morbid conditions, of the reproductive system. The latter are sometimes the coincidents, but I believe much more commonly the effects of Hysteria than its cause. Their relation is by no means constant in existence, and is most variable in kind.

It would appear that the nutrition of the whole nervous system is changed, but that the change is of such kind that it passes beyond our power of recognition, except in its physiological or pathological effects. We cannot see degeneration of tissue here, or too rapid a metamorphosis there; but we can witness the effects of such morbid processes, in movement, in secretion, and nutrition, and we can observe some of the ulterior results of such changes, in emotion and sensation.

The influences exerted by emotion upon secretion and nutrition have been well shown, in their relation to Hysteria, by Mr. Carter,[1] and the inter-relations of physical, mental, and moral life have been very ably treated by Mr. Hovell in a more recent publication;[2] but the primary fact in that condition which we term Hysteria, would seem to lie behind all that is referred to in these considerations, and to consist in that special morbid change of the nervous centres, which either gives to emotion an undue influence, or removes the limitations of its action. There are divers links in the chain of causes and effects, but there is one link at which, in fixing upon the pathology of Hysteria, we must stop and say — all behind this is cause, all beyond it is symptom or effect; here is the one point which determines the hysteric result. Up to this link we find the causes of chorea, of epilepsy, and of insanity, together with and identical with those of Hysteria; beyond it we find neither chorea, epilepsy, nor insanity, but what we term Hysteria: in that link, therefore, we must seek for and find, if we can, the essential fact of the disease. I do not say that we have found it; still we are much nearer its discovery than we were fifty years ago: but I think it better to state, in general terms, wherein this morbid condition lies, than to lose sight of that point, by regarding some outlying facts, and attaching undue importance to certain frequent lines of apparent causation and effect. There is nothing to prove that the vaso-motor or sympathetic system of nerve-fibres is primarily at fault; on the contrary, it often appears that the secretions and the general nutrition are late in suffering; and that the earliest departure from health is to be found in the disturbed balance of mental and emotional operations. If it be held that every change in every organ and every function is, more or less directly, determined by a change in the vaso-motor nerves, Hysteria may be driven theoretically into this "sympathetic" corner; but, when it is made to go there, it will find itself in company with almost every ill to which flesh is heir—with tubercle and corns, with cancer and ataxy. If the "vaso-motor system" furnishes the agency by which all departures from health find their expression, we have yet to discover the nature and cause of those special changes in its action which lead to these particular results.

The most general expression that we can give to the pathology of the hysteric state is, perhaps, this, that it is a malnutrition of the nervous system, so distributed that its higher functions are relatively impaired and subordinated to the lower—that there is diminished power of the former and increased activity of the latter.

Every one knows that, in health, there are numberless processes which are quite familiar to the mind, but which appear very strange when described in technical language. A physiological or pathological discovery is sometimes nothing more than the translation into scientific terms of a "well-known saw;" and what may now be said upon the pathology of Hysteria is little more than such translation. Slight emotional excitement, such as shyness, trifling vexation, or moderate pleasure, may flush the face, quiver the lip, and make the breathing "panting;" strong emotion, such as terror, intense anger, or disgust, may blanch the cheek, fix the jaw, parch the mouth, and hold the breath. Moderate distress may "find relief in tears;" but when grief is deepest the eyes

[1] Op. cit. p. 5 *et seq.*

[2] Medicine and Psychology, p. 56, &c. The gist of Mr. Hovell's argument lies in the following sentence: "The nutrition of the body is not affected, mental power is not impaired, although it may be suspended, innervation is deranged, for the generation of nerve power is feeble, and its distribution is irregular; but it is the sympathetic, the vaso-motory system, the moral power, that is at fault: either from exhaustion of the physical strength of the sensori-motor centres, or because, perhaps most frequently, the purposes of life are in some respects disappointed, and the paresis of disappointment not only saps the strength, but, at the same time that it brings low the nervous system, also renders it peculiarly liable to irritable excitability from opposing and aggravating causes." P. 70, op. cit.

are dry. The postures of the various emotions are known, and have been studies for the artist in marble or on canvas, and for the actor on the stage. If we express this physiologically, we say that emotions lead through certain nerve-tracks to the contraction or elongation of some muscular fibres; that the vessels are dilated in the one instance, so that more blood than usual passes through the organs; and are diminished in the other, so that the circulation is arrested; in the former, occasioning an excess of secretion, in the latter, a defect: that the muscles are spasmodically fixed in one condition, and in another are relaxed; but what we want to know is the primary fact leading to such changes, when they are unusual in degree or persistence, and morbid in kind.

Some individuals, we say, have more "control over themselves," or more "presence of mind," than others; that A never shows what he feels, while B never hides, and never can hide, anything; and if we translate this into technical phraseology, it is but to say that the one is of "phlegmatic," and the other of "nervous temperament;" that A is a stolid, resolute individual, and that B is somewhat "hysteric;" but here again we do but throw the question one step backward.

There is an old proverb to the effect that "it is the last straw that breaks the camel's back;" and if we put this into other terms, it often means but this—that an individual has for a long time gone on bearing what was too much to bear healthily, that he has struggled against it, and by forced effort has made everything appear quite tolerable or even easy; but at last he "breaks down" from some "shock," and then all the "wear and tear" comes out, and friends see that he had done or borne far too much before. Sometimes what is held to be "shock" is a mere trifle compared with the rough handling that had been previously endured without complaint; it is simply because it, a mere "straw," fell upon the already overburdened soul that the great crash came, and that then—all power of resistance being gone, the "back broken," as it were—the brave sufferer was prostrate, crushed, gave way, and the pent-up tide he had kept back so sternly broke through destructively.

In some there is weakness at the outset, congenital, or acquired; in others there is weakness, but it is induced by long patience, vexation, care, or trouble, that have at last done their work, and a work which it may take years to undo. But here, again, we have yet to learn why, out of a hundred individuals similarly exposed, one becomes hysteric, another epileptic, and a third maniacal.

If we refer to the etiology of Hysteria, we shall see that all the facts point in one direction; if we look to the symptoms, we shall see that they are partially explained. In the female sex, at certain ages and under certain conditions, Hysteria is most common, for it is but an exaggeration of that which constitutes the normal characteristics of that sex; in the male sex it is met with when circumstances have gradually converted males into the condition of the other sex; *i. e.*, when emotions have been so played upon that they have, at last, broken through the force of resistance, which held out for a long time bravely, but at length gave way.

The essential fact of Hysteria, then, is the distorted balance between voluntary and involuntary power; volition is defective; emotional, sensational, and reflex activity are in excess; and this distortion may be brought about by the many and divers circumstances of age, sex, position, employment, and the like which have been enumerated in the section on etiology; but the precise nature of the change which is the efficient cause of such distortion—*i. e.*, the primary physical fact in the pathology of Hysteria—has yet to be discovered.

Diagnosis.—If the symptoms already described be borne in mind, and the history of each case be carefully considered, there is not much difficulty in the diagnosis. A physician called in on an emergency may have, however, to distinguish Hysteria from several diseases which it simulates.

From *Epilepsy* it may be separated by negative characters. There is neither the suddenness of attack, the absolute loss of consciousness, the dilated pupils, the complete asphyxia, the bitten tongue, nor the reckless injury of either the person or the clothes. The patient "looks about," the attack lasts longer, there is much sobbing and crying, much exhaustion, but no perfect stupor. The interparoxysmal state of the hysteric patient exhibits features not met with in epilepsy, and *vice versâ*. [See *Hystero-epilepsy*, in this volume.—H.]

From various inflammatory affections, such as *Peritonitis*, *Laryngitis*, and *Arthritis*, hysterical symptoms may be distinguished by a careful use of the thermometer, which fails to show any rise of temperature. Again, the tenderness of parts complained of may be seen to be ideal rather than real, and to bear relation to the skin quite as much as, and often more obviously than, to the deeper tissues. When there is loss of voice, this has the characters already described; there is no attempt made to whisper loudly; the failure is evidently not in the apparatus of voice, as a mechanical production, but in the will to put that ma-

chinery into play. A laryngoscopic examination will show that the throat is healthy, or is simply relaxed—the vocal cords being widely separated, and slight effort being made for their approximation. The pulse-respiration ratio will further show that although the breathing may be tumultuous, there is no real dyspnœa. *Phantom tumors* may be removed by the inhalation of chloroform, while palpation and percussion usually reveal the nature of their constituents.

Organic diseases of the nervous centres, which are sometimes simulated by hysteric paralyses and anæsthesiæ, may be excluded by the conditions already described, when detailing, in the section on symptoms, the mode of their development. Usually the phenomena presented are inconsistent with the idea of any definite disease of either the cerebrum or the spine; the paralyses are imperfect in development, vague in their distribution, and changing in their locality; they are not accompanied by the alterations of nutrition, or of electric contractility or sensibility which are proper to other affections; and the history of the case will usually reveal their true nature. The walk in hysteric paralysis has already been described; but it is further to be observed that the patient does not look at her feet, as those who are ataxic do, but looks around about her to observe the effect of her performance. The ataxic or paraplegic patient tries to walk; the hysteric girl tries to show that she cannot use her limbs: if the former forgets himself, he falls; if the latter forgets herself, she walks.

Neuralgia, when of hysteric origin, has not the real intensity of the genuine disease, as may be proved by withdrawal of attention. There is also an absence of those "painful spots," which are present when Hysteria does not complicate the case. The distribution of pain described by hysteric patients is, moreover, often so wide of all relation to anatomy and physiology, that its true nature may be recognized.

Prognosis.—When once established, Hysteria is very difficult to cure, and this is true under all the conditions of causation. The most difficult cases are those in which it is but an exaggeration of a constitutional defect, inasmuch as it is impossible to cure the malady without changing the individual, and this is by no means an easy task. If the hysteric habit be natural, or have become a "second nature" by long existence, the prognosis is *pro tanto* bad: if it be something quite unlike "the former self," or if it have been only recently developed, there is much room for hope. If the cause exist in the patient, *i. e.*, in the essential features of the individual character, comparatively little can be done; if it lie in external circumstances, much may be done, provided that those circumstances can be changed. If there be definite organic disease, and this be of such a character that it is amenable to treatment, prognosis is so far favorable; but if there be no such disease, and *à fortiori* if the general health be good, the prognosis is unfavorable.

Hysteric symptoms, such as paralysis, aphonia, and the like, are often easily removed when they are of recent origin; but when they have existed for many months, the prognosis with regard to them is unfavorable. It is not, however, hopeless; for in some cases, of even many years' duration, there has been amendment, and—but more rarely—cure.

The prognosis in Hysteria depends, therefore, mainly upon these two things—the nature of the "cause," and the freedom with which treatment may be employed. No human being can cure, the physician can do but little for, one who is born hysteric; *i. e.*, for one whose disease is but an exaggeration, and sometimes only a slight exaggeration, of her habitual, constitutional state. Education might have accomplished much in childhood, but often when the physician is called in, the grooves of life are worn so deeply that he cannot change them, and all that is possible is to soften their sharp edges, or to retard the movement which he can neither stop nor guide. In such cases the prognosis is unfavorable. When Hysteria is an accident, is unlike the earlier promise of the individual, or when it is brought about by long, and at last unendurable pressure from without, then there is much room for hope. When the treatment of the hysteric patient is cramped by the anxieties of friends—limited, in this direction, and in that, by frightened, too sympathetic, or unwise relatives—the prognosis is unfavorable; but when the circumstances are such that the physician can control them all, much hope may be entertained. Upon these points, rather than upon the special character of the symptoms, the prognosis turns. When Hysteria is a disease, and the physician has given to him a *carte blanche* to treat it as he deems best, the patient may be cured: but when it is a constitutional peculiarity, and the physician is checked at every turn by anxious friends, the case is hopeless, and might as well be left alone.

In regard to all special symptoms, the prognosis is more favorable than it is in those diseases which Hysteria simulates; but even here the general principles just stated are the most trustworthy guides in our attempts to forecast the future.

TREATMENT.—The old copy-book maxim, "Prevention is better than cure," expresses but a small portion of the truth in regard to the management of Hysteria: prevention might be easy; cure is often almost impossible. It is not within the scope of this work to describe generally the processes of a healthy education, but some things that are special must be said with regard to prophylaxis.

Bearing in mind the pathology of the disease, it is highly important that its earliest indications should be recognized and combated. When there is a tendency to Hysteria in early life, these things are necessary:—

1st. A strenuous effort to draw the person "out of himself," or "herself," and to develop the faculty of self-control. This should be done, not as an occasional or spasmodic effort, but as the business and prevailing arrangement of daily life; and while it is done, and in order that it may be done, the predisposed person should be unconscious of the process. A child who is "peculiar," disposed to be taciturn, loquacious, "excited," or unduly gay; or who is very readily "upset," and is "so sensitive" that parents and others are "afraid to tell her" this or that, "for fear that it should make her ill;"—one who is "impulsive," and disposed to find fault with herself without just ground; one who is "shy," and hides herself; one who is morose, and who thinks herself "misunderstood;" or one who is retiring, and shuts herself out from the sympathy she craves for;—should be carefully watched, tended, and unconsciously guided away from self, and into some line of feeling, thought, and action, which may interest the mind without fatiguing the body. The worst thing that can be done is that which makes the patient know and feel that she is thought to be "peculiar." Sometimes such treatment is gratifying to her and she likes it—it is easy, and it "seems kind" to give it—but it is radically wrong. Anything that looks like harshness, rigid discipline with a view to improvement, or want of sympathy from want of "understanding her feelings," is shrunk from, or resented by, the patient, and is worse than useless. At the same time, all exhibitions of a want of self-control should be checked, and much of this kind may be done in the nursery, and long before Hysteria is dreamt of. Kindness, firmness, and obvious recognition of "tender points," with judicious, sympathetic, and wise regard to them, may do much to avoid future evil; and sometimes the qualities of mind which will produce such treatment may be met with in a parent, a sister, or a governess. The physician should guide, in little details of daily life, those who have the management of such a child. The object is to make her feel that she is understood and cared for; and the best mode of attaining this object is often indirect in its operation. The mind and the heart should be engaged in some healthy pursuit; interest should be awakened, and exercised in anything—it matters not what—outside the individual; self should be lost sight of, and life made useful.

2d. The bodily health should be most carefully regarded, and this without any admission or appearance of anxiety on the part of others. The points that require attention are the following:—(*a*) *Diet*, which should be sufficient, wholesome, and easy of digestion, avoiding too long an interval between meals, and observing an especial regularity in the times at which they are taken. (*b*) *Rest.* The hours of sleep should be long, and those of rest or lying down not too long. Hysteric girls, or those who are disposed to become such, are in the habit of reading at night and of lying in bed in the morning. Such habits should be broken,—not roughly, for the sake of breaking them, and of "doing something disagreeable," but—by supplying a reason and motive for a different mode of life. (*c*) *Exercise* should be taken regularly, and in the open air, as much as possible without fatigue; and here, again, the exercise should have some other apparent object than a mere piece of tedious hygiene. (*d*) *Recreation* should be ample and merry; but all such things as precocious or preternatural excitements should be avoided. (*e*) *Study* should be systematic and disciplinary, but varied and interesting, and made to subserve some purpose which lies, obviously, outside of mere personal accomplishment or pleasure. (*f*) The various functions of secretion, excretion, and (if they have commenced) of menstruation, should be regulated; and this, again, should be done, as far as possible, without any particular notice being taken of the fact that they may be unhealthy.

3d. Some motive or purpose should be supplied which may give force, persistence, unity, and success to the endeavors of the patient. This is sometimes very difficult to manage; but it is less difficult in early life and in predisposed persons than it is in those who are older and have already shown definite symptoms of the malady. Still, much may be done by those who have a little ingenuity in detecting character, and a great deal of perseverance and determination in carrying out their wishes. The patient should be led to feel that the object is in itself desirable; she should never think that it is suggested for her own treatment or benefit. If she is urged on the former ground, she may do much; if on the latter, she fails entirely. The hysteric patient is often most thankful for and happy in the idea that she is

doing good to others, and she will take great pains to make her efforts successful and pleasing; but she hates the notion of doing anything of such kind as a mere means of self-cure, thinks that the doctor who recommends them is grossly ignorant of her real wants, and that the friends who urge her onwards are singularly stupid or unkind in their advice. To "make an effort," simply because told to do so for her own sake, is sheerly detestable to the hysteric patient, and is sometimes as impossible as it is distasteful; but to exert herself, almost unconsciously, because a motive is supplied, is scarcely felt to be an effort. The patient does what she herself is surprised and pleased with, and derives great benefit from the process.

When the symptoms of Hysteria are developed, the treatment should be conducted upon the principles already laid down with regard to prevention. There is, so far as I know, not one single drug which exerts any specific action on the disease; but there are many medicines which may be used with advantage for the relief of associated disorders. These do not require any special notice here, further than to say that if the patient be anæmic, iron is useful; if deficient in general nervous tone, quinine, strychnia, and vegetable bitters may do good; if there be indigestion with much flatulence, bismuth, charcoal, and alkalies, or mineral acids with light bitter infusions, may give relief; if there be constipation, mild aperients may overcome the difficulty; if there be menstrual derangements, they should be treated upon general principles.

The whole list of anti-hysteric remedies —such as musk, castor, valerian, asafœtida, and the like—appear to have this one property in common, that they do no good, and delay the real treatment of the case, which is not one of "nauseous gums," but of mental, moral, and social management.

Painstaking appreciation of the patient's own feelings; determinate assurance that the disease is a real thing, and no idle fancy; strenuous effort to help the patient in weakness, and to set her right when wrong; fertility of resource in little things; a cheerful but not boastful, a sympathetic and calm, but neither condoling nor anxious, manner; and a strong will, with patient work and tact, may do very much, and may often cure. But the physician should see that he manages his patient, and that all that he has attempted to effect is not undone by frightened relatives or anxious friends.

There are some drugs which are useful under special circumstances; and these are opium, or morphia, where there is loss of sleep, or persistent pain; and the diffusible stimulants, where there is a great tendency to recurrent spasm. Morphia may be administered most effectually, for the relief of pain, by hypodermic injection; it may be given by the mouth, with light food, when there is want of sleep. Chloric ether, ammonia, and musk, often relieve the tendency to spasm; and in some cases Indian hemp has proved of service when other medicines have failed: in the large majority of cases, however, I have found Indian hemp of but little use. Asafœtida, in doses of thirty grains, three or four times daily, is of service in some cases. Bromide of potassium has appeared to me to be singularly useless in Hysteria, failing to relieve either the attacks or the symptoms which exist between the periods of their recurrence.

The *attacks* of hysteric convulsion may be arrested by a plan suggested by Dr. Hare—viz., that of forcibly preventing the patient from breathing for a certain time, by holding the nose and mouth. The effect of such constraint is to make the patient, when allowed to do so, "draw a long breath," this vigorous inspiration being usually followed by a relaxation of all spasm, and a disappearance of the fit. Some attacks are of such short duration, that there is neither occasion nor time for this mode of treatment; but when they are prolonged, I have seen it notably useful.

Dashing cold water on the face and neck may sometimes succeed in doing imperfectly that which Dr. Hare's treatment accomplishes effectually; but even cold water is not always at hand, and when it is—in addition to its other inconveniences in regard to carpets and dress—it often fails to do any good.

A calm manner, the absence of all appearance of alarm, and of either scolding or distressing sympathy,—all of which things the apparently unconscious patient observes much more accurately than do her frightened friends,—will sometimes bring a fit to a speedy end.

Some *special symptoms* of Hysteria require special treatment. *Aphonia* may often be cured by electricity; and the mode of application which has appeared to be the most useful is that of giving sparks to, or taking them from, the larynx. An ordinary plate or cylinder machine may be used for the purpose, and either the patient or the physician may be insulated, and the sparks taken from or given respectively by a brass knob. The interrupted current from a magneto-electric or volta-dynamic apparatus may be used so as to pass the shocks through the throat, or a shock may be administered from a charged Leyden phial. Under all these circumstances, the voice is sometimes instantaneously restored. But when Aphonia has been of very long duration, and such measures fail to affect it, good may be done by directly galvanizing the vocal cords, in the manner proposed by Dr.

Morell Mackenzie. Further, I have known a strip of blister round the throat recall the voice when all means of electrifying have failed.

Paralyses are treated very successfully by Faradization, and by passive movements and frictions, employed by a well-instructed nurse. The electricity should be applied to the muscles affected, and also to the skin which covers them. I have, however, found no mode of treating hysterical paralyses comparable in efficiency with that of placing narrow strips of blister completely round the affected limbs. This method of treatment has succeeded perfectly and rapidly, after all other plans have failed.

Rigid Contractions may be relieved by the continuous galvanic current, but much more successfully by the inhalation of chloroform, and the adaptation of some apparatus to maintain extension when the effect of chloroform has subsided. Passive movements are also of much service in such cases. Drugs may be taken in almost poisonous doses without relieving the tonic spasm.

The treatment of other symptoms must be conducted upon the general principles already laid down, and may be assisted by those local sedative measures to which allusion has been made.

ECSTASY.

By Thomas King Chambers, M.D., F.R.C.P.

By intense concentration on one object, engaging only a few of the intellectual faculties, the mind is liable to lose temporarily its sensitive and controlling power in respect to its other relations. In minor degrees this state is a matter of daily experience with us all, and in minds of average strength it does not seem to go beyond minor degrees. They do not wish or practise such intense concentration; they are able to do and feel all they want to do and feel without overtasking themselves. But there are some, either naturally weaker, and so incapable of full feeling without concentration, or else desirous of a higher degree of emotion than they are healthily capable of; and in these a condition may be adduced allied in some respects to catalepsy, and in some to hysteria—a condition certainly morbid, for it renders the patient unequal to the functions of social life, and is excited by causes which affect some and not others.

Sometimes the patient falls into a state of immobility, in which there is a passive reception of ideas, like that of the Midianite prophet "falling into a trance, but having his eyes open," a state he evidently considered rarer than that of a mere dreamer of dreams. Hoffman[1] describes an ignorant peasant woman of twenty-four, after a fortnight's course of exciting sermons, remaining motionless for more than an hour; after which she gave a few sighs and returned to herself, having seen or heard nothing of what was going on about her, but having had exquisite revelations of the love of Christ. During forty days she had a hundred returns of the same state, which would always be induced by a recitation of a few verses of the Scriptures concerning the love of God. During the fits the pulse remained quite natural. They were finally removed by a change of air and scene, after bleeding and stimulants had failed. The followers of St. Francis, in the days of faith, were often brought into these ecstasies by fasting, meditation, and abstinence from varied intellectual exertion. Nowhere is it so strikingly depicted as by the Tuscan artist Cigoli—a man of by no means lively fancy, for, except an Ecce Homo, he painted nothing well but ecstatic and starving Franciscans. So he is probably truthful. After visiting each Florentine gallery it is difficult to expel from one's memory these strange figures, of marble paleness, kneeling, but sunk on one side from exhaustion, the eyes open, the pupils fixed, the arms extended to embrace the beloved vision, the livid lips parted in smiles, showing the parched dark mouth, the breast heaving with delight. It is necessary to add only one medical fact, derived from M. Sagar's observation[1] of a Capuchin in this state; namely, that the pulse was pretty strong.

One main psychical difference between

[1] Medicina Rationalis, vol. iii. p. 50.

[1] Quoted by M. Tissot, Œuvres, vol. xiii. p. 4.

this and catalepsy lies in the visions which are recollected afterwards with all the force of reality. In true catalepsy all memory of what is done during the fits is completely wiped away—a phenomenon which may assist in the detection of impostors.

A more common development of Ecstasy is where the sufferer feels "borne in upon him," a desire to communicate to others the feelings he is sensible of himself, instead of reserving the experience till afterwards. The simplest instances of this are the scenes which take place from time to time in the Primitive Methodists' or "Ranters'" chapels. The congregation groan and respond to the feelings of the preacher, second his words with their own experience, and various members work themselves up into a state of excitement, repeating the last words of the sentence, "Salvation! salvation!" or whatever else it may be, with continually increasing earnestness, till they end in shouting and sobbing.

The next stage or form of Ecstasy is where the enthusiasm tries to express and exhaust itself in bodily movements. One of the most famous instances of this is the spasm of the *Convulsionnaires de Saint Medard*, a disease which was by neglect allowed to attain most formidable proportions in the last century. It owed its origin to the discussion of dogmas whose character one would never have expected to have stimulated feeling; namely, those which were in dispute between the Gallicans and Ultramontanists on the occasion of the bull "Unigenitus." A popular Gallican deacon, named Pâris, died in 1727, and was buried at St. Medard; and on his grave people began to fall into convulsions, be affected with *clairvoyance*, preach, jump, spin round with incredible rapidity, run their heads against the walls, &c. &c. Of no avail was Louis the Fifteenth's proclamation, which some one parodied—

> "De part le Roi! Défense à Dieu
> De faire miracles dans ce lieu."

In spite of it Convulsionism grew into a sect, and was at last only smothered by the French Revolution.

A transition between this form and the last is exhibited in Revivalism; and its reduction to a certain system and order is shown in the ceremonies of the Jumpers of New York and the Dancing Dervishes of Cairo.

Sporadic cases of this Ecstasy not infrequently occur in the experience of the promulgators of new or arousing doctrine; but judicious discouragement has usually prevented its becoming historical. We meet with it in the biographies of such men as Samuel Wesley and Edward Irving; and a valuable lesson is to be learnt from their wise mode of dealing with it, especially the former.

The mention made of this disease being used by dishonest or foolish people as a bond of union for religious sects, leads to a point in its history which constitutes the main interest it possesses for practical consideration. It is eminently communicable, especially in its more active, noisy, and ridiculous forms.

There is not much worth reading in the Pastorals of Longus; but one expression of his—ἡ τῶν ὀφθαλμῶν ἀλωσις "the contagion of the eyes"—is so picturesque and truthful that he deserves the credit of it. Through the eye instinctive imitation, or sympathy, directs the first intellectual and corporeal efforts of the infant, and makes him grow up in the image of his kind; and, as Longus felt, it is by such means that two adult souls get bound into one. So also through the eye flashes in that morbid state in which a nervous malady fetters the normal powers of control, binding them up as with an electric spasm, and allowing the lowest animal emotions to exhibit themselves. An initiative compliance, a voluntary surrender of the gates of the soul, is doubtless necessary at first: but with each yielding the energy is weaker, and this natural and healthy sympathy may pass into an actual disease of the mind, in which the power of the will is quite in abeyance.

In this way, from the accidental eccentricity, convulsion, or insanity of often a single person, the strange spasmodic epidemics of the Middle Ages arose. We can easily understand the disorganization which they produced among the lower orders, when we read that a few months after a new appearance of the "dancing mania" at Aix-la-Chapelle, on St. John's Day, 1374, there were as many as eleven thousand dancers in the streets of Metz. In this instance, at least, the outbreak took its rise in the scandalous midsummer revels, which had been handed over from paganism to the ascetic Baptist's festival, in spite of the protests of the Church from St. Augustine to Pope Boniface. The origin was a disreputable one, so the dancers hastened to avoid the inference by placing themselves under the protection of St. Vitus, one of the fourteen "Helpers in need."[1] For full 150 years it was in Germany a most serious plague, of which a full history is given in Hecker's "Epidemics of the Middle

[1] Most travellers in Flanders and Germany are probably familiar with the shrines of the fourteen "Nothhelfer" or "Apotheker" saints, spiritual specialists, of whom one cures toothache, another stone, another cancer. It is a curious fact that the regularly educated physicians, St. Luke, St. Cosmo, and St. Damian, are not among them.

Ages." As a translation of that graphic description is published by the Sydenham Society, it is unnecessary to do more than refer to it those who are anxious to trace to its most disgusting results an extreme indulgence in uncontrolled sympathy.

Spasmodic epidemics seem to have appeared in Italy about the same time as in Germany, but to have been for some time confined to Apulia. At the end of the fifteenth century they spread further, and coincident with the spread there seems to have been observed an increase in the numbers of the tarantula or ground-spider. The two facts became associated together, and a panic flashed like lightning through the country that this creature was communicating the disease by its bite. Of "tarantism" in Italy, pure fright was as potent an exciting cause as superstitious fanaticism had been of the "St. John's" or "St. Vitus's dance" in Germany. And for this reason it affected a higher and better educated class of society. Even a sceptical prelate did not find his freethinking a protection. Quinzato, Bishop of Foligno, having allowed himself in joke to be bitten by a tarantula, fell into the disease, and could only be cured in the undignified method adopted by vulgar laymen. (Hecker.) He was obliged to kick off his shoes (such at least is the necessary prelude nowadays) and dance the tarantella.

As an epidemic, tarantism has long disappeared, but sporadic cases are said still to occur, and hysterical women will persuade their gossips that they have been bitten by the tarantula, and that they cannot get rid of their mental fidgets without an immoderate indulgence in the traditional cure by dancing.

Of the exciting causes of Ecstasy, and its allied spasmodic epidemics, the most common is perverted religious feeling, of which elaborate examples are given in the "New America" and other works of Mr. Hepworth Dixon. The reason is that, since the diffusion of Christianity, religion has a more powerful hold upon the emotions than anything else. But all historians agree in attributing much influence also to venereal excitement; and in pre-Christian times, when in default of revelation men worshipped their incarnate passions, we have from the pen of Sappho a description of a purely erotic ecstasy, which can never be paralleled again. In the case of tarantism it seems to have been cowardice which was the exciting cause.

The history of the treatment of exaggerated instances of this disease is instructive to us, not from the likelihood that any of this volume's readers will be called upon to undertake it, but as a suggestive guide for the management of allied states, semi-mental, semi-corporeal, which are as commonly, as it is rarely, under medical charge. Allusion is especially intended to two—*chorea*, the heir to the name, and in a diluted degree to the nature, of the mediæval mad dancers who put themselves under the protection of St. Vitus; and to *hysteria*, which being more usual among women than among men, has acquired a name derived from a part of their bodies anciently supposed to produce the symptoms—a nomenclature often leading to bad practice.

1. If taken in time, ecstatic and emotional exhibitions are capable of being forcibly repressed. For example, in Unst, the most northerly of the Shetlands, an epidemic of convulsive fits occurring in sermon time began to prevail in several parish churches. At one of these, Northmaven, the disease was cut short by a rough fellow of a kirk officer, who carried out a troublesome patient and "tossed her into a wet ditch." Nobody else caught it.[1] From what scandalous scenes Europe might have been saved had the first dancers on St. John's eve been "tossed into a wet ditch!"

2. A strict quarantine prevents infection. In 1796 an epidemic convulsion spread to twenty-four persons in Anglesea. Their landlord, Lord Uxbridge, consulted Dr. Haygarth, and by his advice all communication with the afflicted persons was prevented, and the plague was stayed, as he records.[2]

I have often had chorea and hysteria arrested in hospital wards on separating the patients thus diseased, who had been keeping up one another's malady by sympathy.

3. Order, rhythm, designed consecutiveness, and in short anything implying voluntary control, has a beneficial effect in this class of complaints. Doubtless when once Sappho had grown particular in winding up with a dactyle and spondee the pretty stanzas named after her, when Madam Guyon had learnt to be careful of her rhymes, and Saint Theresa had committed her devotions to paper, all danger of contagious enthusiasm was past. But it is only an intellect of above the average capacity that can undertake to reduce itself to order in this way. Those afflicted in Germany with the epidemic convulsions before alluded to, took to dancing, evidently with an instinctive feeling that rhythmical movement was a relief to their morbid sensations; but in that country the application of it as a mode of cure does not appear to have been turned to such good account by art as in Italy. The

[1] Edinburgh Med. and Surg. Journal, vol. iii. p. 439.

[2] Haygarth, "On the Imagination as a Cause and Cure of the Disorders of the Body." Bath, 1801.

tarantellas I have seen performed in South Italy are very complicated figures, accompanied by an amount of arm-waving, finger-snapping, simultaneous wriggling, slapping of hands, bumping of backs, and crossing one another's footsteps, that can only be accomplished by a strict adherence to time. And the time is marked by a tambourine or drum. These dances are probably much older than tarantism; but for the cure of it they became popular, and from it they got their name. It is impossible to doubt that they had a real influence, even over those who undertook them unwillingly, as in the case of the sceptical bishop above alluded to. The peculiar features of them, to which I should attribute their usefulness, are the marked time and intricate figure, by which they are honorably distinguished from the senseless rotatory embrace now called dancing.

I am sure I have seen decided benefit in hysteria from dancing reels, and there would probably be much more, if time were better marked and kept.

In chorea, marching in timed step is excellent practice for regaining the directing power over the limbs. In stuttering, which is a sort of chorea, spouting poetry before a looking-glass contributes much to the cure.

[HYSTERO-EPILEPSY.

By Henry Hartshorne, M.D.

It has long been a familiar fact to most practitioners, that some cases of Hysteria simulate or approach Epilepsy so closely that their diagnosis requires a very careful estimation of the whole history of each case. Latterly, Briquet,[1] and more especially Charcot[2] and Bourneville,[3] have elaborately described and analyzed cases which are regarded by some pathologists as a combination of the two maladies; although Villermay, Briquet, Tissot, Charcot, and others, consider them rather as examples of exaggerated Hysteria. Landouzy, Saunders, Anderson,[4] McLane Hamilton,[5] and others, also, have recorded cases having more or less resemblance to those of the Salpêtrière. It appears probable that the two diseases may sometimes be actually combined. In some cases Epilepsy is then the primary disorder, Hysteria being engrafted upon it, generally by some powerfully disturbing emotional cause. In other instances, Hysteria having previously existed, Epilepsy has been added, as it were, to it. Marriage, in some cases, seemed to be the immediately exciting cause. In others, the attacks (as in a case mentioned by Hamilton) coincided with the menstrual period.

A personal history of a kind likely to predispose to psychical as well as corporeal functional disorders, is stated to have occurred in connection with each of the most typical cases. Some were prostitutes; others had suffered extreme fright (*e. g.*, during the terrors of the Commune in Paris); or had been the victims of domestic tragedies.

Preliminary symptoms of the attack are nearly always observable, through one, two, or three days. These are, palpitation of the heart, the "globus hystericus," noises in the ears, visual disturbances, and (ascertained by palpation) *ovarian hyperæsthesia.* Strong pressure upon the affected ovary will sometimes arrest the attack. Charcot asserts[1] that ovarialgia is an important part of these seizures; and calls attention to the not uncommon empirical resort to pressure upon the abdomen, in convulsive affections of women, as long ago as the 16th century. Mercado, in 1513, advised abdominal frictions, and about the same time Monartès placed a large stone on the patient's belly during the seizure. Willits,[2] in the 17th century, made a similar recommendation, which was, later, reviewed by Recamier and Négrier. But most remarkable was the *secours* afforded to the "Convulsionnaires de St. Medard," in the 18th century, during their paroxysms. Several methods

[1 Traité Clinique et Thérapeutique de l'Hystérie, Paris, 1859.]

[2 Leçons sur les Maladies du Système Nerveux, Paris, 1872.]

[3 Iconographie Photographique de la Salpêtrière (service de M. Charcot). Par Bourneville et P. Regnard, Paris, 1878.]

[4 Brit. Med. Journal, Feb. 8, 1879.]

[5 Nervous Diseases, &c., Philada., 1878.]

[1 Op. cit., Lecture XI]

[2 De Morbis Convulsivis, t. ii. p. 34.]

were employed: 1st, striking the abdomen repeatedly with a heavy andiron, or a large wooden pestle; 2d, the two fists of a man being thrust, with all his might, against the abdomen; 3d, three, four, or five persons together getting upon the body of the "convulsionnaire;" 4th, long bands being passed around the body, so as to compress it when they were drawn right and left. Charcot ascribes the benefit of all these measures to pressure upon hyperæsthetic ovaries.

For an account of the hystero-epileptic paroxysm itself, we cannot do better than to have recourse to a description, by an eye-witness, of one of Charcot's patients at the Salpêtrière. Dr. Arthur Gamgee, of Owen's College, Manchester, thus narrates[1] what took place in one of the demonstrations occurring during a visit in August, 1878, to Prof. Charcot's wards, made by himself, in company with Drs. Virchow, G. Stewart Turner, O. Liebreich, E. Hart, and others:—

"*Hystero-Epilepsy; Absolute Hemianæsthesia with Right-sided Ovarian Hyperæsthesia; Induction of the Hystero-Epileptic Seizure by Peripheral Irritation; its Arrest by Compression in the Right Ovarian Region; Inhibition of Fits by continuous Application of Pressure.* — The patient, a young woman of considerable vigor and intelligence, is apparently about twenty-two years of age, and is very frequently subject to the most characteristic hystero-epileptic attacks. These attacks had been exceedingly frequent on the day preceding our visit, but had been inhibited by the systematic application of pressure to the right ovarian region, as will be more particularly mentioned in the sequel. They still continued to recur.

"Professor Charcot pointed out that the hystero-epileptic seizure, besides occurring spontaneously, can usually be induced with ease by some modes of peripheral irritation. In the present case, for instance, by suddenly "gripping" the skin of the breast on both sides, about on a level with the fifth rib, and midway between the anterior and posterior boundaries of the axilla, the patient instantly fell into the hystero-epileptic convulsion. The constancy with which the effect followed the cause was demonstrated over and over again to be absolute.

"Although the various phenomena of the hystero-epileptic seizure are known to many readers through the writings of M. Charcot, it may be not uninteresting to describe them with all minuteness as they were presented before us by this patient. The attack may be conveniently divided into three or four stages.

"*The first stage* followed the application of the peripheral irritation without the intervention of any perceptible latent period; its features were the following: The head was thrown violently backwards, the limbs and body became rigid, the respirations infrequent and stertorous; in a few seconds, the tonic spasms were succeeded by clonic spasms affecting the muscular system. A slight remission, lasting for a very few seconds, occurred, which was spoken of as a kind of *entr'acte*, and then commenced the second stage. The first may be termed the *epileptiform stage*.

Fig. 35.

Hystero-Epilepsy, Stage of Rigidity. (Bournevilla.)

"*The second stage* was characterized by extraordinary movements affecting the whole trunk. The back being somewhat opisthotonically arched, the body was thrown with great violence and astounding rapidity alternately on to the occiput and heels. This stage, which, like the first, is of very brief duration, is denominated the *phase des grands mouvements;* during its continuance occur the first hallucinations, to be afterwards referred to. The violent movements cease almost instantaneously, and then follows

[1 British Medical Journal, Oct. 12, 1878.]

Fig. 36.

Hystero-Epilepsy. Stage of "grands mouvements." (Bourneville.)

"*The third stage*, or stage of emotional attitudes (*phase des attitudes passionelles*). During this stage, the patient assumes successively the expression of face, the attitudes, and the gestures which portray varied emotions—intense and vivid. The varied emotional states will be distinguished in the order in which they occurred by letters.

"*a.* No sooner had the great movements

Fig. 37.

Hystero-Epilepsy, Period of Contortion. (Charcot.)

ceased, than, raising herself into a sitting posture, with clenched fists and menacing expression, the patient presented the most startling picture of one threatening;

but almost instantly the picture changed to

"*b*. The whole expression and attitude portrayed cowering, abject fear. Of no longer duration than *a*, *b* was followed by stage

"*c*. The patient now assumed an expression of absolute beatitude. It is impossible to describe the look of saintly happiness, as of one who realized the blessedness of heaven, which the patient presented. It was the expression which some of the old masters have impressed upon their saints and martyrs.

"But now occurred a change no less striking than the preceding.

"*d*. The expression of saintly happiness was succeeded by one of intense joy; the patient sees one whom she loves; she beckons to him to come, to come quickly; he has come. . . . Then succeed gestures which stamp this as the *phase of lubricity* or the stage of the emotional attitudes.

"*e*. Again fear takes possession of the patient; at first it is rats which she sees, and which she appears to fear the attack of, which evoke passionate exclamations of dread and disgust; then it is obviously the fear of some human being which oppresses her, and causes her to beg for mercy.

"*f*. There is no longer fear. The patient hears the strains of music; she is pleased; she herself begins to hum the tune, but only for an instant, for

"*g*. Her singing is followed by weeping, which is broken by reproaches addressed to her parents as the causes of her misery. This last phase (*g*) in the stage of passionate attitudes may be made to constitute a fourth stage, or a stage of recovery, in which hallucinations persist for a time."

Other cases present different features. Sometimes the clonic stage is followed, as described by Bourneville, by throwing out the arms at right angles from the body, as in the position of crucifixion.

Induction of "mesmeric" sleep, with insensibility to pain, and artificial somnambulism, is readily effected in some Hystero-epileptic patients. In them, also, the extraordinary action of metals, magnets, and galvano-electric spirals has been observed and recorded, by Charcot, Bourneville, Westphal, Dumontpallier, Luys, and others, to which allusion was made on a previous page of this volume.[1]

It is, as before said, the eminence of those who have witnessed these phenomena, and brought them before the attention of the profession, that entitles them to respectful attention. Much care has been evidently taken to eliminate as far as possible the element of deception. The transfer of hemi-anæsthesia from one side of the body to the other, under the influence (however explained) of metals, magnetic or otherwise, appears to have been clearly established. It remains, however, yet to be placed beyond doubt how much of this and the connected phenomena is due to physical, and how much to psychical impressions and communications. In any case, the study of Hystero-Epilepsy is of great interest, as furnishing a comparatively new chapter in the annals of Hysteria, to which, chiefly, its characteristics must be referred.]

CATALEPSY.

By Thomas King Chambers, M.D., F.R.C.P.

Name.—This word Catalepsy is derived from the Greek *κατάληψις*, a "seizure" or "arrest."

Definition.—Catalepsy is the name given to intermittent attacks of a suspension, more or less complete, of sensation and voluntary power, without convulsions, accompanied by a stiffening, general or partial, of the muscular system; so that the parts affected retain for a period of variable duration the position in which they happen to be at the invasion of the fit.

Description.—This is one of those pathological phenomena of whose anatomical cause we are entirely ignorant, and therefore it is best defined by its symptoms, instead of committing ourselves to any vague theory of its nature. It is best to call "Catalepsy" any attack which fulfils the conditions above named, and then we shall at once avoid all those discussions with which writings on the subject are laden about "true" Catalepsy and

[1 See article on Hysteria.]

"false" Catalepsy, and the separation into symptomatic and idiopathic, which we have no warrant for making at all. It is as much Catalepsy, and the ultimate morbid state is essentially the same, whether it is followed by a disease with another name, or whether it is followed by restored health.

The following account of a well-marked case by Dr. John Jebb describes the details of Catalepsy more graphically and fully than any I have yet read. He says:[1]—

"In the latter end of last year (viz., 1781), I was desired to visit a young lady who for nine months had been afflicted with that singular disorder termed a Catalepsy. Although she was prepared for my visit, she was seized with the disorder as soon as my arrival was announced. She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form a figure of death-like sleep, beyond the power of art to imitate, or the imagination to conceive. Her forehead was serene, her features perfectly composed. The paleness of her color, her breathing at a distance being also scarcely perceptible, operated in rendering the similitude to marble more exact and striking. The positions of her fingers, hands, and arms, were altered with difficulty, but they preserved every form of flexure they acquired; nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it as firmly as her limbs.

"Upon gently raising the eyelids, they immediately closed, with a degree of spasm. The iris contracted upon the approach of a candle, as if in a state of vigilance; the eyeball was slightly agitated with a tremulous motion, discernible when the eyelid had descended.

"About half an hour after my arrival, the rigidity in her limbs and statue-like appearance being yet unaltered, she sang three plaintive songs, in a tone of voice so elegantly expressive, and with such affecting modulation, as evidently pointed out how much the most powerful passion of the mind was concerned in the production of her disorder, as indeed her history confirmed. In a few minutes afterwards she sighed deeply, and the spasm in her limbs was immediately relaxed. She complained that she could not open her eyes, her hands grew cold, a general tremor followed; but, in a few seconds, recovering entirely her recollection and powers of motion, she entered into a detail of her symptoms and a history of her complaints.

"She informed me that she had no recollection whatever of what passed in the fits; that upon coming out of them she felt fatigued, in proportion to the time of their continuance; and that they sometimes lasted for five hours, though generally for a much shorter period.

"She further related, that the fits returned once or twice a day, sometimes more frequently; but that she was never troubled with them in the night. She sometimes lost her sight and speech, the power over her limbs and her intellectual faculties remaining unimpaired. The fits frequently attacked her without any previous warning; at other times, a fluttering at her stomach, and a fixed pain at the top of her head, occupying a part she could cover with her finger, announced their approach.

"Hysterical risings in her throat, appearance of fire, pains in her eyes, and not infrequently in her teeth, flatulence, a sense of weight in her stomach after eating, with convulsive motions in the region of that organ, were superadded symptoms of which she much complained.

"Her disorder was evidently exasperated at the approach of the catamenia, which were constantly present at the regular period. She was always much agitated previously to a storm of thunder, and every material alteration of the weather produced a sensible effect.

"After she had discoursed for some time with apparent calmness, the universal spasm suddenly returned. Her features now assumed a different form, denoting a mind strongly impressed with anxiety and apprehension. At times she uttered short and vehement exclamations, in a piercing tone of voice, expressive of the passions that agitated her mind, her hands being strongly locked in each other, and all her muscles, those subservient to speech excepted, being affected with the same rigidity as before.

"During the time of my attendance similar appearances were frequently exhibited.

"I was informed by the family of many particularities in the access of the disorder, all denoting its instantaneous effect upon the nervous system. She once was seized in my presence while drinking tea, and became universally rigid at the instant she was advancing the tea-cup to her mouth. Her tears sometimes flowed copiously, while every internal, as well as external, sense seemed entirely locked up in sleep.

"I will now proceed to describe the progress of the disorder, and the mode of treatment, before she was entrusted to my care.

"It appears that for many years before

[1] Appendix to Select Cases of the Disorder commonly called the Paralysis of the Lower Extremities, by John Jebb, M.D., F.R.S. London, 1782.

the access of the cataleptical symptoms, she had suffered much from violent headaches, particularly that species of headache termed clavus hystericus. Her spirits were easily discomposed. Her fingers, upon touching cold substances, would frequently lose their natural heat and feeling. Her habit of body had been uncommonly costive, but of late her bowels were much disturbed by every kind of laxative. Her nervous complaints were always particularly troublesome at the approach of rain and after a sleepless night.

"Her disorder commenced with hysteric fits; to these succeeded a delirium of several days' continuance, attended with slight shiverings, but no other sign of fever; the Catalepsy followed next in order, which at first affected her with only single fits, at a week or fortnight's interval; these gradually advanced in strength and frequency until by her own sufferings, and her sensibility on account of the anxiety of her friends, she was reduced to the most pitiable distress."

Then follow details of the treatment advised by Dr. Jebb, which was judicious and successful. An opium plaster to the epigastrium did good, but the last and longest continued prescription consisted of bark, gentian, and tincture of lavender, which she went on with till quite well.

This exceedingly well-drawn-up description makes one regret that the accomplished author had not continued to apply his pen to depicting the eternal truths of nature, instead of wasting it upon theological and political advocacy. It renders needless the repetition of stock cases which usually illustrate the subject.

Causes.—The most common exciting cause of Catalepsy seems to be strong mental emotion. When Covent Garden Theatre was last burnt down, the blaze flashed in at the uncurtained windows of St. Mary's Hospital. One of my patients, a girl of twenty, recovering from low fever, was woke up by it, and exclaimed that the day of judgment was come. She remained in an excited state all night, and the next morning grew gradually stiff, like a corpse, whispering before she became quite insensible that she was dead. If her arm was raised, it remained extended in the position in which it was placed for several minutes, and then slowly subsided. The inelastic kind of way in which it retained its position for a time, and then gradually yielded to the force of gravity, reminded one more of a wax figure than of the marble, to which Dr. Jebb compares it. A strange effect was produced by opening the eyelid of one eye; the other eye remained closed, and the raised lid after a time fell very slowly, like the arm. A better superficial representation of death it is difficult to conceive. The pupils, however, contracted sluggishly under the influence of light; and the pulse could be felt beating softly at both heart and wrist. She came round again by degrees in the course of the morning, and had no relapse; nor had she any manifestations of ordinary hysteria during her stay in the hospital, I believe.

Less acute but more long-continued mental emotion will sometimes cause it. The same year as that in which the last case occurred, I was attending for menorrhagia from relaxed fibre a young woman, aged twenty-two or twenty-three, who had been a governess in a family I was acquainted with. She was of an affectionate disposition, and had been rather coldly treated—"misunderstood," as it is called. The menorrhagia, too, had pulled her down a good deal, and forced her into involuntary idleness. One morning when I called to see her, she was in bed, unable to move, and scarcely capable of articulating. She said she was just recovering consciousness, but all the limbs were partially stiff, and the neck and back quite so, as appeared by raising her up with the hand at the back of the neck, when the body remained straight, resting on the heels. This state soon passed off, even while I was in the room. But the next morning I found her partially affected in the same way; the left side was rigid, and especially the left arm, which remained stretched out at an angle when so placed.[1] I observed that when I bent the arm, the deltoid contracted as it does when flexion is made by voluntary effort. She then told me that, though quite incapable of moving the limb of her own unassisted will, she thought she could do so if I bade her very strongly. And such proved to be the fact; for, on my rating her soundly and ordering her to get up, she at last obeyed. I explained to her what I believed to be the nature of the disorder, namely, a broken connection (to speak metaphorically) between the will and the nervous system; and that she must rejoin this broken link by painful exertion and violent determination. She had no further relapse.

In both these cases I convinced myself carefully that there was no deception.

Catalepsy is sometimes very brief and sudden. I have a young lady now under my care, for non-assimilative indigestion, of whom I received the following accounts from a mother of more than ordinary intelligence and powers of observation. She said that her daughter was fond of reading aloud, and that sometimes in the mid-

[1] M. Tissot mentions a similar case of partial Catalepsy affecting the arms in a man. (Œuvres, tom. xiii. p. 56.)

dle of a sentence the voice would suddenly stop, a peculiar stiffness of the whole body would come on and fix the limbs immovably for several minutes. Then it would relax, and the reading would be continued at the very word it stopped at, the patient being quite unconscious that a parenthesis had been snipped out of her existence, or that aught strange had happened. She grew much better under tonic and restorative treatment, and gradually ceased to have these singular attacks; but after about a month's interval, as she was one evening engaged in playing a round game of cards, she suddenly went off into a regular epileptic fit, which was followed by sleep, and she did not recover consciousness till the next morning. This fit could be accounted for by certain errors in digestion, and she has had no recurrence of it, or of the Catalepsy, though four months have passed over. So I hope it was epilepsy of an intercurrent or curable sort.

But sometimes the epilepsy preceded by Catalepsy is of a more serious sort. I remember a much-respected lecturer in this metropolis, in whom the *petit mal* of epilepsy assumed this form. He used to be attacked sometimes in the middle of a sentence, with his hand wielded in demonstration before his class. He would remain perfectly stiff for a minute or so, with mouth open and arm extended, and then resume his sentence just where he had dropped it, quite unconscious that anything had happened. After a time the seizures assumed the more usual and more fatal form.

This sort of short attack is not, however, always the precursor of anything so serious. Nor, if traceable to a material cause, is that cause necessarily in the brain. Van Swieten tells a story of a woman, forty years of age, who was roasting chestnuts in the frying-pan, and kept continually stirring them lest they should be too much scorched; in doing which, she was seized with a true Catalepsy. As Van Swieten lived hard by, he was immediately called in; in his presence she suddenly vomited two live worms, and forthwith proceeded with her cookery, quite unconscious of what had happened. She had no relapse.[1]

Other cases are of much longer duration. The death-like state may last for days. It may be mistaken for real death, and treated as such. In the old pre-Christian times we do not hear of this, though it was a sort of thing that would appeal strongly to the feelings and memory. No Sadducee seems to have suggested Catalepsy to discredit the real resurrections recorded in the Gospels,[1] and the fear of being accidentally buried alive is never alluded to by the classic writers, though so picturesque and so capable of poetical treatment. Any cases of apparent death that did occur were burnt, or buried, or otherwise put out of the way, and were never more heard of. But after the establishment of Christianity, tenderness, sometimes excessive, for the remains of departed friends took the place of the hard heathen selfishness. The dead were kept closer to the congregations of the living, as if to represent in material form the dogma of the communion of saints. This led to the discovery that some persons, indeed some persons of note (amongst others, Duns Scotus the theologian, at Cologne), had got out of their coffins, and died in a vain attempt to open the doors of their vaults.

Others were more fortunate. Those who have visited the Lutheran cathedral at Magdeburg, have probably not failed to notice a quaint monument to the Frau von Asseburg. There is her effigy on it in stone, kneeling with her husband, and, in the style of the period, a goodly line of sons on one side and daughters on the other support the pair. The inscription relates how that this noble lady was, after her marriage, supposed to be dead, and placed in the family vault. Luckily the entrance was left unclosed that night, for she rose up, returned to her home and husband, and bore all this fair family after her strange experience of the tomb.

Such events caused no slight panic at the time, and probably led to the custom, still kept up in many parts of Germany, of fastening a bell-pull to the hand of a corpse when laid in the public mortuary. Some cases of resuscitated cataleptics have even occurred in modern times, according to the statement made by Archbishop Donnet to the French Senate last spring.

Catalepsy may be a premonitory symptom of other diseases. Epilepsy has already been mentioned. De Haen relates, in a clinical lecture, a case he saw of a child of twelve, who began by being cataleptic, and ended by reciting the metrical Protestant version of David's Psalms, saying her catechism with proof texts, and

[1] Van Swieten's Commentary on Boerhaave, § 1040, vol. x. p. 170.

[1] Had he done so, it would have been a telling argument for the Council and the Scribes; but it would have small weight with an experienced physician now, nor would it make the miracle any the less in his eyes. He would know that it is quite as supernatural to detect a cataleptic in a funeral-train accidentally met at the city gate, or in one who had lain three days in a tomb completely rolled up in mummy cloth, as it is to raise the dead.

preaching a sermon on adultery.[1] And in several other stock cases, somnambulism seems to have been a complication. Marx saw a girl who became cataleptic from being frightened at a fire (like my patient at St. Mary's), and afterwards went out of her mind.[2] In Goebel's case of a young soldier, Catalepsy complicated the invasion of melancholia.[3] Sauvages says he saw an old man in the hospital at Alais, in whom Catalepsy alternated with quartan fever.[4]

Catalepsy seems to be sometimes voluntary, or at least capable of being brought on by very little external aid. Of this, St. Augustine gives an instance within his own knowledge:—

"There was a certain presbyter of the name of Restitutus in a parish of the diocese of Calami, who, when he pleased (and he was often asked to do it by those who wished to have ocular demonstration of the strange fact), just by having a noise made like as of somebody crying, used to convey himself out of the influence of sensation, and lie like a corpse. So that not only was he insensible to people pinching and pricking him, but sometimes fire had been brought and he burnt with it, without any sense of pain, except from the wound afterwards. The body seemed to be motionless, not in consequence of any voluntary effort, but from want of sensation, as was made the more probable by the absence of any appearance of respiration, as in a dead body: yet people's voices, if they spoke out very clear, he said afterwards he could hear as if they were a long way off."[5]

Persons liable to this form in various degrees lose, by yielding to it more and more, their power of voluntary control, so that exhibitions of it are easily brought on by others who assume an influence over them. They are told in a positive manner that they cannot raise their limbs, cannot open their eyes, cannot feel, and they really seem to lose temporarily motion, sight, sensation. Mesmer turned this artificial production of disease to profit (his own), and it has been largely experimented upon of late years. But the unfortunate subjects of it have brought to their masters so much "gain by their soothsaying," that deception has largely adulterated the real phenomena, and it is difficult to find a genuine patient. For this reason I thought it preferable to quote a case from a writer of unimpeachable shrewdness and honesty, and far from credulous, though destitute of the light of modern science, than to detail the experience of our own generation.

In the artificial disease and in the natural, somnambulism (*clairvoyance*) is a frequent complication, as appears from several cases cited by Tissot. (Œuvres, tom. xiii.)

It was not a groundless idea to suggest that, as we employ counter-irritants to relieve an internal unmanageable inflammation by one which is under our control and less injurious, so hysteria might be cured by inducing in its place an allied malady more subject to our will. But harm seems to be done by it, and probably only a limited number of the English race have a suitable diathesis.[1]

Both in the natural and artificial disease there is exerted a very different influence over the patient by different individuals. The sight of Dr. Jebb's face seems to have acted like the Gorgon's head in reducing his patient to instantaneous marble. My own experience is quite the reverse. Strange nervous phenomena always seem to be frightened away or subside into commonplace at my presence, and so perhaps my report of them is printed in less bright colors than the subject admits of.

Natural Catalepsy seems to become less frequent, or, at all events, the symptoms less marked and strange, as the world grows older. Some are even getting sceptical about its existence, and doubt the propriety of retaining it on our list of diseases. But even if it should be as extinct as the dodo or the great auk, this insult is uncalled for. The circumstances which surround the human race, especially when sick, are so altered, that it would be wonderful if some of the phenomena exhibited in pathological conditions were not altered too. Read the treatment adopted in many of the cases of Catalepsy quoted by the systematic writers. Take, for instance, that which M. Sauvages communicated to the Académie des Sciences,[2] where intermittent attacks of the disease occurred from time to time during several years. Though the patient was pale with a weak pulse, and though the blood could scarce be got to flow from the veins, yet she was bled, once from the arms, many times from the feet, and seven times from the neck. She had five or six repetitions of purgative medicine, not to mention

[1] De Haen, Ratio Medendi, vol. i. cap. xxxiii.

[2] Marx, De Spasmis, § 61.

[3] De Catalepsi, autore Theophilo Goebel. (A Berlin inaugural thesis, 1818.)

[4] Mémoires de l'Académie Royale des Sciences, &c., 1742, last page.

[5] Augustine, De Civitate Dei, lib. xiv. cap. xxiv.

[1] Those who wish to pursue the subject of mesmerism will find it treated of with the broad views of a non-specialist by Feuchtersleben in his Medical Psychology, translated by the Sydenham Society.

[2] Mémoires de l'Académie Royale des Sciences, Année 1742, p. 409.

bouillons apéritives, stomachic opiates, and twenty tepid baths, before they thought of giving her iron, which wrought a cure sooner than one could have expected. I lighted accidentally on another case communicated to the same scientific body by M. Imbert, in 1713.[1] It is that of the driver of the Rouen diligence, aged 45, who fell into a kind of soporific Catalepsy on hearing of the sudden death of a man he had quarrelled with. It appears that "M. Burette, under whose care he was at La Charité, made use of the most powerful assistances of art—bleeding in the arm, the foot, the neck, emetics, purgatives, blisters, leeches," &c. At last somebody "threw him naked into cold water to surprise him." The effect surprised the doctors as much as the patient; it is related with evident wonder how that "he opened his eyes, looked steadfastly, but did not speak." His wife seems to have been a prudent woman, for a week afterwards she "carried him home, where he is at present: they give him no medicine; he speaks sensibly enough, and mends every day."

Again, the "*dame de Vesoul*," whom M. Tissot justly calls "*la cataleptique par excellence*," so characteristic were the phenomena, was attacked during Lent, when she had been starving herself in order to give alms to the poor, and was also worried by a lawsuit which had brought her to Besançon. Yet she was bled in the foot. Fortunately, after three days, her friends took her home to Vesoul. What happened then the reporter says was quite as wonderful as her illness,[2] namely, that she had no more medical treatment, and yet got well without a relapse. I cannot feel the same wonder, for I feel sure that the "powerful assistances of art"—bleeding, blistering, starving, purging, coddling, sympathizing, and admiring—would have converted any of the cases under my charge into equally magnificent specimens of a long-continued intermittent disease.

But the fact of its being partly produced by art does not make Catalepsy a bit less of a reality, for the same may be said of all preventible diseases.

Besides the effect of treatment, it is likely that the unrestrained manners and want of mental control peculiar to the barbarous ages of all nations, would render mediæval Europe liable to exaggerated exhibitions of all physical defects. And, as physical defects are indubitably hereditary, the national temperament would be thereby affected. As an example of what is alluded to, take one scene from early English history, and conceive it happening in the present day. Fancy four members of the Queen's Privy Council calling after lunch on a refractory archbishop who had voted against the Ministry that had appointed him, with the intention of showing him the error of his ways. Fancy them scolding and blaspheming "by God's wounds," giving him the lie, "jumping up and leaping about," "throwing about their arms," "twisting their gloves," "raving like madmen." Fancy him, red in the face, defying them, rushing after them to the door, calling one his lackey, and another "a pimp." Yet this is only a part of the want of restraint shown by both parties when Reginald Fitzurse, William de Tracy, Hugh de Morville, and Richard Brito called on Archbishop Becket on the afternoon of December 29th, 1170.[1] The mere fact of the murder, with the nauseous details of how Tracy picked out the brains with his sword, is not half so strange as such a scene. How many generations does it take to produce descendants of such men free from nervous disorders?

The deficient vitality of which Catalepsy is a manifestation occupies that puzzling part of the circle of life which lies between spirit and matter. We know so little about the chain which connects the two, that its links are reckoned by us as few and short, and we have no names for them. Yet when we see the varied phenomena produced by breaches in the connection, we are led to feel our ignorance of the subject, and to conjecture that these abysses of incertitude veil a long list of vital functions.

In default of names for even the normal functions of this department of life, we must not expect an accurate nomenclature for their aberrations from health; and the most we can do in attempting to classify them, is to observe how near their origin lies to one or the other extremity of the series of vital acts which are interfered with—what relations their phenomena bear on the one hand to mind, and on the other to body. We shall thus have a natural order with pure insanity at the one end, and epilepsy traceable to organic lesion at the other. In the middle will lie ecstasy, Catalepsy, and hysteria, with many a blank between for the anonymous transitional forms. I do not think we can spare any of these names, and instead of clubbing them together, as some would fain do, under the common head of "hysteria," it would appear more useful to divide that disease, according as its emotional, anæsthetic, hyperæsthetic, or convulsive phenomena are most prominent.

[1] Martin's Memoirs of the Academy of Sciences at Paris, vol. iv. p. 360.

[2] "Ce qui ne surprendra peut-être pas moins que sa maladie." (Tissot, Œuvres, tom. xiii. p. 16.)

[1] Hook's Archbishops of Canterbury, vol. ii. p. 497.

I say it would be useful to make a main point in each individual case whether the malady is most related to deviation from mental or bodily health; for I feel convinced it is only by this observation that we can avoid such disappointment as leads many to look upon hysteria, for example, as an *opprobrium medicinæ* which makes them feel the same sort of anger against it as is roused by moral guiltiness, and disposes them rather to punish than to cure the patient who has thwarted them.

Treatment.—As to the treatment of Catalepsy, it is probable that valerian and ammonia, administered in draught or enema, whichever is most convenient, together with a modification of what cured the Rouenese stage-coachman, namely shower-baths, will accomplish all that is wanted in the way of medicine for the slighter cases likely to come under treatment in the present day. In longer continued cases Dr. Jebb's prescription of an opium plaster to the epigastrium, with tonics to the mucous membranes, is rational practice; for Catalepsy seems to depend much on the mucous membrane of the stomach, as is the case with its sister malady, hysteria. But I would strongly urge upon all who have the charge of these and similar mental, semi-corporeal manifestations, to take the hint given me by the second patient, and try to acquire (surely it is to be acquired by trying) the habit of command. Let them exercise it in the direction of supplying the deficient will, not of paralyzing it, of demesmerizing instead of mesmerizing their patients, and it is astonishing how much pharmacopœial medication will be saved to both parties.

Catalepsy may be sometimes feigned. For its detection the most cruel means appear sometimes to have been adopted by our forefathers, such as burning, pinching, cutting, putting into coffins, and otherwise frightening the supposed impostors. A caution is therefore needed, that the trial of these methods would in England very properly subject the experimenter to legal proceedings, the more so as they are quite useless, and prove nothing. No malingerer could successfully feign the peculiar wax-like yielding resistance of a cataleptic muscle, and ought to be immediately detected by a medical man. If a doubt is felt, some expedient may be tried like that of Dr. Marx. Observing that really cataleptic limbs finally, though slowly, yield to the force of gravity and fall by their own weight, he attached a heavy body to the extended hand of a suspected imposter. She bore it up without moving; the intention of the experiment was explained, and she confessed her fraud.[1]

The points intended to be made concerning Catalepsy are these:—

1. That it is a rare pathological condition of mind and body, allied in its causes to hysteria, but not so apt to become chronic.
2. That it is not dangerous in itself, though it may be the precursor of dangerous disease.
3. That it may be artificially produced, but is not easy to feign.
4. That the treatment, moral and physical, should be conducted on the same principles as the treatment of hysteria.

SOMNAMBULISM AND ALLIED STATES.

By Thomas King Chambers, M.D., F.R.C.P.

Somnambulism is a slumber so morbidly profound that resisting spontaneity is lost, and the obscure images, known as ordinary dreams, are able to exert a motor power. "Sleep-walking," where even the intricate concatenated motions necessary to preserve the body's balance are performed, is the most striking and dangerous exhibition of this state, and therefore has given a name to the disease; but it differs in degree only from sleep-talking, sleep-eating, and a form of nocturnal incontinence of urine and of spermatorrhœa. That it is not a partial waking is shown by the difficulty always found in fully waking a somnambulist, and also by the bewilderment and slow return of consciousness afterwards. This bewilderment, moreover, is often followed by headache and a clamminess of the mouth, just like that of the condition known as "the intoxication of sleep" in those who have slumbered too heavily and too long. Again, decided somnambulists are entirely ignorant of what they have been doing during sleep: whereas dreams which

[1] Marx, De Spasmis. Halæ, 1765, § 19.

occur during a partial waking are always remembered more or less. Again, the automatic acts done during partial waking are very short, have no continuity, and quickly end in a decided condition; whereas the acts of the somnambulist are consecutive one upon another. It seems impossible, therefore, to agree with Drs. Symonds, Hartmann, and others, who have regarded it as an incomplete sleep.

Still less can we agree with the superstitious awe which would represent it as an exalted state, in which the soul is freed from the trammels of the corruptible carcase. It is in truth a lower life, in which "the sceptre of reason is surrendered to a physically-directed fancy." (Feuchtersleben.) Instead of nearing the angels, man thus approaches temporarily the nature of ill-bred horses who refuse to lie down in their stalls, birds who roost standing on one leg, and gorged dogs who—

> "Weary with the chase,
> Lie stretch'd upon the rushy floor,
> And urge, in dreams, the forest race
> From Teviotstone to Eskdale Moor."

This morbid sleep usually arises in the first instance from eating too much. Perhaps the overloaded stomach presses on the solar plexus, and produces a partial paralysis in the coats of the arteries, and so in the cerebral circulation. The explanation is the more probable, because sleeping with the head too low is another predisposing cause, whose action would be on the brain.

Strong mental emotion, excessive exertion of the intellect, violent grief, love, &c., probably act in the same way; namely, by arresting digestion and causing a weight at the stomach.

When, however, the habit is once established, it is persisted in even after the gluttony or emotion has been discontinued. In this it follows the rule of all morbid states of the nervous system, which are peculiarly apt to be retained in spite of the removal of their causes.

It is most common in youth, and at about the age of puberty. Then the sexes are equally subject to it; but later in life it seldomer attacks men than women.

Somnambulism is sometimes hereditary. A young lady, about whom I have been consulted a few times this summer, will often (sometimes two or three days a week) go off in the evening into a peculiar dreamy state. She talks and answers questions, though after an interval, walks about the house, goes to bed, remains quiet at night, and sometimes recovers her ordinary condition on waking: but sometimes her mother will go into her room and find her dressing in a vague, dreamy way. After a while she will stare, stretch herself like a person waking from sleep, and resume her natural lively manner. Her memory is always quite blank as to anything said, seen, or done during this condition. She has never had any hysteric or epileptic fits. Such are the symptoms, and her father asked me what name I should give to the disease. I hesitated at first, and then said that some might call it perhaps catalepsy, but that the more proper name was Somnambulism. That was curious, he observed, for his mother had been afflicted with what was called both catalepsy and Somnambulism, and he had heard it was in the family. I have also recently understood that a younger sister of my patient is falling into the same state, but I have not seen her. This lady had occasionally got out of bed when in her unconscious state, but it happened so seldom that no alarm on that score was expressed by the family.

Somnambulism is inconvenient to other people from the fright it causes, and dangerous to the patient from the awkward positions it puts him in when unprotected by reason. But it is by no means inconsistent with a fair condition of general health. It is, for instance, not unfrequent amongst boys and girls at school who bodily and mentally are quite equal to their companions.

At schools accidental accesses of it are liable to be fostered into a habit by the patients' room-fellows talking to them, and otherwise "drawing them out," when in this state.

Like epilepsy, and indeed all diseases of the nervous system, it is apt to become periodical. Some persons will walk or talk, or wet their beds, &c., once a fortnight, week, or month, and so on with great regularity. That does not arise from an accumulation of secretion or excitability; for at first, and while the original cause is predominant, several attacks occur often close together in succession, and then cease. It is rather an evidence of the weakness becoming constitutional, after the original cause has been removed.

Somnambulism has in some rare cases alternated with catalepsy, of which M. Sauvages has recorded an instance. (*See* Catalepsy.) More generally, it alternates with a normal state.

There are cases recorded where the somnambulistic sopors have been so frequent and so long, that there is as much of a sleeping as there is of a waking condition, and thus has arisen the singular phenomenon called "double consciousness." Trains of thought are carried on from one attack to the next, though in the normal interval the mind is quite unconscious of them. A remarkable instance of this is recorded by Dr. Dewar, in the "Transactions of the Royal Society of Edinburgh," vol. ix. p. 365. A servant maid began by

being subject to attacks of extreme sleepiness: then in these sleeps she began to be talkative. Soon there appeared more method in what she said: she personated an episcopal clergyman, went through the baptismal service for three children, and delivered an extempore prayer. Another time she was a jockey at Epsom, and rode round the kitchen on a stool. On awaking, all these pranks were quite forgotten, but in the succeeding fit she remembered all that occurred. Thus, one night a villain indecently assaulted her when somnambulistic. On the morrow the insult was forgotten, but shortly afterwards she had a fresh attack and told her mother of it. She got well after an emetic and the return of the catamenia, which had been absent.

Dr. Abercrombie adds two more cases related to him by non-medical persons, and for that reason (probably) accompanied by more wonderful phenomena. (On the Intellectual Powers, Pt. III., sect. iv. § 2, II.) In principle these phenomena are quite in analogy with healthy dreams, which scarcely ever take cognizance of recent facts of the waking state. For example, in my own dreams, though I had the misfortune to lose a leg two years ago, I always seem to walk about as in youth. And I certainly remember a room-fellow at school who used to talk in his sleep on a class of subjects he never mentioned by day, and who seemed to recollect when in the same state next night that he had spoken of them before. But that an education should be carried on, and languages acquired, during somnambulism, as some strange stories record, is hard to credit. Possibly some confusion existed in the minds of the observers, and they mistook the waking for the sleeping state.

Somnambulistic phenomena have sometimes accompanied the artificial catalepsy of the mesmeric trance. They are called "*clairvoyance*," not that the patients see particularly clearly, but that the common sensorium is very receptive of those slight suggestions which it would neglect at other times, when its attention is occupied with the external world; and so they appear to careless observers to see with the tips of their fingers, the epigastrium, &c., when their eyes are closed. Singular exhibitions are thus produced. But with practice this rapid obedience to slight suggestion is soon acquired by even stupid people; so that jugglers have no difficulty in obtaining sham cases for shows, by no means easy of detection. This prevents the investigation of the subject by scientific persons.

TREATMENT.—1. The patient must be removed from the company of those who would be disposed to foster into a habit by experiment the recent establishment of the disease. This applies particularly to young persons at school, and those brought under the dominion of mesmerizers.

2. The patient must be prevented from falling into that morbidly deep sleep in which the special phenomena of the case are produced. This can be accomplished by waking them up once or twice in the night, before they begin to walk, talk, or do other unseemly acts. It may be done with great advantage during the second hour of sleep in cases of simple sleep-walking and of bed-wetting.

A young lady under my care who used to rise and make water on the floor without being aware of it, was relieved by this means.

M. Trousseau[1] knew a wealthy and beautiful girl, from whose feet wooers had been driven away by an incontinence of urine occurring nightly. At last the impediment to marriage was overcome by a hero. M. Trousseau cynically calls him "*un individu sans fortune*," implying that the girl's purse was her only attraction; but poetic justice requires a better motive for an act so richly rewarded; for, like the knight of whom the "Wif of Bath" tells, he found her "bothe faire and good," when expecting, like him, the latter only; the disgusting affliction vanished straight away—

> "And thus they live unto hir lives ende
> In parfit joye"—

at least they have the chance of doing so. Doubtless it was the prevention of over-profound sleep which cured her.

Some years ago a foolish young man from the country brought for my opinion an instrument which he had purchased of an advertising quack, designed to cure spermatorrhœa by compression of the urethra and prostate. The disease in his case, being purely imaginary, was of course incurable; but I should not wonder if the plan had been found useful, acting as an obstacle to morbid sleep. However, it would require careful medical superintendence.

A clergyman (who corresponded with me anonymously, and therefore I can say nothing of his general health) took by my advice, unsuccessfully, several remedies for spermatorrhœa, till he suspected that he abused himself during sleep; he tied his hands by a string to the bedpost, was awakened several times a night, and cured.

Another patient, troubled with really involuntary emissions, cured himself by having an alarum which he set so as to wake him occasionally in the night.

Other attacks of spermatorrhœa I have found to take place during the abnormally heavy morning doze which lazy people

[1] Clinique Médicale. Leçon LX.

indulge in after they have really had enough rest—the intoxication of repose. These patients should be told to get up and dress immediately after their first waking. It soon cures them.

3. Care should be taken that the head lie high in the bed, and that the body be not covered with too great a weight of clothes.

The son of an old and intimate friend of mine used to suffer when a child from incontinence of urine. Soon after puberty this inconvenience ceased, and has not returned. But at eighteen he has come to me complaining of seminal emissions, which have occurred on a few occasions more than once in a night. On inquiry, he said that on waking up after a defilement he had often found his head right under the bolster. He was advised to be careful in keeping a good hard pillow well down under the shoulders, and he has not suffered since.

4. Though prevention by means of keeping off too profound sleep is desirable, yet patients should not be wakened when walking, or in any other unnatural posture. They should be led back quietly to bed

"Donec discussis redeant erroribus ad se."

Otherwise the fright is dangerous, especially to hysterical persons.

5. Light meals and digestible food are essential, and special expedients should not be trusted in till the general health has been brought up to the average.

SUNSTROKE.

By W. C. Maclean, M.D.

Definition.—A disease of the nervous system, excited by heat, sometimes following exposure to the direct rays of the sun, particularly when to heat is added the pressure of tight and unsuitable clothing and accoutrements, or both; more frequently occurring when the above conditions combine with exhaustion, induced by great fatigue in hot weather, or from the effects of high temperature, night and day, on men breathing the vitiated air of crowded barracks or ships.

The affection is generally preceded by premonitory symptoms, such as thirst, heat, and dryness of skin, vertigo, congestion of the eyes, frequent desire to micturate, followed by syncope, often instantly fatal (the cardiac variety of Morehead), or by insensibility and stertorous breathing, with or without convulsions (the cerebro-spinal variety of the same author).

In both varieties the mortality is high, and unexampled congestion of the lungs is the most common morbid appearance observed after death.

Synonyms. — Insolation; Sun-fever; Coup de Soleil; Calenture; Heat-apoplexy; Ictus solis; Erythismus tropicus.

The first is the name by which the affection is designated in the official classification of diseases in use in the British army.

History.—Sunstroke has been known and recognized as a dangerous disease from early times. Fatal examples of it are recorded by the sacred writers, and these have been referred to by most modern authors who have written on the subject. It is worthy of note that one of the blessings promised to those who shall be partakers of the better life that is to come, is, "that the sun shall not light upon them, *nor any heat*,"—a promise full of meaning to the inhabitants of the "dry and thirsty land" to whom it was first made.

Men of European birth who become sojourners in Hindostan are hardly more solicitous to protect their heads from the direct rays of the sun than are the various races who are children of the soil. In China, on the other hand, the inhabitants expose their closely shaven heads to the hottest sun with apparent impunity.[1] But when so doing they generally make vigorous use of their fans, as if they attached more importance to having a free current of air about their faces, than to protecting their heads from the sun's rays. Sportsmen in India constantly expose themselves in the hottest weather when in pursuit of game. Those who use

[1] I have recently, however, seen an account of an epidemic of insolation, which attacked one of our trading ports in China, after many days and nights of unusually high temperature. The mortality was high, and by no means confined to the European community.

reasonable precautions, who protect the head and spine by a head-dress adapted for the purpose, wear loose clothing of a suitable material, and abstain from stimulants, rarely suffer from Sunstroke.[1] [The same is true of those who play cricket and base-ball in the United States; where the temperature in July and August is sometimes very nearly as high as in India.—H.]

On the other hand, as will be shown further on, men who are made to undergo fatigue under a hot sun, dressed as British soldiers used to be in such circumstances, in tight-fitting clothes, and encumbered with heavy and badly-adjusted accoutrements, wearing a head-dress which not only gave no protection, but concentrated the sun's rays on their heads, suffered from insolation in great numbers in a most fatal form.

Sunstroke, if we are to judge from the older medical returns of the Indian army, was not a frequent or a fatal disease. Even in the eight years ending 1853-4, as appears from Dr. Hugh Macpherson's instructive analysis of later Bengal medical returns, only thirty-eight cases are recorded. This would be very puzzling if we did not know that a great many cases, which would now be entered without hesitation under the head of *insolatio*, were in those days "returned" under the heads of continued or remittent fevers; while those proving quickly fatal, with insensibility, convulsions, stertorous breathing, and such-like symptoms, were considered to be cases of cerebral apoplexy, and registered accordingly.

For example, in the case of the two wings of H. M. 13th Regiment, referred to by Martin, which marched, after some very ill-judged exposure and drilling in the sun, from Nuddea to Berampore in the midst of the hot weather, the men suffered terribly. As the result of one march, "the day closed with a sick-list of sixty-three, and eighteen deaths," all of which appear to have been registered as cases of apoplexy. It is certain from the description left by the medical officers, that the cases would in the present day be considered to have no pathological relation to apoplexy, yet Dr. Henderson was at a loss "whether to consider them cases of remittent fever or apoplexy." (Martin.) The symptoms were clearly those of insolation. Many other examples of a like kind could be given.

The following are some of the best historical instances of insolation occurring in the field and in barracks; they have been brought forward in more or less detail by nearly all recent authors on the subject, and for the last five years I have used them in my lectures in illustration of the different forms of this affection.

In May, 1834, the 68th Regiment, quartered in Fort St. George, Madras, attended the funeral of a general officer. The regiment paraded in full dress at an early hour in the afternoon in one of the hottest months in the year, their tight-fitting coats buttoned up, their leather stocks, as stiff and unyielding as horse-collars, round their necks, heavy cross-belts so contrived as to interfere with every movement of the chest, heavy shakoes on their heads, made of black felt, mounted with brass ornaments with wide flat circular tops, ingeniously contrived to concentrate the sun's rays on the crown of the head, and without protection in the way of a depending flap for the nape of the neck So dressed, the men marched for several miles. Before the funeral parade was over, the soldiers began to fall senseless—one died on the spot, two more in less than two hours. Men suffering from insolation in various degrees were brought into hospital all that night and part of next day. The cases that did not prove fatal, although their real nature was correctly understood by Dr. Russell, acting surgeon of the regiment, were all registered as cases either of continued or ephemeral fever. The symptoms in the fatal cases were thirst, excessive heat of skin, extreme prostration, immediately followed by gasping respiration, coma, stertor, lividity of the face, and death. After death no morbid appearance was found in the brain, but in the lungs of all there was extreme congestion. There lingers a tradition of this parade in Madras to this day.

The 63d Regiment suffered in the same way, at the same place, and under circumstances precisely alike. (Martin: Influence of Tropical Climates.)

Of the next example the writer of this article was an eye-witness. The 98th Regiment joined the expeditionary force under Lord Gough in China in 1842. The regiment came from England in the *Belleisle*, an old 74-gun ship, and suffered from overcrowding. On the 21st of July, the 98th took part in the attack on Chin-Kiang-Foo, the final military operation of the war. The men were dressed precisely as already described in the case of the 68th Regiment. In this condition they

[1] Staff-surgeon Becker informs me that while on active service in China, a sudden order was given for a movement in the heat of the day. One commanding officer opened the canteen before the men marched; by way of precaution they partook freely of spirits. The effects of this injudicious measure were soon apparent: the men who thus indulged suffered twice as much from Sunstroke as those who did not. On this occasion also it was noted that a large proportion of the victims had heart disease, in one or other of the forms I have elsewhere shown to be so common in the army.

had to take possession of a steep hill exposed to the fierce rays of the sun shining out of an unclouded sky. A great many men were struck down by the heat, about fifteen died on the spot, falling on their faces, as Dr. Parkes, on the authority of another eye-witness, has correctly described (Practical Hygiene); they gave a few convulsive gasps, and died before anything could be done for their relief.

The best history of an outbreak of insolation with which the writer is acquainted is that given by Dr. Barclay, of the 43d Light Infantry, and published in the second number of the *Madras Quarterly Journal of Medical Science*. The 43d Regiment performed one of the most extraordinary marches on record, having marched from Bangalore, in the Deccan, to Calpee, in Central India, a distance by the route taken considerably exceeding eleven hundred miles. The exigencies of the public service at that time (1857–58, memorable as the years of the mutiny in Bengal) were such, that this march, with the exception of a few brief halts at stations by the way, was made continuously, and a great portion of it was accomplished during the hottest season of the year. The men were exposed to a very high temperature by night as well as by day.

Dr. Barclay, while in a valley at the foot of the Bisramgunge Ghât, observed the thermometer at 118° Fahr. in the largest tents during the day, 127° in the smallest, and on one occasion he observed it at 105° at midnight. This prolonged exertion and continuous exposure to excessive heat by night as well as by day, exceeded the limits of human endurance. When they reached Nagode, "the indications of exhaustion in the altered looks of the men, their loss of flesh, and their evidently failing strength, were so obvious that they forced themselves on the observation of every one." But further on, on the march from Humeerpore to Calpee, Dr. Barclay records, "There was scarcely a man in the regiment whose strength was not reduced to a level with that of a child."

It is remarkable that no case of insolation occurred until the 28th of April, *i. e.*, until the 43d had marched 969 miles—until, in fact, the signs of exhaustion, first noted by Dr. Barclay, were apparent. From that date they increased in frequency. When at the foot of the pass, named above, cases "were brought to the hospital tents at every hour of the day and night, and although a large proportion of them recovered, two officers and eleven men were buried under one tree in the neighborhood of the camp." (Dr. Barclay on the Natural History of Insolation.)

Boudin relates a terrible example of the effects of heat on a body of Belgian soldiers on the line of march, which may be fairly taken as a striking instance of the evil consequences of tight clothing and accoutrements under exertion in a high temperature. On the 8th of July, 1853, a body of men, 1200 strong, marched from Beverloo to Hasselt. They started at eight o'clock in the morning. Only 500 reached Hasselt in the evening. Nineteen perished *en route*, and a great number in a state of furious delirium were taken to hospital. I do not think that anything so disastrous as this occurred during the unavoidable exposure of British soldiers to the fierce heat of the sun in Central India, in the years of the Mutiny, 1857–58. It is remarkable that the temperature on this occasion did not exceed 33° or 35° Centigrade. M. Boudin adds, that two well-known Egyptian astronomers, MM. Mahmoud and Ismaël, who were in Brussels on that day, assured M. Quetelet that they suffered as much from a temperature of 30·7° C. in that city as in Cairo under a temperature of nearly 50° C.: "*Nouvelle preuve de la nécessité de tenir compte de la qualité de la température.*"

But, as has been said, insolation occurs in barracks as well as in the field. The two best and most carefully observed examples of this form of the affection are those recorded by Dr. Butler, of the 3d Light Cavalry, at Meean Meer, and by Mr. Longmore, then surgeon of the 19th Regiment, stationed at Barrackpore, in Lower Bengal, and both published in the *Indian Annals of Medicine*.

Dr. Butler records that his men had not been overworked or fatigued, but at a time when the heat was excessive (102° in the shade) they were overcrowded. "Assuredly," says Dr. Butler, "those barracks most crowded, least ventilated, and worst provided with punkahs and other appliances to moderate excessive heat, furnished the greatest number of fatal cases."

Mr. Longmore's evidence on the same point is most important. Out of sixteen cases thirteen occurred in barracks or in hospital, and Mr. Longmore notes that one-third of his cases, and nearly half the deaths, "occurred in one company of the regiment quartered in the barrack which was manifestly the worst conditioned as to ventilation, and, indeed, in every sanitary requirement."

Mr. Longmore remarked also that "the patients seized in hospital were lying in two wards on the leeward side, and from circumstances of situation the warmest and most confined."

Insolation has frequently been observed on board ship, but almost always under conditions similar to those in barracks; that is, where overcrowding and impure air are added to the influence of excessive heat. Insolation is not uncommon on

board the mail steamers in the Red Sea in the hot months of August and September; it has been observed that most of the cases occur while the sufferers are in the horizontal position in ill-ventilated cabins.

M. Bassier, Surgeon in the French Navy, reports (Dissertation sur la Calenture) that in the month of August, 1823, the man-of-war brig *Le Lynx*, cruising off Cadiz, had eighteen cases of insolation, out of a crew of seventy-eight men. The heat was excessive ("33 à 35 *degrès Cent.*") and much aggravated by calms. In this case the ship was overcrowded: "*le bâtiment, très petit, offrait peu d'espace pour le coucher de l'équipage.*

M. Boudin (Statistiques Médicales) quotes from the same author the case of the French man-of-war *Duquesne*. This ship, while at Rio Janeiro, had a hundred cases of insolation, out of a crew of six hundred men. Most of the men were attacked, *not* when exposed to the direct heat of the sun, but at night when in the recumbent position—that is, when breathing not only a hot and suffocating, but also an impure air.

[An important fact is, that heat-stroke is very much more common in cities than in the open country. Tokio has had 100 cases in a single day; New York, 60; Philadelphia, 20. By the statistics of the New York Board of Health it has been shown that, when the thermometer is over 90° Fahr. the number of cases of heat-stroke is not exactly proportional to the maximum or average of daily temperature. The existence of another factor may be here suspected; probably, the condition of the atmosphere in regard to humidity, electricity, or pressure.—H.]

Etiology.—I have already remarked, that men will bear a high temperature in the open air with comparative impunity, provided (*a*) that it is not too long continued, (*b*) that the dress be reasonably adapted to the temperature, (*c*) that the free movement of the chest be not interfered with. As already remarked, British sportsmen in India often pursue their exciting amusement in the hottest weather; but as they are careful to dress suitably, they seldom suffer from insolation.

It will be remarked, that in all the examples of insolation in the direct rays of the sun given above, the sufferers were soldiers dressed and accoutred precisely as men ought not to be in the circumstances in which they were placed. Dress and accoutrements, then, are powerful aids to high temperature.

The case of the 43d Light Infantry, as related by Dr. Barclay, brings out another predisposing cause, which appears to exercise a powerful influence, viz. exhaustion, the result of prolonged exertion.

This appears to act in various ways. First, there is a great waste of tissue, for a time,—that is, so long as the functions of the skin, lungs, bowels, and kidneys continue in tolerable activity, the blood is maintained in a state probably not far from its normal condition. But as exertion continues under a temperature seldom falling below 90° or 92° Fahr., and often reaching, as we have seen, 100°, 107°, and sometimes 118°, in a well-made tent, the function of the skin ceases, and the result of this must be not only the loss of the cooling effect of evaporation, but also blood impurity. Again, all observers note that under such circumstances, obstinate constipation of the bowels is a constant condition, still further promoting this impure condition of the blood. But not only may we reasonably suppose that the blood must be in an abnormal condition from the above causes; it is very imperfectly replenished by healthy, well-digested food. "The appetite," says Dr. Barclay, "gradually failed, and a feeling of nausea was generally complained of, the sight of food often exciting loathing."

In other instances there was nearly complete anorexia. It may be supposed that the activity of the kidneys may, to some extent, compensate for the lost function of the skin and the impaired eliminating action of other organs. But not to dilate on the fact that frequent micturition, although a common, is by no means an invariable symptom in the premonitory stage of insolation, is it not possible that the inability to retain urine in the bladder is quite as much due to its quality as its quantity? "I cannot hold my water," was the almost invariable complaint of Dr. Barclay's men; and Mr. Longmore carefully noted the same thing in his cases.

Dr. Obernier is of opinion that although the secretion of urine is in excess at the beginning of a march, the quantity is lessened by prolonged exertion, doubtless because the blood has lost much of its water through profuse perspiration; and he states that suppression of urine often precedes Sunstroke. "Now, suppression of urine means retention of urea in the blood," and accordingly in two cases of Sunstroke Dr. Obernier found urea in excess of the normal quantity in the blood. Yet the same author declares that in his experiments on animals subjected to the effects of heat until they exhibited signs of Sunstroke, he "could not discover a trace of urea in their blood."

If we look again to the cases quoted as occurring in barracks and ships, it will be seen that another cause besides heat was in operation. In all the examples given of insolation in barracks, the observers have noted the ill-ventilated, overcrowded condition of the places where the majority of the cases occurred. Many of the small

bungalows occupied by officers in military stations in India, are quite as hot as any barrack-room, yet nothing is more rare than to see officers affected with this form of insolation. In the French ships, overcrowding and imperfect ventilation, with their necessary consequence, impure air, were noted by the surgeons who reported the cases.

It is then evident, from the above facts, and from many more of the same kind that might be adduced, that the pressure of tight and unsuitable clothes and accoutrements, excessive fatigue, with all its consequences, and the impure air of ill-ventilated barracks and ships, are powerful predisposing causes of insolation.

[Another obviously predisposing cause, in the cities of the United States, is *intemperance*. A large proportion of the victims of insolation are those who drink freely, whether actually drunkards or not. In 1878, when a considerable number of deaths from Sunstroke occurred, a sort of panic in regard to the agency of alcohol and over-exertion in its promotion was started by the public press. Whether this had a beneficial effect or not, it happened that on the hottest day (16th) of July, 1879, with the thermometer 100° in the shade in some places in Philadelphia, no cases of heat-stroke were there reported. —H.]

But it cannot be doubted that heat, and, speaking generally, heat long continued, is the true exciting cause of this formidable affection. The recently published observations of Dr. Obernier, of Bonn, put this opinion beyond doubt. Fick, a German physiologist of reputation, maintains that under the controlling influence of radiation and evaporation through the lungs and skin, the temperature of the blood in man is always the same.[1] But Obernier's experiments confirm the opinion urged in this article, viz. that, if through any cause the cooling effect of the above processes be interrupted, "*as by warm and tight clothing*, by an elevated temperature of the outer air, by exposure to direct sunshine, or the overheated atmosphere of the engine-room, or even the forced deprivation of cold water: the effect is an accumulation of heat in the body, and an injurious if not fatal action of this heated blood on the nervous system and through it on the heart."

The observations of physiologists have shown that the human body produces four times as much heat under considerable muscular exertion as during sleep. Obernier's exact experiments prove that the temperature of a man walking for half an hour increased by ½° Cent. or 0·9° Fahr. After two hours' walking in sunshine, his temperature rose 3·6° Fahr. If exertion be continued in a hot atmosphere, or with the clothing so often adverted to in this article, or without the use of cold water, until the cooling processes of radiation and evaporation fail, "then the action of the heart grows weaker and weaker, the lesser circuit of the blood becomes overcharged, the venous vessels of the head grow turgid and sensibly expand; these symptoms develop gradually, but the resulting disease—Sunstroke—makes its appearance suddenly." Obernier offers no opinion on the question whether or not nerve tissue undergoes any change of structure under heat. At this point the observations and experiments of Kühne, recorded in the second edition of Ludwig's Physiology, are full of interest.[1] This physiologist found that after exposing frogs to a high temperature, an electric current could with difficulty be transmitted along their nerves; at first it was lessened, and finally stopped altogether.

Other experiments by the same observer demonstrate that if the heat in any vertebrate animal exceeds 113° of Fahr., coagulation of the albuminous principle in the muscular system at once takes place.

[Similar investigations, by H. C. Wood and others, have added nothing of much importance to the above results. It appears certain that when a temperature a few degrees above 100° Fahr. is reached in the human body, the normal conditions of the muscular and nervous tissues and of the blood are all in danger of alteration. This becomes more or less serious according to the extremity of heat attained, and also according to the vital energy of the system at the time. Fatigue, intemperance, perhaps the depressing influence of humidity, and certainly that of the foul atmosphere of ships, barracks, or large cities, by lowering vital resistance, may promote the production, by heat, of disorganization of nerve-corpuscles, sarcous elements, or of the blood. Either of these, if carried to a consider-

[1] My friend Staff-surgeon Becker will shortly publish a series of most interesting observations on his own temperature on a voyage to India. Dr. Becker lived as much as possible on a uniform diet, which he weighed exactly, and ascertained the amount of urine excreted daily. He took his temperature with unfailing regularity eleven times in twenty-four hours. Every tenth day he fasted, and took his temperature every hour. The temperature in the external air was also carefully registered. The chart shows that the temperature of the observer rose or fell one degree with every twenty degrees of rise or fall of that of the external air. The disturbance on the days of fasting was very marked, the temperature invariably falling, and not recovering for many hours after food was resumed.

[1] Ludwig's Physiologie, vol. ii. p. 732.

able extent, may cause death. The capacity of the body to endure with safety exposure to an external temperature much above 100° Fahr. is due to the protective effect of cutaneous transpiration and evaporation. The sun temperature of New York and Philadelphia is often as high as 132°. I found it on one occasion in Maryland, 135°. Livingstone, in African deserts, found it 136°. But the hot-air bath is often taken as high as 150°—250°; and Chabert, the "Fire-king," is reported to have gone, specially protected, into an oven heated to 600° Fahr.—H.]

There is no agreement among observers as to the effects of extreme dryness or moisture in increasing or diminishing the effects of heat. Insolation has been observed in both conditions. In the case of the 43d Regiment, the hot, dry land winds were blowing. Mr. Longmore also notes the extreme dryness of the air at Barrackpore during the outbreak there; and in all the examples given, the disease disappeared with the first heavy fall of rain, attended with a rapid fall in temperature. On the other hand, Dr. Baxter, of the 93d Highlanders, who gives four cases of Sunstroke observed at Sealkote, considers that Sunstroke is much more likely to occur when the atmosphere "is largely impregnated with watery vapor." (Dublin Quart. Journal of Med. Science, No. 81, Feb. 1866.)

Mr. Naylor also is of opinion that cloudy days, with "a moist condition of the atmosphere," favor the occurrence of insolation (Morehead's Clinical Researches). Exact observations on this point, with the wet and dry bulb, are much required.

It would appear that a hot and moist condition of the air is most favorable to the production of insolation in barracks, because not only does such a condition diminish the cooling effect of the evaporation from the skin, but interferes with the artificial means used to reduce the temperature of the overheated rooms.

Duration. — The disease may prove fatal, as we have seen, in a few minutes, or the symptoms may last from one to forty-six or forty-eight hours.

Termination.—The disease terminates either in death or recovery, which may be complete or partial, certain sequels in a considerable number of cases appearing. These are persisting headache, the pain being either fixed or shifting; a chorea-like affection of the muscles, generally those of the forearm and hands; epilepsy, particularly in those who have suffered from this disease in youth, or who have a hereditary tendency to it. In some cases mental weakness, which may prove permanent, follows Sunstroke. In one example, that of an officer of distinction, who lost his hunting-cap while pursuing a wild hog at speed, and in the eagerness of the chase rode for miles bare-headed, Sunstroke was the result; from that hour his mind was affected, and complete recovery never took place.

Symptoms.—Dr. Morehead has divided Insolation into three varieties—the Cardiac, the Cerebro-spinal, and the Mixed. In the present state of knowledge this classification is useful, and it certainly appears to be founded on correct pathology.

In the Cardiac variety, although it is probable that the sufferer is himself conscious of some premonitory symptoms, there is seldom time for their full development so as to attract the attention of bystanders before the patient falls, gasps, and in some severe cases expires before there is time to do much, or anything, for his recovery, death taking place by syncope. This is the form most frequently seen in men exerting themselves in the heat of the sun when dressed and accoutred as were the soldiers of the 98th Regiment at Chin-Kiang-Foo, or at the funeral parades at Madras above described.

In the so-called Cerebro-spinal cases, premonitory symptoms generally give notice of the coming danger. These are heat of skin—this is never absent; the heat is attended with extreme dryness, and is remarkably ardent and stinging, exceeding that of the worst form of remittent fever, which is sometimes as high as 107° Fahr.—giddiness, congestion of the eyes, extreme debility, nausea, and frequent desire to micturate. This last symptom is much insisted on by Longmore, and Dr. Barclay says that "I cannot hold my water" was often the first complaint made by many of his patients. It is a notable thing that headache is by no means a common symptom; it does not appear to have been complained of in a single instance in Dr. Barclay's cases.

Again, a wild shout of laughter, or an attempt to escape in terror from some imaginary enemy, sometimes precedes the more serious symptoms, to be presently described.

M. Bassier, in the case already referred to of the French man-of-war at Rio Janeiro, mentions that the utmost difficulty was experienced in preventing the men from throwing themselves into the sea: "*Ils devenaient incohérents dans leur discours, poussaient des curis, menaçaient de geste et de regard, entraient en fureur, et semblaient mettre tous leurs soins à découvrir une issue qui leur pérmît de s'élancer à la mer.*"[1] On one occasion I saw a man in

[1] Frenchmen under the influence of insolation seem strongly impelled to self-destruction.

Boudin relates that in 1836, "pendant une

this condition suddenly possess himself of the arms of a sentry to defend himself from an imaginary enemy.

It is not by any means always that we have an opportunity of seeing the above premonitory symptoms. Where men in barracks are sensible of the approach of any of them, they generally assume the recumbent position, and in that state pass into a state of coma, the attention of their comrades being first called to their condition by their stertorous breathing.

After a longer or shorter continuance of the above symptoms, the patient becomes insensible; the heat and dryness of the skin augment; the respiration becomes hurried, noisy, labored; the pupils contract, and are quite insensible to light; the conjunctivæ become more congested, "pinky" (Barclay); the heart acts tumultuously; the pulse in men in asthenic condition being at first rapid, but distinct, but as the case progresses unfavorably, becoming compressible, feeble, and irregular; convulsions are frequent, but not invariable: sometimes they appear early, in other cases they immediately precede death.

Dr. Barclay expressly says, that "in a large proportion of cases, from the commencement of the attack to its termination in death, the patient never moved a limb or even an eyelid."

In the Mixed form of Morehead, the symptoms partake of both varieties, and the fatal event is brought about partly by coma, partly by syncope.

Diagnosis.—The diseases with which this affection appears to have been confounded are cerebral apoplexy, and various forms of fever, such as ardent continued fever, and even some of the graver forms of remittent. With the first named it has no pathological relations at all, and it is to be regretted that the term "apoplexy" continues to be appended to any of the names in use to distinguish this disease. In both apoplexy and the cerebro-spinal variety of Sunstroke, there is coma; but the pulse in apoplexy is slow, generally full, sometimes intermitting. In Sunstroke it is quick and sharp. In apoplexy the breathing is slow, irregular, and explosive in expiration; in Sunstroke it is rapid, noisy, but not explosive. In apoplexy the pupils are usually dilated, or one is more so than the other; in Sunstroke both are contracted, and the conjunctivæ are deeply congested. The skin in apoplexy is not hot, and is often cold and moist; in Sunstroke it is always, except in some rare examples of the cardiac variety, very hot and excessively dry.

Paralysis (hemiplegia) is the almost invariable result of cerebral hemorrhage; it never follows immediately on Sunstroke, and is rare even as a sequel of that affection.

From ardent continued fever, the premonitory symptoms already enumerated, more particularly the frequent micturition, and the early supervention of insensibility, will distinguish it. The above, with the history of the case, ought to suffice to distinguish it from the hot stage of remittent fever.

Pathology.—On one point, at least, all modern pathologists are agreed, viz., that the superheating of the blood which precedes and accompanies Sunstroke, has a depressing and not a stimulating effect on the nervous centres; and as the general recognition of this has had a powerful effect in leading to a more rational form of treatment, it is a decided step in advance.

The opinion given by Dr. Morehead, that the sthenic constitution of the newly-arrived European predisposes to the cerebro-spinal variety of insolation, must, I venture to think, be taken with some qualification. Without doubt it was this variety that prevailed most in the 43d Light Infantry; yet, from Dr. Barclay's description of the miserable condition to which his men were reduced before the disease appeared among them, they were in anything but a sthenic condition. On the other hand, the men of the 63d Regiment at Madras, and the 98th at Chin-Kiang-Foo, fresh from Europe, were in the very opposite condition; yet it is clear, from the description given of the symptoms, that they suffered from the cardiac variety. It appears to me that the constriction of the men's chests by tight coats, the pressure of their unyielding stocks and accoutrements, had much to do in determining the particular symptoms—an opinion strengthened by all that I have learned from a careful study of the effects of dress and accoutrements on the organs of circulation and respiration, and by the revelations of the committee appointed to investigate this subject. At Chin-Kiang-Foo, the soldiers of the 18th Royal Irish, the 49th, and 55th Regiments were quite as much exposed to the sun as the 98th; they did not suffer from insolation, but they marched and fought without stocks, and with their jackets open.

I am also strongly impressed with the opinion that blood impurity, induced by the vitiated air of overcrowded barracks, tents, and ships, powerfully aids heat in bringing about that condition of the ner-

expédition du Général Bugeaud dans la province d'Oran, on a pu compter en quelques heures, 11 suicides et 200 hommes atteints de congestion cérébrale, sur un colonne de quelques milliers d'hommes."

vous centres which leads to the development of the terrible symptoms of insolation. Were it not so, the high temperature so often observed in the small houses and tents, particularly of many junior officers, would make this disease more common than it is among them.

[There appears to be practical importance in the distinction between two classes of cases, which may be respectively designated as *heat-apoplexy* and *heat-collapse*. The former is most likely to occur under direct exposure of the head to the rays of the sun; a genuine Sunstroke. It is most often met with, also, in persons of a rather plethoric habit, or inclined to determination of blood to the head. Heat-collapse, on the other hand, is quite as likely to take place in persons who are fatigued, or whose vitality is low, in the shade, at a high temperature; or, as in China and India, even at night. In the first, heat-apoplexy, there is stupor, with fulness of the vessels of the head and neck, and a full, sometimes slow, pulse. The second, heat-collapse, is much more common, and is marked by all the signs of exhaustion, as above described; with a rapid and feeble, often irregular pulse, breathing accelerated or labored, face rather pallid than flushed, and vessels not distended. In this state, the patient may be conscious almost to the moment of death. As above said, however, mixed cases are numerous.—H.]

Morbid Anatomy.—The blood is invariably found in the same condition as after death from lightning, or blows on the epigastrium—that is, fluid.

When death occurs so quickly as it generally does in this affection, there is really no time for much organic lesion; some congestion of the cerebral vessels is a common, but by no means an invariable, appearance.

The most common condition in all varieties, but more particularly in the cardiac, is congestion of the lungs, with distension of the right heart. This congestion is more complete in this than in any other disease.

Mortality.—If Sir Charles Napier is correct in the statement he makes in a letter published in his Life, that out of forty-four cases of Sunstroke which occurred at Nussurpoor on the 15th of June, 1843, forty-three proved fatal, this is the highest mortality on record.

Dr. Barclay only included severe and well-marked cases in his table, and his death-rate was 42·734 per cent.

Dr. Butler's at Meean Meer was 43·3 per cent.

Dr. Morehead gives the following table:—

	Treated.	Deaths.
Mr. Hill's collected cases . .	504	259
Dr. Taylor's, Gazeepore . .	115	16
Mr. Longmore's, Barrackpore	16	7
Mr. Lofthouse, 14th Light Dragoons	80	10
Dr. Simpson, 71st Regiment .	89	24
Mr. Ward, 3d Bombay E. Regiment	25	6
Mr. Ewing, 95th Regiment .	60	17
Sir Hugh Rose and Dr. Stuart, 25th Regiment	200	—
Field Hospital, Hansi . . .	29	10

Prognosis.—The most unfavorable signs are prolonged and complete insensibility, without movement (this is a much more unfavorable symptom than occasional convulsions); intense heat of skin, persisting notwithstanding the free use of the douche; increasing congestion of the eyes; tumultuous action of the heart; failing pulse; lividity of hands and feet.

If convulsions appear after such symptoms have been present for some time, they indicate the near approach of death.

Prophylaxis.—1st, In barracks. The measure now about to be carried out in the construction of barracks in India, viz., having dormitories in which not more than from ten to fifteen men can be accommodated, will do more to diminish the frequency of barrack insolation than any single remedy with which I am acquainted.

Meanwhile, I cannot too strongly insist on the propriety of at once pitching a sufficient number of tents for dormitories, whenever the night temperature rises to 90°, so as to diminish, by at least one-half, the number of men in barracks during the night. The manner of cooling both tents and barracks is so well understood in India, that it is useless to insist upon it here.

In future, troops in India are never likely to be drilled in a hot sun, or paraded to attend military funerals in heavy marching order at 3 P.M. Even in that "military hothouse" Madras, where such customs "lingered long and latest died," it is to be hoped they will never more be heard of.

As a rule, save under the presence of real military necessity, European troops are not likely to be moved during the hot weather; and where such a cruel necessity exists, the terrible lessons taught during the Mutiny are not likely to be forgotten.[1] When troops of necessity must

[1] Dr. Obernier lays it down as a rule that when the thermometer in the shade marks from 86° to 88° Fahr. all marching of troops, if for practice only, ought to be avoided.

According to the same authority, a man

march in hot weather, they should do so in as "open order" as military reasons will allow, and frequent halts should be called to allow the overheated bodies of the men to cool. This point is much insisted on by Marshal Ney, doubtless from observation of its necessity.

The necessity for light clothing, suitable protection for the head, neck, and spine, is now well understood and provided for by existing regulations.

At such times it will be good economy to engage an extra number of water-carriers, so that a never-failing supply shall be at hand, not only for drinking, but for *douching* purposes. I cordially assent to the practical suggestion urged by Dr. Barclay, that the hospital tents used on such occasions should be the best that can be made, and should be furnished with the best appliances, to maintain as low a temperature as possible. If a patient suffering from insolation can quickly be brought into an hospital tent 15 or 20 degrees cooler than the one from whence he has been taken, his chance of life will be immediately increased. Nor is it necessary to say much on the good effects of temperance.

It is to be hoped that the day is not distant when the spirit ration will be a thing of the past.

TREATMENT.—In the days when insolation was commonly mistaken for cerebral apoplexy, the lancet was usually the first resource. The mortality even now, under a mode of treatment more in accordance with sound pathology, is often exceedingly high ; but when blood-letting was the rule, recovery was the rare exception.

A few years ago, in an outburst of insolation on board one of the mail steamers in the Red Sea, this was the mode of treatment pursued. A fatal issue resulted in every case.

During active service in the presence of the enemy, an officer of rank had Sunstroke. The assistant-surgeon in medical charge of the battery where this happened had the sufferer instantly removed to the nearest shade, stripped him, used the douche freely, and had the satisfaction to see his patient revive and consciousness return. An *official* superior, "*an older, not a better*" physician, unhappily coming up at this critical moment, insisted on opening a vein ; a few ounces of blood trickled away and so did the life of the officer. Mortal syncope immediately followed the operation.

It is needless to insist on this point, for, as Dr. Morehead has observed, "there is now great unanimity of opinion" on the treatment of Sunstroke, and by universal consent the lancet has no place in it.

At the earliest possible moment let the sufferer be carried to the nearest shade, stripped, and assiduously douched with cold water over head, neck, and chest. If this be effectually and quickly done, the powerful impression on the cutaneous nerves will soon re-establish respiration, at first by gasps and catches, soon in a more regular and tranquil manner. It will also reduce the heat of skin. It may require to be done again and again ; in hospital it may be necessary to envelop the patient in a wet sheet, and to apply the fan or pankah over him vigorously until the skin is reduced to a more natural temperature—a measure, however, requiring to be done under medical supervision.

The patient should be encouraged to drink freely, and if vomiting follows, this will often aid in relieving the congestion of the lungs. The douche used as above described is a powerful remedy, and, as Dr. Abercrombie long since pointed out, it may be abused, particularly if it is applied too long to the shaven head. Morehead has also well cautioned us against its prolonged use in a routine way, when the skin is cold and clammy, and the respiration sighing—under such circumstances we must restrict ourselves to dashing water over the face and chest. When the heat of the skin is excessive we may avail ourselves, if ice is at hand, of Dr. Parkes's suggestion, and give an enema of ice-cold water. We should also apply ammonia, with the usual caution, now and then to the nostrils. The bowels being always constipated, the sooner they are relieved the better, by the use of purgatives and enemata. If the skin refuse to act, even after the free use of the douche, and maintains its high temperature. a trial may be given to *Warburg's Tincture*, the most powerful sudorific with which I am acquainted.[1] The occurrence of moderate diarrhœa seems to favor recovery. Support and a judicious use of stimulants must not be neglected.

If sensibility be not restored and maintained by the douche, a blister should be applied at once to the nape, and, if need be, to the shaven head. There is much unanimity as to the good effects of this measure.

The late Sir James Simpson, of Edinburgh, long ago taught us how invaluable the inhalation of chloroform is in the convulsions of children depending on cerebral irritation. In India I have saved the lives of many by acting on this advice. Dr. Barclay in like manner found chloro-

engaged for an hour in foot-racing, the temperature of the air being 61° Fahr., experiences a rise in temperature of 5° even when perspiring profusely.

[1] *Vide* article Remittent Fever, vol. i. p. 365.

form inhalation useful "in the convulsive form of the disease, attended with extreme nervous irritability,"—a class of cases in which, he adds, "the douche is inadmissible from the agony it occasions." In some instances life was saved by this remedy; in all it was prolonged.

[Dr. A. R. Hall, in India, reported beneficial results from the hypodermic injection of sulphate of quinia. In 1868, Dr. H. Norris, at the Pennsylvania Hospital, treated successfully four cases of heat-stroke by the hypodermic injection (a quarter of a grain at a time) of sulphate of morphia.—H.]

Treatment of the Sequelæ of Sunstroke.—Although by careful and judicious treatment many recover from the immediate effects of Sunstroke, considerable numbers are incapacitated by it for service in India, or in any hot climate, without at least a more or less prolonged stay in a cold climate. This is precisely what we see after concussion. Out of the large number of cases of concussion I have seen in India from falls from horseback, I have hardly seen one make a complete recovery without a visit to Europe.

Persistent headache is one of the most common sequels to Sunstroke.

At Netley during the invaliding season we are never without such cases, and very obstinate and intractable some of them are. When the pain is fixed, counter-irritation to the nape is recommended. I have seldom seen it of much use. I have seen this troublesome symptom follow Sunstroke in this country, and be quite as intractable and obstinate as after insolation in India. A lady bathing at Cowes had a slight "stroke" of the sun; she did not lose consciousness. For more than a year after the occurrence this lady (who was under my observation) suffered from headache as severely as any of our invalids from India sent home after Sunstroke in its gravest forms. Of late I have been more successful with the bromide of potassium than with any other remedy. It is far, however, from being a specific, often it fails entirely, as it did very notably in the case just recorded.

Great attention to the functions of the skin forms an essential part of the treatment in all the varieties of the sequelæ of Sunstroke, for it is impaired in all. Frictions, bathing, exercise in the open air, are essential. When the headache is not fixed, but shifting, it will often be found to depend on a weak condition of the digestive organs, and careful treatment suited to the peculiar features of each individual case is required.

In epilepsy following Sunstroke the prognosis is generally favorable. The fits usually subside on the patient being removed to a temperate climate.

ALCOHOLISM.

By Francis Edmund Anstie, M.D., F.R.C.P.

Definition.—A disease of the general nervous system, induced by continued excesses in the use of alcoholic liquors. It manifests itself usually in a chronic, but occasionally in an acute form. Its characteristic phenomena are muscular tremor and progressive muscular weakness, insomnia, hallucinations of sight and (less commonly) of hearing and smell, busy delirium, diminished or deranged intellectual and moral force, together with dyspepsia, slight jaundice and morning vomiting. In advanced cases there are also paralysis of sensation or motion, or both, convulsions, epilepsy, dementia, and general degeneration of the tissues of the body. Tendency to death slight when the original cause of the malady can be removed. The fatal result is either produced by exhaustion from protracted acute delirium, or slowly brought about by progressive degeneration of the nervous centres, or of some important organ of nutrition.

Synonyms.—Alcoholismus chronicus, Delirium tremens, Delirium potatorum, Mania potatorum, Ebrietas, Chronic alcoholic intoxication, Trunksucht, &c. &c. These various names obviously apply to various aspects of the disease accordingly as it occurs in the acute or the chronic forms, between which there was formerly no sufficient distinction made.

History.—The "history" of Alcoholism, to use the expression in its proper medical sense, is simply the story of the excesses of each individual patient as re-

gards the daily allowance of alcohol, the duration of intemperate habits, and the kind of liquor taken, especially as regards its degree of concentration.

ETIOLOGY.—This part of the subject is extremely complex. The simplest portion of it is that which refers to the exciting causes, and it will be best to dispose of this first.

1. The *exciting causes* of Alcoholism can be better appreciated at the present time than formerly, because our increased knowledge of the physiological action of alcohol has enabled us to explode some errors of theory which were almost universal in medical writings and popular belief about the disease. The prime source of these errors was the general tendency to notice only the more acute nervous affections which are caused by alcoholic excess—namely, delirium tremens, maniacal excitement, and terrifying hallucinations (horrors). These phenomena had been observed to occur frequently, and were believed to occur always in consequence of a temporary abstinence from drink after a course of excessive indulgence. This presumed sequence of cause and effect fitted exactly with the classical denomination of alcohol as a "stimulant," a member of an ideal class of medicaments which possessed the peculiar property of exciting vital function in such a manner that after a longer or shorter period a "recoil" was inevitable, under which the forces of life were reduced below their natural level. The great feebleness which was observed to characterize the acute delirium of drunkards was supposed to be due to this kind of exhaustion from the withdrawal of an accustomed stimulus.

At present our ideas are very different. In the first place it has been abundantly shown by various writers, of whom Ware[1] was the earliest, that abstinence from drink by no means always, or even most frequently, precedes the outbreak of delirium tremens or of alcoholic mania: on the contrary, these accidents commonly overtake the patient in the midst of his excesses. Secondly, alcohol, in doses which singly are capable of producing drunkenness, and frequently repeated may bring on acute delirium, &c., has been proved to be a true narcotic poison, of the same class as the so-called anæsthetics, chloroform, and sulphuric ether. Given in these large doses, its influence is entirely in the direction of paralysis—suspension of nervous activity; and this suspension of nervous activity (increased by other sources of deficient vital power, which we shall have to notice as incidental to the circumstances of the chronic drunkard) is itself a sufficient explanation of the nervous debility which brings about the delirious crisis. And thirdly, the modern researches which have enabled us clearly to identify a chronic alcoholic intoxication, often reaching over a period of many months or years, have revealed the fact that in the multitudes of instances the acute attack merely exhibits in full development symptoms which had been partially recognizable for a long time previously.

It may now be taken for certain that the phenomena of which we have to treat under the denomination of Alcoholism, are due in the first place to the direct action upon the nervous system of a blood-supply charged with a high percentage of alcohol. If we surround a living nerve (partially dissected from its connections) with alcohol of a certain strength, we find that it becomes paralyzed—*i. e.*, incapable of transmitting impressions—through its affected part; while a very weak mixture of alcohol and water is incapable of producing this effect. Similarly, if an animal absorb into its circulation a certain quantity of alcohol within a given time, the nervous centres and the peripheral nerves become (though in less degree) paralyzed. That this effect is, at least in part, due to direct action of strong alcohol upon the nervous tissue can hardly be doubted, considering the analogy of the well-ascertained local effect in the above experiment: there is, however, a co-operative cause of no small importance; namely, it has been ascertained by the researches of various observers that the impregnation of the blood with large quantities of alcohol interferes with its absorption of oxygen: it thus becomes unfitted to support healthy nervous functions. Under these combined influences the nervous tissues, and particularly those of the central organs, become more and more unfitted for the performance of their proper functions: and this change progresses with a rapidity proportionate to the strength and frequency of the alcoholic influence. It is counteracted only by one circumstance—the elimination of portions of the alcohol from the system, which goes on by the medium of all the excreting glands, but more especially by the kidneys, the skin, and the lungs. Upon the activity of these organs in performing this task probably depends, *cæteris paribus*, the impunity of the drinker from the ill effects of the poison upon his nervous centres. Thus it comes to pass that the occupation and many surrounding circumstances of the drinker modify his symptoms in an important manner, as will be noticed more particularly under the head of Predisposing Causes.

It is necessary here to recall the prin-

[1] Remarks on the History and Treatment of Delirium Tremens. Boston, 1831.

cipal facts which are known with respect to the action of alcohol upon the organism. This substance is easily absorbed from the stomach, especially when that viscus is empty. If the dose be moderate and the administration well-timed, the effect upon the nervous system is simply that of a restorative stimulant. Sensations of fatigue are dispelled, the mind works more freely, a healthy sense of warmth is diffused through the body, and the arterial system acquires an increased tonicity if it was hitherto deficient in that quality. The latter fact, which is due to the influence of the remedy upon the sympathetic nerves, is capable of being demonstrated in a very interesting and convincing manner. The sphygmograph of M. Marey has the power of accurately representing, by its registration of the pulse-wave, the degree of arterial tonicity present; and by this unfailing test it appears that the small vessels, when relaxed in a condition of fatigue, are brought, by a moderate dose of alcohol, to a proper tension, from which they suffer no recoil. If, on the contrary, the dose has been immoderate, or administered at a time when it was not required, the pulse-waves give a precisely opposite indication — that, namely, which proves that arterial relaxation has occurred; and simultaneously with this the pulse becomes abnormally quick. At the same time other symptoms of a paralytic nature are observed, confined in the first instance to the spinal nerves and to the fifth cranial nerve. The former show their weakness by the occurrence of slight feelings of numbness, and an impairment of muscular sense in the extremities; the latter indicates its affection by the occurrence of slight numbness of the lips. The vaso-motor fibres of the fifth nerve discover their partially palsied condition by flushing of the face, congestion of the conjunctivæ, and lachrymation. The cerebral hemispheres next give notice of the alcoholic influence by the occurrence of intellectual confusion, and the hypoglossi becoming simultaneously affected, the muscular movements of the tongue become difficult, and articulation is impeded. The further stages of drunkenness consist in more or less noisy or sentimental delirium, passing gradually into coma; palsy, more and more complete, of voluntary motion and sensation; the medulla oblongata is palsied, and breathing ceases; and, last of all, the organic nerves of the heart become incapable of performing their functions, and cardiac life ceases. During all this process the secreting glands are affected, but in varying degrees: the kidneys in particular are singularly little acted on in some cases, and very strikingly in others; and the diuresis, which is the result of the latter condition, is the best safeguard against fatal results, as it involves a large elimination of alcohol. MM. Lallemand, Duroy, and Perrin were the first observers who clearly proved the elimination of unchanged alcohol, and the phenomena appeared to them so striking that they concluded, too hastily, that the whole amount of any dose of alcohol taken into the system was thus eliminated by one or other secreting surface. The facts adduced by these authors do not, however, justify any such inference. I have repeated their observations with much care and especial attention to the dose administered — a point singularly neglected by MM. Lallemand, Duroy, and Perrin. The result of these observations is, that a moderate dose (*e. g.* a pint of light beer or a glass of sherry) produces very slight effects in the way of elimination, which last over a few hours only, and cannot be reasonably supposed to represent the elimination of more than a small fractional part of the alcohol imbibed. On the contrary, when a dose has been taken sufficient to produce more or less profound intoxication, alcohol is so copiously eliminated by skin, lungs, and kidneys, especially the latter, that there is some reason to think that as much as from a fourth to a third of the dose taken leaves the body in an unchanged condition within the course of forty-eight hours. Even in this instance, however, there is no sufficient reason to suppose that all the alcohol leaves the body in an unchanged form; indeed there are the strongest reasons for thinking the reverse. It is true that the intermediate compounds, between alcohol on the one side, and carbonic acid and water on the other, which would represent the stages of transformation of the former into the latter, have not yet been satisfactorily proved to exist in the organism after a dose of alcohol has been taken; but in truth nothing like an efficient search has yet been made for them. The researches of Lallemand show us that elimination may go on for a period of forty-eight hours, and my own experience appears to indicate with certainty that not a trace of elimination of unchanged alcohol can be detected at a later date than this. But this is entirely contrary to what we know of the behavior of those poisonous substances which are wholly eliminated in an unchanged form, especially when, like alcohol, they are of a low diffusive power. According to the analogy drawn from such cases, alcohol, were it entirely eliminated in an unchanged form, ought to be traceable by the delicate chromic acid test in all the secretions for a period not of two *days*, but of two *weeks* or more, from the time of its ingestion. Probabilities are therefore greatly against the total elimination of alcohol in an unchanged form, even from the chemical point of

view; and to conclude this part of my subject, I may say that probabilities are converted into what most persons will be inclined to consider certainties, when the remarkable physiological influence of alcohol in supporting vital power is taken into consideration. But as I have dwelt fully on these matters elsewhere, it is unnecessary to repeat facts which would take up a great deal of space in the telling.[1]

The exciting causes of Alcoholism may be understood, then, to be the repeated direct action of blood *strongly* impregnated with alcohol on the tissue of the nervous centres and branches, rendering them physically incapable of the due performance of their functions, and the influence of an insufficiently oxygenated blood-supply consequent on a morbid condition of the blood-corpuscles.

2. The *predisposing causes* of the disease are much more complicated. They should be divided, in the first place, into (*a*) those which are occasional, and (*b*) those which are constant.

(*a*) The occasional predisposing causes include those external circumstances which expose persons to the temptation of drink, and those internal sensations, produced by temporary illness, which bias them in a similar direction, or, by weakening the nervous system, render the effects of drink more sensibly felt.

Occupation is an important influence. For instance, a large number of cases present themselves at the hospital which are directly traceable to the frequent presence of the temptation, as in the case of workmen at breweries and distilleries, and potmen and waiters at taverns. In a higher grade of life, public-house keepers and the clerks and travellers for wine and spirit merchants, are especially liable to Alcoholism. There is, however, by no means such a preponderance of cases due to this direct temptation as might be supposed. A very large number of patients come from the classes whose business exposes them greatly to the *inclemency of the weather;* thus cab-drivers, coal-porters (especially the workmen who lade the river barges), and hawkers are very commonly large drinkers; they very frequently become the subjects of Alcoholism, and would be still oftener affected in this way were it not for the assistance to elimination which their outdoor life renders. *Monotony* of occupation is also highly predisposing, especially when combined with much confinement in close rooms. Amongst occupations of this kind there are none which have furnished me with so many and such serious cases as the trades of shoemaker and barber. The want of active outdoor exercise, of course, represses elimination, and much increases the evil. I have seen few more desperate cases of Alcoholism than some which have occurred in barbers who have been habitually confined to miserably small shops, and at the same time have earned enough money to pay for a great deal of drink.

Depressing Mental Influences are powerful predisposers to drinking habits; and besides this, they directly increase the liability of the nervous system to be affected with symptoms of Alcoholism, in virtue of their weakening operation upon it. There is a vulgar notion that drink is the simple and uncomplicated cause of the greater number of *crimes* committed by the poor. The truth is, that, in recognizing the indisputable fact that drunkenness is often followed by crime of a worse kind, people are apt to overlook large portions of the history of the criminal, and especially the wretched poverty in which he is usually reared. The demoralizing influence of this poverty is the central fact on which we ought to concentrate our attention; it is a common cause of general reckless behavior, of which drunken habits are only a part, although they doubtless render the commission of fresh crimes more probable. The same recklessness of despair has often been seen

[1] The reader is referred for ampler information to the papers of M. Baudot, in the *Union Médicale* for 1863, and to my work on "Stimulants and Narcotics." (London, 1864.)

Since this article was written and in type I have had time to make new researches, which enable me greatly to strengthen my statement as to the comparatively trifling extent to which alcohol is eliminated from the body in an unchanged form. With the assistance of my friend and colleague, Dr. Dupré, I have made experiments on a large scale, and with increased care, which prove indisputably that when alcohol is taken in non-intoxicating doses, the total elimination, in the twenty-four hours, only amounts to a fraction (generally a small one) of a grain for the kidneys, and even smaller quantities for the lungs and skin. Even in cases of intoxication I now believe that a very much smaller proportion of unchanged alcohol is excreted than I had supposed, and enormously less than MM. Lallemand, Duroy, and Perrin had represented. The total period during which any elimination goes on is also proved to be much shorter than had been previously supposed.

By a curious coincidence, it happened that a German observer, Dr. Schulinus, was investigating the same question simultaneously with myself. His results, obtained from a very elaborate and careful series of experiments, closely correspond with my own. Dr. Schulinus' paper will be found in the *Arch. d. Heilkunde* for 1866. My own observations, which were made in ignorance of the German researches, are included in my lectures at the Royal College of Physicians, which appear in the *Lancet* of July, August, and September, 1867.—F. E. A.

to produce intemperance in drink, where poverty had no share in its origin. In the higher classes we not unfrequently see men who have failed in some cherished speculation, or women who have lost the only object which they cared about in life, take to drink with an almost insane vehemence, although they may never have shown any such tendencies before. It is not that there is any particular temptation in the taste of the drinks to which they have recourse, for it is a fact that even the most refined and delicate women, when they take to these practices, altogether neglect the really fine-flavored alcoholic compounds: they do not drink wine, for instance, but brandy, or some equally coarse and strong spirit. In truth, it is an accident which leads them to select alcohol: under other circumstances they would take opium or hashish, or any other intoxicant which came conveniently to hand, or they would plunge into the indulgence of some special vice which promised them excitement. They merely wish for *oblivion*. And this is the very motive which drives the poor in many cases to drunkenness, and which simultaneously inclines them to commit other rash and criminal acts; the wish to escape, in any direction possible, from the hideous dulness of a life which is one monotonous pain. *Starvation*—actual severe deprivation of food—cannot be an active predisposing cause of drunkenness; for the opportunity of getting liquor is cut off by the extreme degree of poverty which brings about such a state of things: it is rather the sense of embarrassment and misery, consequent on the difficulty or impossibility of paying debts, that is common in the lowest ranks of the middle class, which prompts to drinking habits.

The influence of various forms of disease in predisposing patients to Alcoholism is twofold. In the first place, there are many conditions of chronic weakness and suffering which are susceptible of great relief, when they are at their worst period of aggravation, by the use of alcohol; and this fact, accidentally learned by the sufferer, is from ignorance often perverted. The diseases of this class which are the most important are the whole group of neuralgiæ, the depression and faintness attending the menstrual period of some women, and the debility and low spirits which often distress nursing mothers. Under the influence of such disorders patients are extremely apt to use alcoholic drinks recklessly, and the foundation of drunken habits is thus laid. This subject is so important that I may be excused for dwelling on it, at the risk of a digression, because it is scarcely possible in a very few words to convey my meaning without danger of being misunderstood. It is frequently charged upon those physicians who recommend alcoholic stimulants in disease, that they are encouraging patients to indulge in one of the strongest temptations to drunkenness. This accusation is entirely unjust, if applied to those who administer the remedy on scientific principles. I have endeavored to show elsewhere, that it is the use of doses which are large enough or ill-timed enough to produce symptoms of *narcotism* that can alone implant in the patient that craving for drink which forces him against his will to indulge, with constantly increasing intemperance, in the abuse of alcoholic liquors. But the ignorance of the layman who attempts to regulate his own medicinal use of alcohol frequently leads him to confound two radically distinct modes of operation, by either of which this substance may be made to relieve pain and nervous depression or restlessness. The use of such moderate quantities as fall short of producing any, even the earliest, of the *intoxicative* symptoms which have been already described, while it frequently relieves the patient's distress, leaves no disastrous after depression or craving, but simply restores the nervous system to its healthy state. But pain, and various other forms of *malaise*, may also be relieved by the use of true narcotic or intoxicant doses which paralyze the nervous system for a time; and it is this kind of temporary relief which involves depression, and a sense of craving for stimulants during the period in which the drinker is recovering from his narcotic stupefaction. It is this wasteful misuse of alcohol, in the absence of scientific knowledge, which becomes a predisposing cause of drunkenness in the case of patients affected with the class of maladies to which I have now referred.

One special variety of chronic temptation to drink, depending on temporary bodily conditions, deserves more notice than it has yet received, but can be only briefly indicated here. I refer to the influence of the sexual orgasm which distresses particular individuals of both sexes (but especially females) in whom the development of puberty takes place in a difficult or irregular manner. The symptoms with which young females are affected under these circumstances are commonly treated (as "hysterical") by various household remedies, which contain more or less alcohol, such as sal-volatile, eau-de-Cologne, and various warming tinctures. These remedies are often swallowed in most improper and unnecessary quantities; and there is too much reason to believe that in this way the foundation of secret drinking is not unfrequently laid.

(*b*) There is another kind of predisposing cause, which is *constant* in its operation, and which is probably at least as influen-

tial, both in producing alcoholic excess and in aggravating its ill effects, as any of these occasional causes which have been enumerated—viz. a peculiar inherited constitution of the nervous system. In the course of a large experience of Alcoholism among the hospital out-patients, I have been greatly struck with the number of drinkers who have informed me that their relations, either on the paternal or the maternal side, have also been given to drink. And a still larger number are found on inquiry to come of families in which some nervous disorders (especially insanity, epilepsy, and neuralgia) have been markedly prevalent. The doctrine of the hereditary transmission of a neurosis,—which, according to the special pressure of external circumstances, may take the form either of intellectual insanity, of emotional impulsiveness combined with moral weakness, or, on the other hand, of convulsive or neuralgic affections,—has been much insisted upon by recent alienist writers, and especially by Moreau in his very able treatise on "Psychologie Morbide." My own experience has led me to a firm conviction that particular causes of nervous degeneration affecting individuals do very frequently lead to the transmission, to the offspring of those persons, of an enfeebled nervous organization which renders them peculiarly liable to the severer neuroses, and which also makes them facile victims of the temptations to seek oblivion for their mental and bodily pains in narcotic indulgence. I believe that things often work in a vicious circle to this end; and that the nervous enfeeblement produced in an ancestor by great excesses in drink is reproduced in his various descendants with the effect of producing insanity in one, epilepsy in another, neuralgia in a third, alcoholic excesses in a fourth, and so on. Among the higher classes, where it is easier than in the case of the poor to obtain tolerably complete family histories extending over two or three generations, careful inquiry elicits facts of this kind with surprising frequency. So strong is the impression left on my mind by what I have observed in this direction, that I am inclined to believe that the great majority of the most inveterate and hopeless cases of alcoholic excess, among the higher classes, are produced by two factors, of which the least important is the circumstance of external momentary temptation in which the patient has been placed, while the more momentous and weighty cause is derived from an inherited nervous weakness which renders all kinds of bodily and mental trouble specially hard to be borne. It need hardly be remarked that, in this view of the case, the fatal rapidity with which habits of intemperance exaggerate themselves is only what might be expected, seeing that the nutrition of the nervous centres would be still further impaired by each successive indulgence in poisonous doses of alcohol, and the power of moral resistance to feelings of depression and misery would be proportionately weakened.

Symptoms.—The symptoms of acute Alcoholism are in general well known, and there is little difficulty is understanding their access and the order of their succession. What is far less generally understood is the slighter and more chronic form of Alcoholism which, in the majority of cases, precedes by a considerable time the occurrence of the delirious affection. Accordingly, it will be well to commence with the description of this chronic disease, since its characters may be properly considered as representing the earlier stages of a great constitutional malady.

(A) *Symptoms of Chronic Alcoholism.*—It is upon the motor nervous system that the influence of chronic excess is first discernible in the largest number of cases. Of an extremely large number of patients who present themselves at the out-patient department of Westminster Hospital suffering from this disease, certainly more than two-thirds, upon careful analysis of their complaints, state that a muscular inquietude, which might or might not amount to actual tremor, was the first disagreeable symptom which they noticed. In cases of gradual access the affection at first may amount to no more than an inability to keep the limbs of the body still without a special effort of attention—the exercise of the will being sufficient to render the muscles perfectly steady. The degree of motor disturbance is distinct from and independent of the peculiar mental restlessness to be presently noticed as arising somewhat later, although the occurrence of the mental affection very much aggravates the tendency to involuntary movement. This distinction is noticeable in relation to the nocturnal state of the patients. Long before the occurrence of terrifying dreams, of nocturnal delirium, or of hallucination—even before there is *conscious* nocturnal disturbance of the mind at all—the patient feels an inability to sleep which appears to depend on the condition of the motor nervous system. Repeatedly I have been assured by persons suffering from the slighter degrees of Alcoholism that they go to bed with a sense of at least average drowsiness, but an invincible disposition to turn restlessly from side to side in the bed entirely prevents them from getting any sleep.

It is not very often that a patient asks for advice at the early stage of the disease, which is represented by the pres-

ence of the above symptoms only. More commonly he does not come under medical notice till the motor disorder has reached a further stage; and his complaint is now, probably, that he suffers from persistent muscular tremor. This symptom develops itself first in the extremities. Magnus Huss declares that it always appears earliest in the hands; but it is probable that this is a mistake, for in a majority of the cases which have come under my care the lower extremities were first affected; while it is less easy to detect tremor of the lower than of the upper extremities, and the former often escapes notice for some time after its commencement. Huss notes correctly the fact that even in the stage of persistent alcoholic tremor the patients can at first, by a strong effort of the will, restrain their movements for a time, but on the cessation of the effort the tremulousness is ordinarily worse than ever. A very old and general observation is to the effect that the tremor of Alcoholism is almost invariably worse *in the morning*, and it has been usual to assign as the reason for this, that the accustomed stimulus of alcohol has been withheld for some hours. The statement is plausible, because it is the fact that a glass of beer, or wine, or brandy, taken under these circumstances, will at once diminish the unsteadiness of the muscles: but another fact may be mentioned which strongly opposes the theory—viz. that common foods, such as bread and milk, or broth (if the stomach be not too much irritated to digest them), will answer precisely the same purpose. In truth, the excessive morning tremor of the chronic toper is due chiefly to exhaustion from failure to get sleep. What sleep he has had has been of an unrefreshing kind, and a complete condition of nervous prostration naturally results, from which he can only be rallied by food or drink.

Coincidently with the establishment of persistent muscular tremor, and sometimes earlier than this, certain cerebral symptoms present themselves. One of the commonest of these is a buzzing or a rushing sound in the ears, which is frequently, though not always, accompanied with dull diffused headache. Vision is also affected, with varying degrees of severity, the most trifling symptoms being the appearance of *muscæ volitantes*, or of "clouds" before the eyes. Flashes of light are a more serious phenomenon, and their occurrence at night, just before the patient drops into his first uneasy half-slumber, is frequently the immediate precursor of the more definite visual hallucinations. Momentary attacks of vertigo are common. By this time the peculiar alcoholic insomnia is fully developed in the great majority of cases: the patient tosses from side to side during nearly the whole night, getting only broken snatches of sleep, and these almost always attended with disturbing, and often with frightful, dreams.

The mental condition is now usually such as to distress the patient and to impress the medical observer who sees the case for the first time. Its chief feature is the uncertainty of purpose which the sufferer displays: independently of any fixed delusion, or even of a distinct feeling of terror, there is a mental inquietude which makes it impossible for him to settle to any ordinary occupation, or to complete the tasks which he begins. To this is added either violent temper or a feeling of dread which may be vague and unaccountable, or (in bad cases) may arise from actual delusions, such as the belief that an enemy is constantly lying in wait to inflict an injury, &c. This sort of delusion is not to be confounded with another kind, which consists in a vivid apprehension by the patient that he is in danger of falling down a precipice even when he is walking on firm ground in broad daylight, and which seems to me to be connected with rapidly progressing impairment of muscular *co-ordination*. Cases which display the latter feature are commonly of a dangerous type, and, unless energetically treated, pass rapidly to a hopeless condition as to recovery. The sensation as described to me is not like that of ordinary vertigo, or of fainting; it resembles the disagreeable nightmare which every one has experienced on first falling asleep after an indigestible supper, or, still more closely, the hideous feelings which some persons (myself among the number) suffer from under the action of a large dose of Indian hemp. But it is not usually found among the earlier symptoms of Alcoholism.

The above is a fair description of the nervous symptoms under which the patient commonly suffers when he first applies for relief. The disorders of common sensation which are frequently produced by alcoholic excess are, in my experience, usually later in their advent. When the patient comes under notice, he may present either of several conditions as regards his outward appearance. There is not often, at this early stage, any very great emaciation, even in the case of habitual spirit-drinkers; but there may be every degree of fatness, from the unwieldy bulk of the country publican, who chiefly fuddles himself with beer, to the slight frame of the London hairdresser, who too often makes away with two or three quarterns of gin or rum daily. It is a great mistake, however, to push so far, as is often done in descriptive works, the contrast between the respective influences of spirit-drinking and beer-drinking. The haggard wretches

whose portraits Hogarth has drawn in his picture of "Gin Lane" are emaciated to that degree quite as much from utter want of all the comforts of life as from the direct influence of spirit-drinking; and, in fact, one sees, in the classes whose circumstances are a shade more easy, plenty of gin-drinkers who (living chiefly on gin) have a good allowance of fat, if not of muscle. The countenance of the drinker (whether of spirits or beer) usually presents two remarkable features in conjunction, viz., great flabbiness of the muscles of expression, and red, watery eyes; the conjunctivæ are also very generally more or less jaundiced. To this is often added redness of the nose and cheeks, and an eruption, resembling acne rosacea, around the nose and the mouth. On inquiry we learn that, besides the already-described nervous symptoms, the patient suffers from morning vomiting, or at least nausea. This is nearly always the case, but there may be any amount—or no amount—of general symptoms of gastric or intestinal irritation, except this one symptom; and the tongue, in correspondence with these variations, may be in nearly any state, from perfect cleanness and moistness to dry red glaziness or thick yellow furring: the latter is its more common condition, especially at the back part. The morning vomiting is in my opinion not a mere dyspeptic disorder, but a true part of the nervous phenomena of receding narcosis. One symptom, which it is not easy to explain, but which nearly always exists, even where there are no signs of dyspepsia, is a peculiar foul breath-smell, which it is impossible to describe, or to mistake when once it has been smelt. It is quite unlike the odor of the alcoholic liquor itself, and may be separately distinguished even when the latter is also present.

Considering the enormous quantities of spirituous liquors which are drunk by many of the patients who apply for relief from the consequences of chronic Alcoholism, it would be natural for the reader who holds the usual opinion as to the origin of cirrhosis of the liver to expect that serious symptoms, produced by the latter disorder, must often complicate cases of the former. The case, however, is far otherwise, in my own experience. Of an immense number of patients in whom the nervous disorder has been clearly identified, I have only seen thirteen cases in which the symptoms of cirrhotic disease called for any special treatment, although a certain degree of cirrhosis was doubtless present in many of the others; and I cannot avoid the conclusion that some very powerful element, over and above the influence of alcoholic excess, is needed to produce the severe type of that disease. To a less, but still a remarkable extent, the same observation holds good for kidney disease of the degenerative kind. With regard to these disorders, I am convinced that other depressing influences must bear a large share of the blame ordinarily attributed to alcohol. How is it possible to form any other opinion, when of the multitudes of drinkers whose kidneys must be daily traversed by blood containing large quantities of alcohol, so few present any characteristic change of the urine, or other recognizable symptoms of renal mischief? Be this as it may, it is certain that renal, and still more hepatic, complications are very rarely the source of serious embarrassment in the treatment of chronic Alcoholism of the ordinary type which is indicated by such a group of nervous symptoms as is above described.

Not to anticipate unduly what will have to be said under the head of Prognosis, it may be stated here that the form of the disease which we have so far considered, is decidedly curable, tending in fact to right itself on the simple adoption of a plan of complete abstinence from the exciting cause of the mischief, combined with a nourishing and supporting diet, unless in the rare instances where sundry complications, which may fairly be called *accidental*, happen to receive a dangerous development. These complications arise out of the local irritant action of the more concentrated alcoholic liquors on the gastro-intestinal mucous membrane or on the air-passages, and will now be described.

The irritant effects of alcohol on the alimentary canal are chiefly seen in the case of spirit-drinkers, and more particularly in those who drink spirits *neat*, or highly concentrated. Beer-drinkers do, indeed, often suffer from a simple form of dyspepsia, and there is little doubt that slow degenerative changes are usually set up in the stomachs of these patients; but, except in the case of enormous habitual excesses, the dyspepsia is a transient phenomenon which rapidly disappears on the adoption of a rigid plan of abstinence together with a simple medicinal treatment. The more concentrated alcohols, however, when used for any length of time, may set up a formidable irritation which produces intense congestion of the stomach or the intestines, or both: in short, a greater or less portion of the tract in which the radicles of the portal veins take their rise is subject to severe engorgement. Perhaps the most serious consequence of such an action is the occurrence, which we now and then witness, of *profuse hemorrhage* from the stomach or bowels. According to what I have seen, this is rare. I have not met with a dozen cases of this kind altogether: two of these—one a case of hæmatemesis, and the other of intestinal hemorrhage—occurred in the same week,

in the practice of Westminster Hospital, quite lately. It is a frequent thing, however, for drinkers to be affected with hemorrhoids, from which more or less bleeding takes place.

Great numbers even of the heaviest drinkers never develop any further *specific* symptoms of Alcoholism than those which have been already described, and their vicious habit, if it shortens their lives, does so chiefly by impairing their general nutrition, and thus rendering them less able to resist the attacks of intercurrent acute disease, and at the same time more predisposed to constitutional maladies, such as gout for instance, to which they may chance to have a hereditary bias. Others suffer from attacks of delirium tremens (to be presently described), once and again. But in many other drinkers the nervous symptoms, still preserving a more or less chronic type, assume a far more serious development; and we have now to speak of these more extreme developments of chronic Alcoholism.

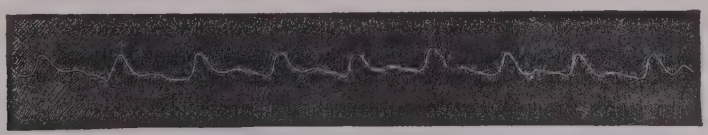
Of the earliest symptoms which indicate a dangerous degree of nervous degeneration, the occurrence of marked sensory paralysis is one of the most frequent. Unlike the corresponding affection of the motor nerves, sensory paralysis is most commonly exhibited in a slight degree in the upper extremities before it appears in the lower. The occurrence of any considerable degree of sensory palsy in the lower limbs is a sign of grave import: the patient so affected, unless he be induced at once to adopt a proper abstinence, and an appropriate medical treatment, is almost certain very quickly to experience some serious organic lesion of the brain. Simultaneously with the occurrence of a considerable degree of sensory paralysis, there is usually a great development of the muscular tremor, which, in several cases which I have seen, approached closely to the type of paralysis agitans. The mental powers are by this time usually affected in a marked degree—the most common mental condition being one of general intellectual enfeeblement and moral degradation, marked by cowardice and untruthfulness. At this point the progress of the case may diverge in either of several directions. In patients whose family history is strongly marked with the taint of insanity, a tendency to suicide is often developed, or else the sufferer sinks rapidly into a state of confirmed and incurable dementia. In others the function of muscular co-ordination is interfered with to a degree which makes the case resemble, at first sight, the affection known as Locomotor Ataxy. In others there occurs a sudden break-down of nervous fibres in the corpora striata, or optic thalami, which produces a stroke of hemiplegic paralysis. In others, along with some symptoms of mental alienation, a general motor palsy is so distinctly observed as strongly to suggest the idea of commencing general paralysis of the insane. In others the rupture of a cerebral artery leads to an effusion of blood and the sudden occurrence of an attack resembling ordinary apoplexy. In others, again (but this is a very small class), the patient suffers attacks of convulsions indistinguishable from those of simple epilepsy. Epileptic attacks, occurring in this way, as a symptom of a very advanced stage of the nervous degeneration developed by chronic Alcoholism, are broadly distinguished, in a clinical and prognostic point of view, from the much commoner attacks of epilepsy in a subject known to be predisposed to or actually affected with that disease, as a mere consequence of a somewhat unusual alcoholic excess: the latter are of comparatively slight consequence, while the former indicate an altogether hopeless phase of alcoholic degeneration of the nervous centres. They are almost always accompanied by an advanced degree of dementia.

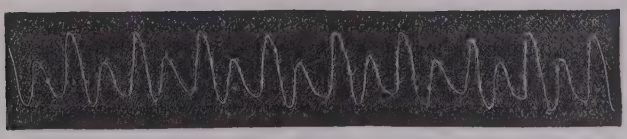
(B) *Symptoms of Acute Alcoholism.*—If we set aside the case of common drunkenness, as being rather an instance of narcotic poisoning, to be dealt with by toxicologists, than a morbid affection coming under the definition which we have placed at the head of this article, we may describe the symptoms of acute Alcoholism as presenting themselves under four principal forms—namely, Delirium Tremens, Acute Mania from drink, Acute Melancholia from drink, and Oinomania.

1. *Delirium Tremens.*—The clinical history of this disease was much misunderstood in former times. It used to be believed that in the majority of cases the delirious affection was produced, not by the direct poisonous action of alcohol upon the nervous system, but by the circumstance of an habitually intemperate person's leaving off the use of his accustomed potations. As a matter of fact it had frequently been observed that a sufferer from delirium tremens had ceased to drink for one, two, or three days before the access of his more acute symptoms, and the exhaustion caused by the loss of his ordinary stimulant was supposed to produce those symptoms. Dr. Ware, of Boston (1831), was one of the first writers who pointed out that this statement includes a fallacy of observation. From an analysis of 100 cases, he proved that the cessation of drinking, where this occurs, is in fact produced by a feeling of revulsion to strong liquors, which is a part of the early symptoms of the acute disease in many cases; and, on the other hand, that very many patients do not leave off drinking at all, but the delirious attack supervenes in the midst of a debauch. This observation

has been confirmed by Dr. Gairdner, and many other excellent writers, and at present the classical theory of exhaustion from withdrawal of an accustomed stimulus has but few upholders. [Surgeons are familiar with the occurrence of delirium tremens in intemperate persons who have suffered severe injuries, or have undergone amputation or other severe operations. It is reasonable to suppose that this *may* be due to the contributive or exciting causative action of surgical shock, and not to the withdrawal, under treatment, of their accustomed excessive stimulation. Yet it is hard not to think, that in these, and some other cases of suddenly enforced abstinence, this withdrawal does have something to do with bringing on the attack.—H.]

The first warning of the approach of delirium tremens is ordinarily given by the occurrence of complete insomnia. The patient may have long indulged to excess in drink, or he may be quite a novice in intemperance, but in any case a greater debauch than usual has commonly been perpetrated; and the sufferer finds himself quite unable to obtain any sleep, or at most can only gain short snatches of slumber, disturbed by horrifying dreams and visions: and during his waking moments, even in broad daylight, he suffers from hallucinations of sight which commonly take the form of disgusting or terrifying objects, such as snakes, insects, monsters, or of armed men pursuing him with threatening gestures. More rarely he hears voices denouncing threats, or mocking him: occasionally he experiences delusive sensations of disgusting smells. Often the occurrence of distinct visual hallucinations while the patient is awake is the first sign of the passage from chronic Alcoholism (which may have lasted for months or years, with a varying degree of insomnia, and perhaps with habitually distressing dreams) to the acute affection. During the first day or two days the patient is in an extraordinarily depressed state, with slow and feeble pulse, cold extremities, and a profuse sweating. The mental state is one of great anxiety, but there are usually no real *delusions:* even where visual hallucinations are present, the patient can by an effort of the will recognize them as such, and momentarily banish them from his sight. During all this time there is so complete an absence of appetite, in the great majority of cases, that no food, or scarcely any, is taken, and this circumstance probably mainly conduces to precipitate the onset of the second stage. In this the mere anxiety and nervousness is exchanged for incoherence of speech and wild excitability of manner, which sometimes takes the shape of causeless anger (though even then nearly always mixed with cowardice), and sometimes of great terror, which the sufferer often accounts for by pointing to imaginary terrific shapes which seem to people the room, and which he is constantly seeking to push aside with a restless motion of his hands. He talks incessantly, in a rambling fashion. Even when his terror or his anger is at its height he can generally be momentarily restrained by the influence of any onlooker who addresses him in a firm and determined manner, and may even be reasoned temporarily out of his hallucinatory imaginations. The pulse has now become quick (from 100 to 130 or 140 a minute): it is sometimes small and thready, sometimes soft and voluminous: but in every case which I have examined it gives a tracing, by the use of Marey's sphygmograph, in which the form of the pulse-waves closely resembles that which is observed in fevers and inflammations of a typhoid type, and is especially remarkable for the prominence of the phenomenon called "dicrotism." The annexed tracings will give a more accurate idea of the quality of the pulse than any description of the sensations which it communicates to the finger:—





Muscular *tremor*, which, from its striking prominence in many cases, has given the disease its name of *delirium tremens*, is by no means universally present. According to Craigie, whose observations on this point I believe to be correct, they are usually observed in the cases of confirmed dram-drinkers; and in many instances I have found on inquiry that they were only an exaggeration of a tremu-

lousness of the extremities which had already existed for months or for years. But even when the characteristic tremulous movement of the arms and hands is not present there is a constant restlessness; the patient shifts constantly in the bed, and will get out of it twenty times in an hour if he be permitted to do so. The eyes are in almost constant movement; the pupils are usually, though by no means always, dilated. The temporal and carotid arteries throb violently in most cases; very often the face is flushed, but sometimes it remains deadly pale; nearly always there is much sweating, which is obviously due, in great part, to the constant muscular movements. The tongue is protruded, on the request of the physician, with an almost choreic jerk. It almost always trembles; usually it is covered with a yellowish fur, but it may be clean, red, and glassy on the one hand, or brown, dry, and cracked on the other.

It is usual to assign a limit to the second stage (which may last one, two, or several days) at the period when the patient first falls into continuous slumber; and no doubt the classical descriptions which assign this as the critical event to which convalescence may be expected to succeed, find a considerable superficial justification in clinical facts. But, in common with some of the most careful observers, I believe that to be a very erroneous and mischievous opinion which ascribes to a few hours' sleep anything like a distinctly curative power. It is true that in many, perhaps most, instances, the patient awakes, after his first sleep of considerable duration, in a condition of comparative convalescence. But, on the other hand, numerous cases have been observed in which the patient has sunk into profound slumber for many hours, and has awakened as delirious as ever, or in a state of complete prostration, which has rapidly terminated in death. Interesting considerations will be brought forward on this point, under the head of Prognosis, particularly with regard to the condition of the pulse, and the amount of success which has attended the efforts of the attendants to get the patient to take nourishment. In fact, the occurrence of sleep, even of considerable duration, marks with accuracy the commencement of convalescence only where we find the patient, on waking, clear in his intellect, free (or nearly so) from hallucinations, and with a pulse greatly reduced in frequency and yielding a sphygmographic trace such as will be presently described. The stage of convalescence, once established, presents nothing particularly worthy of description. But instead of sleep occurring at all, the patient may pass from mere delirium into a comatose condition, with muttering delirium, eyes open, staring, and fixed, restless movements of the limbs more marked than ever, picking at the bed-clothes, or possibly profound stertorous coma, or violent convulsions, these symptoms being followed speedily by death. In other cases the patient, in the midst of violent delirium, with great excitability, suddenly collapses, as it were; the pulse becomes hurried, intermittent, and thready; the features pinched and ghastly, the breathing gasping, and death ensues in a minute or two, sometimes even in a few moments.

2. *Acute Mania* from drink presents symptoms which, though sometimes puzzlingly like those of simple delirium tremens, can usually be discriminated from the latter. The patient, who (invariably, as far as my experience goes) possesses some hereditary predisposition to insanity, is seized, in the midst of a drinking bout most commonly, with active maniacal delirium of a violent kind, and frequently displays a marked tendency to homicidal acts. In most of the cases which I have seen the whole aspect of the countenance and manner of the patient is different from that of delirium tremens, and there is comparatively little of the busy tremulousness of the hands so often seen in the latter disease. I believe that cases which are attended with positive intellectual *delusion* are nearly always of this, or else of the melancholic kind. The pulse, whatever its degree of apparent strength or weakness, as tested by the finger, is seldom so markedly *dicrotous* as in delirium tremens. [A modification of this kind of attack occurs in rare instances, to which the name of *alcoholic phrenitis* or *inflammatory delirium* may be applied. I have seen but one case of it; but the same man had been two or three times similarly affected, as I learned from his previous medical attendant. After a hard drinking spell, he became delirious; with very little tremor, a hot head, skin generally of a high temperature, pulse rather rapid, full, and strong. Acting upon information received of his other attacks, I took several ounces of blood from his arm.[1] This, with a saline cathartic, was followed by improvement in all his symptoms, and recovery within a week. Altogether exceptional as such cases are, it is well to be aware that they are possible.—H.]

3. *Acute Melancholia* from drink presents the usual characteristics of melancholia from any other cause, but is marked by a special tendency to suicidal acts. The influence of a sound, protracted slumber, which in *mania* from

[1 It is proper to say, that, agreeing with the general principles of Dr. Anstie's article, I have never taken blood in any other case of delirium from alcoholism.—H.]

drink is usually very beneficial, is far less so in *melancholia* from the same cause, as far as my limited experience goes.

4. *Oinomania.*—The fourth variety of acute Alcoholism is that curious affection which Roesch was the first to describe with precision, and which is now commonly called oinomania. It is, in truth, rather a variety of constitutional insanity than of alcoholic disease; but as the outbreaks owe many of their characteristic symptoms to the influence of drink, the disorder requires notice in a treatise on Alcoholism. The sufferers from oinomania are, I believe, usually descended of families in which insanity (and often insanity of the same type) is hereditary. Patients of this class very commonly, though not always, display their tendencies early in life; sometimes, indeed, on the very first occasion on which the opportunity for the free use of strong drink presents itself. It should be clearly understood that the term "monomania," which is often applied to the disease, very imperfectly describes the condition of the victims. Closer investigation of their mental state will usually discover the fact that they are liable to periodical recurrences of causeless exultation and bursts of self-confidence on trifling occasions; they then display great obstinacy, and a marked excitement of the animal passions generally: indeed the commencement of a drinking bout is often accidentally precipitated by the circumstances of temptation in which they are placed by loose company. Under the influence partly of an uncontrollable impulse, and partly of intoxication, they will perform truly insane acts; they take useless and purposeless journeys to remote places, or they lose their usual sense of decency, and expose themselves to disgrace by public acts of a degrading character. They exhibit symptoms which in many respects resemble those of simple delirium tremens, though there is usually a marked absence of that anxious terror which is almost always present in the latter complaint, and also a far less decided incapacity to sleep; indeed, there is sometimes very little insomnia. After lasting for a few days, a week, sometimes even a month or six weeks, the attack seems to wear itself out, as if rhythmically; and the patient generally recovers very rapidly his usual health, though he suffers "horrors" for a day or two. The condition of these patients in the intervals between these attacks is very different from that of the ordinary confirmed sot. Very often they live perfectly sober and chaste lives, and are even remarkable for active and intelligent management of their affairs. But this condition only lasts for two or three months, or six months, or at most a year, and then the old symptoms recur, and the patient is uncontrollably hurried into excesses of the most violent kind. Very rarely indeed is a sufferer from this disease really cured; it usually recurs with increasing frequency throughout life, and frequently ends in declared and permanent insanity.

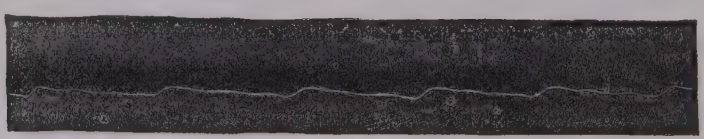
[It seems to be more generally admitted by the profession in America than in Great Britain, that *uncontrollable* intemperance constitutes a form of insanity in a large number of instances. Opinion is divided, however, here as well as abroad, as to whether the drinking habit *alone* is to be credited, in most cases, with the production of oinomania (or methomania, a better term) as an affection in which the will is overborne by the morbid impulse to drink liquor. Some consider that either insanity or a predisposition to it existed before; of which the methomania is only a partial manifestation. My own conviction, from observation, is, that long-continued intemperance very often does induce, in persons otherwise sound in mind, a condition, mental and physical, in which, with opportunity and the absence of restraint, it is impossible for them to resist the tendency to take alcohol in excess. This may be most properly called methomania.—H.]

Diagnosis.—The diagnosis of alcoholic diseases of the nervous system is not unfrequently surrounded with difficulties, especially in the case of the chronic forms. Chronic Alcoholism produces nervous symptoms which are particularly liable to be confounded with the following diseases: 1, chiefly with commencing general paralysis; 2, with paralysis agitans; 3, with lead-poisoning; 4, with locomotor ataxy; 5, with hemiplegia or paraplegia from ordinary softening of the brain or spinal cord; 6, with epilepsy; 7, with senile dementia; 8, with hysteria; 9, with the nervous *malaise* associated with some forms of dyspepsia. The general group of leading symptoms whose presence enables us to affirm the diagnosis of chronic Alcoholism rather than that of any of these diseases is as follows: The patient suffers from restlessness of mind (without delusions), insomnia, muscular fidgetiness, or actual tremor, morning vomiting; and presents flabby features, and watery eyes, and slight jaundice of the conjunctivæ. These symptoms make the diagnosis highly probable. If to them is added the occurrence of vertigo, muscæ volitantes, and terrifying dreams, it is greatly strengthened; and it is raised to the point of certainty, in my opinion, if there be also actual visual or auditory hallucinations in the form of visible shapes of men, beasts, &c., or audible voices. Indeed, the concurrence of distinct visual or auditory hallucination with

only four other of the above-mentioned symptoms—viz., insomnia, morning vomiting, muscular tremor, and causeless mental restlessness—would of itself very nearly persuade me of the existence of alcoholic poisoning. Cases of commencing general paralysis (the most embarrassing counterfeits of the disease) may nearly always be distinguished by the presence of *mental exaltation*, the condition of the toper being uniformly one of mental depression, on the whole. The very rare cases of general palsy which do not display mental exaltation are wanting in the other features of Alcoholism, unless indeed when drink has been the exciting cause. As far as I have seen, chronic alcoholic poisoning *always* produces three or four of the leading symptoms which I have mentioned as specially diagnostic; and where an acne-like eruption of the face is also present, this settles it. The diagnosis of the acute forms of Alcoholism is usually far less difficult. We can generally get at a knowledge of the patient's mode of life in these cases; whereas the chronic toper is very commonly, especially if a woman, most cautiously and skilfully reticent and deceitful, and often conceals her habits even from her nearest relations. A case of considerable difficulty may arise in the distinction between delirium tremens and some forms of acute mania not caused by drink. The existence of delusions, not mere terrors, should bias us in favor of the diagnosis of mania, as should also the tendency to commit particular acts of violence, and especially lustful propensities; while the predominance of hallucination, especially when combined with terror of mind, tremor, and busy delirium, should predispose us to recognize delirium tremens. For the means of diagnosis between the different forms of acute Alcoholism the reader is referred to what has already been said under the heading of symptoms.

Prognosis.—The prognosis in chronic Alcoholism, except in its more advanced forms, which are marked by the occurrence of serious paralytic or convulsive symptoms, or by considerable mental impairment, is highly favorable as regards recovery from the immediate symptoms. Mere abstinence, combined with simple but energetic treatment, to be presently described, will suffice in such cases to procure a rapid removal of all the unpleasant symptoms. Unfortunately, too many patients are biased by long habit, by hereditary constitution, or by the dismally depressing circumstances of their daily life, in a way which renders their return to intemperance indefinitely probable. When once the more serious symptoms—such as paralysis, or epilepsy, or extreme and persistent muscular tremor—have occurred, cure, even for a time, is far more difficult, and the moral degradation of the patient, especially if a female, is so great as to allow small hope that abstinence will be observed.

In delirium tremens the main elements of prognosis are the occurrence or non-occurrence of sleep before the patient is very much exhausted, the condition of the pulse as tested by the sphygmograph, and the degree of success which attends the physician's efforts to get nourishment into the system. Sleep, as already remarked, is not of itself curative. The disease, in proportion to its original virulence, has a course of longer or shorter duration to run: this depends in great measure on the quantity of the poison taken, the sufficiency of the assimilative processes, the original strength of the constitution, and the degree in which it can be supported by well-assimilated food. Thus the prognosis is bad in the extreme when the dose of poison has been very large, the patient's constitution feeble, his powers of assimilation weak, and, in addition to this, disease of the glandular organs (especially of the kidneys) exists. Such a case is well-nigh hopeless. Almost equally bad is that in which any severe degree of *pneumonia* complicates the malady. The test, however, of the patient's chances which more than any other I am inclined to value, is the indications given by Marey's sphygmograph. In proportion as the pulse shows a tendency towards the normal form indicated by this tracing



are the chances good. On the contrary, such a pulse as the following

offers the extreme type of that typhoid form which is of most evil augury. This latter tracing was taken from a man, aged 40, who, after remaining for nearly a week in the delirious stage, fell into a sound sleep, which lasted for six or seven hours, awoke

apparently so much improved as to his nervous symptoms, that a somewhat confident opinion was pronounced in favor of his recovery. I augured the worst from the pulse-tracing; and in fact the patient sank rapidly, about twenty-four hours later. A somewhat extensive experience of this means of prognosis enables me to recommend it with much confidence. Mere rapidity of pulse counts as nothing in gravity, in my opinion, in comparison with the obstinate maintenance of the typhoid form of pulse-wave.

It is almost needless to remark that the circumstance of an attack being the first of the kind which the patient has suffered, renders it much less dangerous to life, as a general rule, than a second, a third, or a fourth would be; but there are important exceptions and qualifications to this law. Thus it may happen that a first attack of delirium tremens seizes a patient who has passed the line of middle age, and whose nervous system has been already much enfeebled by chronic disease or bad feeding, but who has never till recently indulged to excess in drink. Such an individual runs a great danger of sinking under the first acute attack; and the reason of this may be partly found in the feebleness of his system, and partly in the circumstance that his eliminating organs, especially his kidneys, have not become habituated to the irritation suddenly thrown upon them by blood containing large quantities of unchanged alcohol. The same embarrassment of eliminating organs suddenly charged with unaccustomed alcohol is doubtless the cause that a *young* man's first debauch (such at that of a young sailor, *e. g.*, put on shore after his first voyage) so often causes an attack of delirium tremens; but here the constitutional strength usually enables the patient to bear up till the natural process of cure has time to be accomplished.

The prognosis both of acute mania and of acute melancholia from drink is decidedly good, at any rate on the occasion of first attacks, and provided that the affection is promptly treated. The probability of the case passing into one of confirmed insanity is of course progressively increased on the occasion of each successive acute attack.

The prognosis of oinomania is in one way very hopeful, in another almost entirely hopeless. The attacks of the acute affection may recur any number of times without any serious result: the patient, after a variable number of hours, days, or weeks, returns to his sober senses, and resumes his usual course of life. The hopelessness of the case lies in the taint of insanity which almost always lies at the foundation of the complaint, and which makes it almost impossible that the patient can effect a thorough reformation of his habits. However virtuous his intentions may be, and however strongly he may be urged by every consideration of prudence, or affection for those whose interests may depend upon his conduct, it appears as if he were impelled by a really irresistible force to yield himself, at certain intervals, to the temptation of drink. When the outbreaks become, as they usually do in the end, greatly more numerous than at first, there is reason to apprehend the speedy supervention of confirmed insanity.

Complications.—Of the complications of *chronic* Alcoholism it would be impossible to speak in detail, on account of their great number and variety. The only point to which I think it necessary to direct attention, is the question of the comparative liability of drinkers and of sober persons to phthisis. It appears certain, from the most careful statistics, and especially from those recently collected by Dr. Sutton, that the liability of drinkers to the ordinary forms of phthisis is considerably less than that of temperate people. On the other hand, every physician has now and then observed cases, which may be classed as "galloping consumption," which have occurred in persons who have been leading drunken lives, and which arrive with great rapidity at a fatal termination. I believe these victims of acute phthisis from drink are always descended of tuberculous families; and I think it likely that the starting-point of the actual tubercular deposit is to be found in continuous paralysis or semi-paralysis of the "nutritive" fibres contained in the pulmonary branches of the pneumogastric nerve, which is kept up by the patient's drinking habits.

Of acute Alcoholism, the only complication of which I shall separately speak is that of pneumonia. Nothing is more insidious than the occurrence of pneumonia in a subject whose nervous system is deeply poisoned with alcohol. A crucial instance of this occurred in the person of a patient who died in King's College Hospital many years ago, without its being suspected that anything more than delirium tremens was amiss, but whose right lung proved, on post-mortem examination, to be hepatized from apex to base. In this case there was no cough, no expectoration, no pain in the chest, and only so much frequency of breathing as seemed sufficiently accounted for by the restless muscular movements of the patient. It is most important, in every case of delirium tremens, that the chest should be periodically examined with care.

PATHOLOGY.—The pathology of Alcoholism naturally divides itself into three portions. The morbid influence which the poison exerts is of three kinds: in the first place, it acts as a local irritant (when highly concentrated) upon the mucous membrane of the stomach and the alimentary canal generally; and in the second place, after absorption, it affects the rate of movement and the vitality of the blood, and as a consequence of this impairs the nutrition of every organ of the body. And thirdly, it is clear that the nervous centres, independently of the ill effects on their nutrition of the blood-changes, have a certain chemical attraction for alcohol, which accordingly is found to accumulate in their tissues.

In the alimentary canal, and particularly in the stomach, the local effects of habitual large doses of concentrated alcohol are seen in permanent congestion of the bloodvessels, exaggerated or vitiated secretions from the gastric glands, and ultimately a degenerative change in the structure of the submucous tissues, which consists in the disappearance of characteristic secreting structures, and the hypertrophic exaggeration of fibrous tissue. Absorbed into the blood in large proportions, alcohol increases largely the amount of fatty matters in that fluid, and promotes congestion of certain important organs. The congestion of the lungs, liver, kidneys, &c. seems to be partly due to altered chemical relations between the blood and the tissues of those organs, and partly to a paralytic action of the alcohol upon the vaso-motor nervous system. It is by this latter action that I am inclined to account for the abnormal production of *sugar* in the liver, which has been experimentally observed by Bernard and Harley to follow the introduction of concentrated alcohol into the portal vein, and also for a largely increased excretion of water from the kidneys, which is one of the most invariable consequences of large doses of alcoholic liquors. It is indeed doubtful whether the degenerative changes which result from prolonged alcoholic poisoning are not in great part due to the direct chemical influence of alcohol upon the nervous tissues. The characteristic changes which have been observed in the brain, medulla oblongata, &c. of confirmed drinkers, consist essentially of a peculiar atrophic modification, by which the true elements of nervous tissue are partially removed, the total mass of nervous matter wastes, serous fluid is effused into the ventricles and the arachnoid, while simultaneously there is a marked development of fibrous tissue, granular fat, and other elements which belong to a low order of vitalized products. Essentially similar changes are observed in the lungs, the liver, the kidneys, the heart, and the larger arteries, which (after the nervous centres) are the most frequently affected. The cranial bones are also thickened by a deposit which is not of the nature of a true hypertrophy, for the bones lose much of their original texture, and become dense, almost porcellanous. There is much in these changes which reminds us forcibly of the effects on nutrition of tissues produced experimentally by Schiff and Mantegazza by the section of compound nerves, such as the fifth cranial, and the sciatic and crural of the lower limb; and suggests the idea that in alcoholic poisoning the starting-point (or at least one starting-point) of degenerative tissue-changes may consist in paralysis of those nervous branches which preside specially over nutrition, the distinct character of which has been so well pointed out by Brown-Séquard.[1]

It is highly probable, however, that a considerable portion of the degenerative influence of the continued excessive ingestion of alcohol is due to a chemical interference with the natural course of oxidation of the blood and tissues. Notwithstanding all that has been urged in favor of the view that alcohol is not transformed within the body, the balance of evidence is strongly in favor of the belief that a considerable portion of every dose of alcohol which is ingested does undergo oxidation in the system, and that to the diversion from its ordinary purposes of the inspired oxygen must be ascribed the diminished activity of elimination of carbonic acid, of urea, of chlorine, and of the acids and bases of the urine, which undoubtedly does occur in the subjects of alcoholic poisoning.

TREATMENT.—1. The treatment of the chronic form of Alcoholism varies according to the stage of the disease which has been reached. In that large majority of the cases which come under our notice, in which the patient merely complains of nervousness, of inability to sleep, of muscular tremor, and perhaps of the slighter forms of visual hallucination, together with some dyspepsia and with morning vomiting, the treatment required is extremely simple. One has only to insure that the patient practises a proper abstinence from drink—to insist upon his taking a diet as rich in nitrogenous matters as may be, but at the same time such as his digestive system can appropriate—and to administer certain tonic medicines; and in nearly every case we may count upon a rapid disappearance of the un-

[1] *Vide* Lancereaux, Archives Gén., Oct. 1865, for a full account of the Morbid Anatomy of Alcoholism.

pleasant symptoms of which he has complained. With regard to the first item, the prescription of abstinence from drink, a good deal of difficulty may arise, and there is room for difference of opinion as to the expedient course. I wish to express the decided opinion that complete abstinence may always be carried out without any immediate danger to life or health, if proper care be taken to substitute a substantially nourishing diet. The danger of pursuing this course is not a physical but a moral one: all kinds of pledges which, as it were, *bind* the individual, have a tendency to lessen the force of such notions of personal responsibility as he may retain; he is apt to rest his confidence on the oath or formal resolution, which he has taken, instead of teaching himself the virtue of self-restraint, as he would have to do if he were to accustom himself to the moderate use of alcoholic liquors. This is a question, however, which must be left to the practitioner's judgment in each case. The administration of a highly animalized diet is often a matter of difficulty at first, owing to the feebleness of the digestive powers, which renders the use of solid meat impossible, and even that of soups very difficult. Under these circumstances the greatest possible benefit may be derived from the administration of some of the better so-called "concentrated" preparations of meat, more especially Gillon's beef-juice, and a solid extract from this which is prepared by Messrs. Bell, of Oxford Street, as also the better specimens of the extractum carnis of Liebig. [I have found to answer very well, a strong homemade beef soup; with the fat carefully removed, and a good seasoning of *cayenne pepper*.—H.] Without entering into the vexed question of the exact nutritive value of these preparations, there can be no doubt that they are powerfully reviving to an exhausted nervous system, and that simultaneously with the general improvement which they produce, the digestive organs become strengthened to deal with more bulky forms of animal food. The direct medicinal treatment of chronic Alcoholism in its milder forms is very simple. The presence of dyspeptic symptoms, unless they are very aggravated, and there is reason to believe that serious organic changes in the abdominal viscera have taken place, ought not to distract our attention from the main object of fortifying the nervous system; for with the observance of a proper abstinence from their exciting cause they will rapidly subside. The nervous tonic in which, after a great many trials of different remedies, I have come to repose the greatest confidence, is *quinine* in one-grain doses two or three times a day. It should be given from the very first, if possible; and this may be done, even when the stomach is very irritable, by administering the remedy in effervescence, with bicarbonate of potash and citric acid. The symptoms which most of all distress the patient, in the majority of cases, are the persistent wakefulness and the tendency to visual hallucinations or to appearances of black specks, flashes of fire, &c., before the eyes: the insomnia is also, of course, a great obstacle to that repair of the nervous energy without which recovery is impossible. But it would be a mistake to suppose that soporific narcotics, in doses which in a comparatively healthy patient would produce a stupefying effect, are well adapted to relieve this wakefulness: on the contrary, they generally aggravate the nocturnal restlessness, besides seriously impairing the general health. Nothing has been more marked, in my experience, than the superior efficacy of direct tonics, and especially of quinine, in producing that nervous tranquillity which makes sleep possible. When these medicines prove insufficient, I have found a remedy, which has been recommended by several authors, very useful—namely, sulphuric ether, either given in half-drachm doses three times a day, or a single dose of one drachm at bedtime. A good addition to such a night-draught is half a drachm of tincture of sumbul.

Another remedy, which has proved very successful in the hands of my friend and late colleague, Dr. Marcet, is the oxide of zinc, which, according to that author, has a powerful effect in inducing sleep. He recommends it to be used at first in doses of two grains twice daily, but this quantity may be progressively increased, if necessary, until ten, twenty, thirty grains daily, or even larger quantities, are taken. I have given this medicine very patient trials, both in the smaller and in the larger doses, and I cannot say that I have been so favorably impressed by its action; and on the whole I am inclined to think that in the majority of cases quinine acts much more satisfactorily. It must also be borne in mind, as Dr. Marcet himself admits, that in certain subjects, especially the anæmic and the chlorotic, the continued administration of zinc is observed to produce a prejudicially depressing effect on the constitution. Nevertheless there is no doubt that oxide of zinc occasionally proves a valuable remedy. I think it should not be administered in larger quantities than at most six grains daily; and I concur with Dr. Marcet in the recommendation that it should be given shortly after a meal, as it otherwise sometimes occasions nausea.

A much more effective remedy than zinc appears to be the bromide of potassium in ten- or twenty-grain doses three times a day. Although I have not yet

had the opportunity of trying this medicine so extensively as I should wish, the results obtained have been very good. In several instances it has at once removed distressing wakefulness, dreams, and visual hallucinations. It is occasionally impossible to give this drug, however, from its exciting gastric irritation.

Now and then we find that sleep is not to be obtained by any of the remedies above mentioned, and we are driven to the use of some of the more recognized hypnotics. Of these one of the most effectual is the extract of Indian hemp; it should be given in small doses; from a quarter to half a grain of a good extract is quite sufficient, and a larger quantity is more likely to do harm than good to the majority of patients. Opium, if given at all, should be administered in the form of morphia, hypodermically injected; one-tenth to one-quarter of a grain is sufficient. But a medicine which is quite as effectual in many cases is good bottled stout given in one single dose of half a pint at bed-time.

In the more advanced cases of chronic Alcoholism, where the nervous centres are undergoing serious degenerative changes, as evidenced by the occurrence of paralysis, epileptiform convulsions, or grave mental deterioration, further remedial measures are required. Of these the two which have yielded me by far the most satisfactory results are cod-liver oil, and phosphorus in the form of the hypophosphites of soda or lime. Cod-liver oil, to be really of use, must be continued in tolerably full doses over a long period. Employed for so long a time as three or six months without intermission, I have seen it produce striking benefit even in advanced stages; and in some instances where it failed to produce anything like a cure, it caused great amendment of the most serious symptoms. The hypophosphites in five- and ten-grain doses, three times a day, have been particularly valuable, in my hands, in the treatment of cases which were distinguished by commencing paralysis of sensation. In one case which was marked by epileptic convulsions, with much impairment of the mental faculties, the combined use of cod-liver oil and bromide of potassium produced very beneficial effects. Another class of cases, those in which the predominant symptom is a very considerable degree of muscular tremor, are often greatly benefited by strychnia. Very small doses only are to be used; it is well to commence with the $\frac{1}{48}$th of a grain, and increase this to not more than the $\frac{1}{32}$d of a grain, three times daily. Doses much larger than this have invariably seemed to do decided harm, especially increasing the tendency to vertigo, visual hallucinations, and noises in the ears.

2. The treatment of Acute Alcoholism. (*a*) Delirium tremens is a malady the treatment of which has experienced several changes correspondingly with the progress of accurate clinical observation. In former times—indeed a very few years since—the notion universally prevailed that the delirious symptoms were owing to the exhaustion which was chiefly kept up by want of sleep; and, consequently, that the production of continuous sleep for several hours was the sole and all-important means of cure. It was therefore the custom to ply the patients with larger and larger successive doses of opium, with a view of drowning the delirium in narcotic stupor. Great mischief arose from this wide-spread belief and practice. In the first place, it has often happened that the patient, without ever sleeping at all, has passed first into a condition of coma-vigil, next of stertorous breathing, and at last sunk, fairly poisoned with opium. Again, a fact which was disregarded by the earlier authorities was this, that, without exerting any poisonous action upon the centres of consciousness, opium occasionally spends almost the whole of its depressing force upon the visceral nerves. A minor consequence of neglecting this fact was, that the patient's chance of assimilating food was often entirely ruined by the paralyzing action of the drug upon the digestive organs: a much more serious one was the accident which has doubtless often happened, and which occurred in cases within my knowledge—namely, the rapid induction of a cardiac paralysis, the patient (without any cerebral signs of poisoning whatever) suddenly becoming ghastly pale, the pulse fluttering and coming to a standstill within a few moments. One such example was particularly striking, as it immediately followed two large doses of opium, which had been given in the vain hope of procuring sleep; the second dose was equally inefficacious as a soporific with the first, but its deadly effect upon the circulation could not be mistaken.

The idea that patients in delirium tremens require to be narcotized into a state of repose, may now be said to be abandoned by those best qualified to speak on the subject. In truth, the condition of the brain requires that sort of treatment which shall fortify and stimulate its functions. I have already argued at length, in another work, that every stimulant, when given in such restricted doses as alone deserve that name, is a promoter, but not an exhauster, of function, and that the idea of any depressive *recoil* following its action is purely fictitious. There are, accordingly, a great number of remedies of which the larger doses are narcotic, and the smaller stimulant, which in the latter form are capable of giving more or less

relief to the symptoms of delirium tremens. It is not worth while to enumerate all these. The typical member of the group of stimulants is simple, easily digested food; and the successful treatment of delirium tremens, in nine cases out of ten, depends on the regular and continuous supply of suitable nutriment, whereby the functions of the nervous system are supported during the struggle towards recovery. The principal kinds of food which are desirable are milk, soup, or strong broth with bread in it (and given *very hot*), the concentrated meat-foods already recommended under the head of Chronic Alcoholism, and raw eggs beaten up. The necessity for the administration of some nutriment of this kind is imperative; and if the stomach be at first too irritable or the anorexia too complete to allow of feeding by the stomach, it must be given in the form of enemata, so as not to lose a day, nor even a few hours.

It should be observed, however, that there are two classes of patients, in one of whom it is, and in the other it is not, desirable to employ some preparatory treatment of an *eliminative* kind. The value of *purgatives* has been recognized by many writers. They are eminently suitable to those cases in which a young and somewhat robust person has brought on delirium by drinking a very large quantity of spirits; in such instances a dose or two of medicine, producing free watery evacuations, effects a wonderful improvement (no doubt by ridding the alimentary canal of much of the alcohol which it has taken in). Where the strength of the patient is sufficient to allow of this plan being safely carried out, it will be found that the subsequent assimilation of food is rendered more easy and rapid, and that the stage of convalescence is comparatively soon attained. But in debilitated subjects it is far better not to attempt any forced increase of the eliminative processes, but to commence at once with the administration of the more easily digested foods in small quantities frequently repeated. The irritation of the stomach may be combated by the administration of ice, and of small quantities of soda-water and other aerated drinks, and one of the best modes of commencing the necessary feeding is by administering milk, mixed with one-third its bulk of lime-water, at frequent intervals. Everything is to be hoped for a patient who has been well supported by food from the early stages of the attack.

Of late years an important question has been raised concerning the therapeutic value of digitalis in delirium tremens. The practice, introduced by Mr. Jones of Jersey, of administering very large doses of tincture of digitalis (from half an ounce to an ounce and even more), was a startling innovation on the traditional practice in the use of this drug; such doses having formerly been universally regarded as dangerously poisonous, and calculated to produce fatal depression of the circulation. It has been proved, beyond doubt, that in a large number of cases these doses are at least harmless, and the testimony of a good many observers has now apparently established the fact that the delirium may frequently be quieted, and sleep obtained, by the employment of digitalis in this manner. It must be owned, however, that the question still remains in a very unsatisfactory position. The great majority of the cases have been treated with the *tincture*, and not with any simple preparation of digitalis: that is to say, the patients have, in fact, received half-ounce or ounce doses of *proof spirit* over and above the drug intended to act upon the disease. But it is well known that alcohol, in common with all the stimulant class of remedies, has often a beneficial influence in states of low delirium. In the presence of the very conflicting statements on the action of digitalis which have been published by different writers, I have endeavored to clear the matter up by employing a strong *infusion* instead of the tincture; but it is unfortunately impossible, in many cases, to get the patients to take the remedy in this shape, and I have thus been hindered from effectively carrying out the experiment. The powder, given in pills, would be a better form. From the observation of a few cases treated with digitalis, in one form or another, which have been under my own treatment or that of friends, I have been led to the provisional conclusion that in all probability a large number of the reported successful cases have either been instances of a spontaneous favorable termination of the disease, or have been slightly helped towards their happy issue by the alcohol which is contained in the tincture ordinarily employed. This consideration leads us naturally to consider the very important question—whether alcoholic liquors should or should not be used in the treatment of delirium tremens.

I am inclined to think that the moral argument has great weight here. In all cases, and more especially in first attacks, the subjects of which, we may hope, are not irremediably debased by drunken habits, it appears to be incumbent on us to use the time of sickness as an opportunity for possible reformation, unless alcohol were *necessary*. It would, therefore, seem to be our duty to commence the work by giving the patient's system an entire rest from the action of alcohol during the period for which he is under our authoritative guidance. In young subjects, therefore, and in first attacks, it is proper to abstain altogether from the use of alcohol.

It is more difficult to carry out this plan with older patients, and with those who have been for a long time accustomed to depend upon strong drinks for a large part of their ordinary nutrition. In every case, however, I think it is our duty to abstain as long as possible from the use of alcohol, and before resorting to a treatment of such doubtful propriety, we ought to try less harmful narcotic stimulants. Opium and Indian hemp fulfil the indications which we require, under these circumstances, better than any others of their class. Opium *should never be administered by the stomach*, but always in the form of morphia hypodermically injected, in the dose of one-tenth to one-fourth or one-half grain. Where there is any reason, from the quality of the pulse, to believe that the circulation is much enfeebled, Indian hemp, in doses of a quarter to half a grain of good extract, is a less objectionable remedy, and I have seen it produce excellent effects.

A very important question is the propriety or otherwise of employing the inhalation of chloroform, in order to quiet the patient sufficiently to enable him to sleep: on this matter there has been the greatest difference of opinion. My own experience of this remedy may be summed up as follows:—In the first place, I have known from personal friends of two cases (and many others have been recorded) in which the patient died suddenly, from cardiac palsy, while the inhalation was proceeding. Secondly, I do not believe, though I have frequently tried it, that the action of small doses of a weak atmosphere of chloroform (such as would be free from the danger of producing cardiac palsy) is sufficient to induce sleep, or even to greatly induce the patient's agitation, in the majority of cases. And lastly, remembering how few persons possess a high degree of skill in exactly graduating the dose of chloroform-vapor, it appears undesirable that it should come into general use in delirium tremens. For it is certain that the evil effects of a narcotic depression of the heart's action are much more serious in the case of this disease than of many other complaints. Given internally, in doses of twenty to thirty minims (or an equivalent amount of chloric ether), chloroform is less dangerous, but, as far as my experience goes, not more successful. Other practitioners, however, have met with more success in its use, and some have pushed it to much larger doses; but considering that forty-five minims taken internally by a healthy man has been known to produce full anæsthesia (though this is usually too little to produce such an effect), it is not advisable to run the risk of larger doses than I have named.

In all probability another remedy, which has only lately become the subject of attention in respect to delirium tremens, will prove one of the best of all the auxiliary means for quieting nervous agitation and hastening the advent of convalescence. I refer to the bromide of potassium.

In twenty-grain doses repeated every two hours the bromide succeeds, in a large number of cases, in calming the nervous agitation and procuring really refreshing sleep; it should be pushed till as much as two drachms have been taken in consecutive doses, if sleep is not procured before; but very commonly not more than three or four doses are required. As soon as the patient wakes out of sleep the administration should be resumed. My own experience is now sufficient to assure me that this treatment is incomparably more effective as well as more safe than the use of opium. It is more especially fitted for young and vigorous patients however, and especially to those who, while preserving considerable muscular power, have so injured their nervous centres by large excesses as to induce epileptiform tendencies.

A second remedy has lately been discovered, which appears to me to exactly fill the place of an appropriate remedy for those cases for which the bromide is not suitable—I mean the hydrate of chloral. Given in twenty-grain doses repeated at an hour's interval, chloral appears to me to act in a manner superior to that of any drug which has been used in delirium tremens. It is rarely that more than three doses are required to produce calm and refreshing sleep.

A remedy which has been used with great success in many cases, and with most unfortunate results in others, is tartar emetic. The handling of this drug in delirium tremens is an extremely difficult thing, for it requires much judgment to decide whether the constitutional strength of the patient is sufficient to support its undoubtedly depressing effects. I venture to believe that the directions, so often given, to employ antimony in cases which are distinguished by "active" delirium, with a bold and threatening (instead of a timid) expression of countenance, congested conjunctivæ, &c. are quite worthless. Such symptoms afford no measure of the patient's real strength, nor are they any warrant for the use of antimony; for this remedy must be given in considerable doses, if it is to do any real good: from a quarter to half a grain should be given three or four times, at intervals of one or two hours. When a favorable effect is produced, it is always accompanied (and I believe caused) by an increased secretion from the kidneys, or by profuse sweating, by which probably the elimination of alcohol is favored. Scantiness of either or both these secretions is therefore the true

indication for antimony. But it is necessary, even when these indications exist, to form a very accurate judgment of the strength of the circulation, and this, if we trust to the finger's estimate of the radial pulse, is most difficult. Fortunately, the use of Marey's sphygmograph will enable us to form a far more correct opinion than was formerly possible on this point. The symptoms which indicate a dangerous action of tartar emetic are faintness, *cold* sweating, and intermittence or irregularity of the pulse: the latter symptom should be carefully looked for with the help of the sphygmograph, which may detect it when the finger could not. If the first dose produces even a slight irregularity of cardiac rhythm, the medicine should be at once suspended. I wish to express the decided opinion that bromide of potassium and chloral are practically the only drugs we need ever employ in delirium tremens. [It may be believed, however, that some practitioners will not give up their confidence, based on experience, in the value of *moderate* doses of opium, at least given at night, in average cases. Alcohol, also, in diminishing quantities, does seem to aid in the cure of the feebler cases. If a man has been drinking a quart of whiskey daily up to the time of his attack, a pint or a quart of ale or porter will be to him only a mild tonic beverage, aiding his digestion. The popular idea of "tapering off" is not altogether devoid of scientific as well as clinical foundation.

The prolonged *warm or hot bath* will sometimes do a great deal of good in delirium tremens. Also, in cases of very obstinate insomnia, the application of a blister to the back of the neck may have the happiest effect.—H.]

The treatment of the *complications* of delirium tremens hardly requires any special remark, except perhaps as to the complications of *pneumonia*. It is of course necessary, as a general rule, to be specially careful to avoid unnecessarily depressing treatment of affections the original cause of which is the action of a depressing narcotic poison such as alcohol: but this rule is of twofold importance in the case of pneumonia supervening in acute Alcoholism. I am satisfied that I have seen the life of a patient sacrificed by the administration of two or three consecutive quarter-grain doses of tartar emetic, under the idea that this treatment was specifically indicated by the affection of the lung. Tartar emetic, blood-letting, both general and local, purgatives, and every other depressing treatment, are to be utterly proscribed in alcoholic pneumonia, an affection which is attended with much greater debility, especially of the heart, than its superficial symptoms would appear to indicate. The sphygmograph is very useful as a test of the real condition of things.

One important branch of the treatment remains to be briefly noticed. It is in all cases most highly desirable that a skilled attendant should be procured, and in cases where the patient is at once violent and of considerable strength, two trained persons, with experience of the treatment of lunatics, should be placed in constant attendance. It is scarcely necessary to say that the utmost violence of a patient should never induce us to employ bandages or the strait-waistcoat, if it be anyhow possible to secure sufficient nursing assistance.

(*b*) The treatment of acute mania from drink is a subject which belongs properly to the department of mental disease, and

(*c*) The treatment of alcoholic melancholia is in the same position, as is also

(*d*) The treatment of oinomania.

[On the last subject, however, it may be well to advert to some principles established by experience. Many persons, habituated to excessive drinking, becoming incapable of resisting their craving for liquor while it can be obtained, can be cured only by withdrawal to a secure place at a distance from all opportunities of indulgence. *Inebriate retreats* or asylums have been established for this purpose; and those at Boston, Philadelphia, Binghamton, Chicago, and elsewhere have now been sufficiently long in existence for a fair judgment to be arrived at in regard to their utility.

About 30 per cent. of all admitted to these institutions have been reported cured, after retirement for periods varying from three months to a year or longer. Permanence of cure is, in such cases, more indeterminable than in cases of ordinary insanity, as so much depends upon the *will* of the individual. A methomaniac may be said to be cured, when he has recovered the power to continue abstinent while at full liberty, if he chooses to do so. But he may not so choose; and then his cure has been in vain, however real it has been in itself, medically or therapeutically considered.

The plan of management at such retreats is essentially like that of the best hospitals for the insane; only with less need of apartments and inclosures for confinement of their inmates. Occupation, exercise, and wholesome diversion, by aid of garden or farm work, or various arts, etc., with books, lectures, religious services, conversations, and excursions, make up the "moral treatment." Medically, the therapeutics most required will be, the use of tonics and calmatives to the nervous system. Iron, cod-liver oil, quinine, valerian, assafœtida, and the bromides (of potassium, sodium, or ammonium, at the option of the adviser) will be the only drugs likely to be called for; unless un-

usual insomnia may for a while demand the employment of hydrate of chloral at night. A nourishing diet, with abundance of animal food in most cases, will be of great importance. More free use of stimulating condiments, as mustard and pepper, will be suitable with these than with any other class of patients. Bathing, particularly the cool (with the strongest, the cold) shower bath, is also to be recommended; its effects, however, being watched carefully in each case.

Six months ought to be the shortest period of retirement. A year will be better, to promote a secure recovery. The great difficulty is to obtain this prolonged seclusion. Legislation is necessary, and to some extent has been effected in a few of the United States, providing for the committal, upon proper evidence, and in due form, of persons rendered, by methomania, incapable of taking care of themselves, to suitable institutions; precisely as the same thing is done for other varieties of insanity.

Two extreme views are still in conflict upon this subject: that of those who regard drunkenness as only and always a vice, to be visited by severe punishment and outlawry from society; and that of others, who consider it merely a form of insanity. Actually, it begins almost always as a more or less vicious excess, the culpability of which depends upon a variety of circumstances. After long indulgence, however, it becomes a psycho-physical disease; as positively as if it had its origin only in material conditions. A prominent fact, of great practical consequence, is, that the tendency to it becomes hereditary. Certain families are well-known to exhibit exceptional proclivity to habits of intemperance. Among the evils of Alcoholism, this is not one of the least; and to it may be added the very frequent predisposition of the children of drunkards to cerebro-nervous disorders, leading in many instances to their early death.—H.]

In order to give as much continuity as possible to my description of the diseases grouped under the term "Alcoholism," I have purposely avoided long digressions upon the views held by other writers, and have made comparatively few quotations of their writings. But in order that the reader may have an opportunity of comparing this article with the teachings of other modern writers, I subjoin the following list of the principal works which are now looked upon as possessing authority on this subject:—

Sutton, Tracts on Delir. Trem., &c.: London, 1813. Roesch, Papers in Ann. d'Hygiène, t. xx. 1838. Rayer, Mémoire sur le Delir. Trem.: Paris, 1819. Ware, John, Remarks on the History and Treatment of Delir. Trem.: Boston, 1831. Peddie, Dr. J., On the Pathol. of Delir. Trem. and its Treatment without Stimulants or Opiates: Edinburgh, 1854 (pamphlet). Laycock, Dr., Pathology and Treatment of Delir. Trem.: Edin. Med. Journ. vol. iv. 1858–9. Huss, Magnus, Chronische Alkohols-Krankheit (German Edit.): Leipzig, 1852. Marcet, Dr., On Chronic Alcoholic Intoxication, Second Edition: London, 1863. Carpenter, Dr., Use and Abuse of Alcoholic Liquors: London, 1850. Various papers by Dr. G. Johnson, in the *Lancet*.

VERTIGO.

By J. Spence Ramskill, M.D.

Definition.—The sensation of moving, or the appearance of moving objects, without any real existence of movement.

Description.—Vertigo may present two forms: in the one the patient complains of giddiness in himself, external objects remaining stationary; in the other external objects assume various abnormal positions: for example, articles of furniture in the room, or patterns of paper on a wall, seem to chase each other round the apartment; or, in rarer cases, the vehicles in the street appear upside down, or the pavement undulates, or feels elastic. On attempting to walk, the patient may feel himself drawn or impelled forwards, sidewards, or backwards, and he can only prevent himself obeying the impulse by a strong effort of volition. Minor degrees of disturbed balance, and the commonest sense of uncertainty of gait demand the same exercise of volition, for there is in all cases a perpetual fear of falling down or of rude contact against other persons or against surrounding objects. In slight cases Vertigo occurs only on movement; in severe ones, when at rest also, and even during sleep.

With both forms of Vertigo we occasionally find perversions of the special senses. Patients complain of mistiness of vision, of being unable to see more than half an object, or of one half being out of all proportion to the other half, of exaggeration in size of an object, of deafness, or of hyperæsthesia of the sense of hearing, the noise of passing vehicles assuming the intensity of thunder, or of metæsthesia or a perverted sense, ordinary loud sounds appearing clear, but soft and distant. In a distinct variety of Vertigo there is real deafness of one or both ears.

Associated with these functional disorders there are complaints of tinnitus aurium, a noise of pumping water, of intermittent pulsations of fluids, of the hissing of a tea-kettle, of the noise of machinery, in fact of many kinds of noises which defy and escape description; most commonly the noises are permanent, although they may vary in intensity whilst the Vertigo is intermittent, yet the noises are loudest during the vertiginous attack.

Prognosis.—It may be taken as a rule that in Vertigo unconnected with visceral disease, and in persons under the age of fifty, there is not much danger to life, nor from what is most usually dreaded, viz., paralysis. Sudden and violent attacks of an intermittent character are unusually eccentric in origin, whereas a constant sense of uncertainty in movement, and a susceptibility to the induction of giddiness from the movement of passing objects, especially if combined with a cloudiness of intelligence, or rather a want of the usual clearness, indicates usually a centric disturbance. When, however, a severe attack occurs, without any palpable cause, to a person after the climacteric has been reached, a cautious prognosis must be given, and the more so if it be associated with vomiting, or constant nausea, tingling of extremities, the sense of pins and needles in one hand or foot, or of neuralgia of a group of muscles, or of those of one limb. The just fear in such a case is the fear of impending apoplexy. A discovery of dilated heart, of valvular disease of that organ, of degeneration of kidneys, with the presence of albumen in the urine, will make the prognosis more serious still. Organic disease apart, Vertigo has been known to exist during a long life, and indeed, unless some other suggestive symptoms are superadded, it cannot be considered a dangerous disease. In fact, the longer the complaint has existed in any given case, the less dangerous it appears to be.

Etiology.—The *direct proximate* cause of all Vertigo appears to be a disordered cerebral circulation; whereby, on the one hand, the special senses convey a false impression to the sensorium, or, on the other, a faulty co-ordination of muscular action is induced.

Of *remote causes*, it seems probable that any acute disease, or any sudden perversion of function of any important viscus on the body, may cause Vertigo, either directly or by reflex action. Thus we find stomachal vertigo as the commonest of all forms of the complaint, excepting only the invasion of all, or almost all, acute inflammatory diseases, the exanthemata, &c.; next, poisoning of blood, whether by disease, as from cachæmia, excessive smoking, intoxication, or paludal poison; then organic disease of heart, of right or left side, after such disease has reached a certain point, which acts by altering the cerebral circulation in a two-fold manner. In like manner the suppression of a long-accustomed hemorrhage acts, whether it be in the form of epistaxis, bleeding from hæmorrhoids, or prolapsus ani, or from the menstrual flow ceasing too suddenly.

The rapid suppression of an extensive chronic cutaneous eruption is an acknowledged common cause, and it is explicable on the same principle.

Varieties.—The varieties of Vertigo may be practically divided into eccentric forms, or those arising from functional disorder of any viscus or viscera in the body; or centric, from organic disease in the brain itself, or by blood-poisoning. There is a third variety, important enough to demand separation from these groups. I have called it essential Vertigo. It is not associated with any other head symptoms, and there is no appearance of depraved general nutrition. It occurs mostly in persons about thirty years of age, and is a rare form of the disease, as compared with Vertigo arising from other demonstrable eccentric causes. In other respects, a patient suffering from it will declare himself in perfect health. In all the cases I have seen, the complaint has been associated with a decidedly weak heart, a feeble small pulse, and with symptoms I take to indicate a dilated right ventricle. Another characteristic may be said to be this, that it is not materially improved by remedies, unless these are accompanied by rest and freedom from anxiety of every kind.

Stomachal Vertigo. — The most common and most tractable eccentric variety arises from disorder of stomach, or of functional derangement of the liver and upper part of the alimentary canal. It often occurs suddenly in the middle of the night, or without any warning at any period of the day, and in a state of apparent robust health. From its violence it suggests the idea of imminent danger.

The following case may be taken as a type: A merchant, some three hours after breakfast, after transacting some business of an exciting character, was quietly walking from a neighboring office to his own, when he was suddenly seized with violent Vertigo. He reeled and immediately laid hold of an adjacent gas pillar; he felt sick. Resting a few minutes, he felt the giddiness subsiding, and tried to walk; but with the first step the Vertigo returned in greater violence, accompanied by a strange tightness of scalp. He asked a passer-by to assist him, and with the help of this second person managed, reeling or rolling, to reach his office, a distance of a few hundred yards. Seated in his chair, the symptoms gradually subsided; and in a few hours, after a free evacuation of the bowels, he was free from the Vertigo, but he felt weak and shaken, and complained of a heavy diffused headache. From a very careful examination these facts were elicited. The Vertigo seemed to be of both forms described at p. 690. He felt giddy in himself, and his legs were feeble, but the objects in the streets were also strange. The shop windows seemed moving forwards, passers-by were racing after each other, the ground felt to his feet uneven, billowy, as if elevated and depressed, and he felt constrained to lift his feet over the apparent elevations. Yet he was distinctly conscious of this illusion, and tried to conceal it. The headache occupied the entire head; it was not acute; it gave the sensation of weight rather than of pain; it was not more frontal than vertical and occipital in its seat. There was no discoverable disorder of stomach or of any individual viscus, and, beyond the sudden attack of diarrhœa, nothing to suggest disorder of the abdominal viscera. He attributed the attack, and probably correctly, to having eaten very heartily a breakfast of which sausages and Devonshire cream formed a part, and to a hasty and very imperfect habit of mastication. During the ensuing month this patient had five separate attacks of the same violence, but without the same disturbance of bowels, and without being able to discover any cause, most assuredly not in the matter of diet, in which he had become exceedingly careful. Yet he was completely and permanently cured by the remedies adopted for stomachal Vertigo. As a matter of fact, it is very rare to find any positive signs of stomach disorder in these cases. They are named stomachal because remedies addressed to the stomach cure, and cure readily and quickly. With respect to the kind of Vertigo experienced, it does not, exclusively, take either of the two forms; it assumes both characters in some individuals. However, it is often so entirely connected with the appearance of external objects to the patient's eye, that the internal sense of giddiness is with difficulty made manifest. Curiously enough, it is rarely that patients complain of exaltation or defect of hearing, or of tinnitus aurium, although both these complaints are very common in the chronic stomachal forms of Vertigo. The *rationale* of the symptoms would appear to stand thus: Digestion progresses satisfactorily up to a certain point, when, owing to some temporary cerebral excitement, perhaps of transacting business or of deep thought, the process is suspended, an irritation is conveyed to the bloodvessels of the brain, *viâ* the splanchnic or pneumogastric nerves, and a disorder of circulation and of brain nutrition follows, with a corresponding disorder of function of the particular parts of the brain affected. Like causes produce like effects; and, moreover, in disorders of the nervous system it seems that a perversion of function, once induced, is easily reinduced, and by slighter causes. Hence it is not surprising that, if a patient has suffered from this acute stomachal form of Vertigo once, he will be subject to recurrent attacks.

Chronic stomachal Vertigo is of very common occurrence, and one often supposed to indicate the commencement of congestion, of organic disease of brain, of minute tissue change, premonitory of softening, or of threatening apoplexy; and the treatment which has been adopted under such erroneous diagnosis has only served to render the Vertigo permanent. Patients in this form of disease do not usually complain of the common symptoms of dyspepsia. There is never any acute pain referred to the stomach after food; often there is a slight weight, a somewhat tender epigastrium, only, however, felt on deep pressure, evidences not so much of a perverted as of a slow digestion. Complaints are sometimes made of a pain radiating from the stomach to the back, to the cardiac region, or to a general undefined uneasiness about the entire epigastric region. Rarely can more than this be made out by a most careful cross-examination of the patient's stomachal symptoms. In the lower ranks of life, however, such as we find in hospital practice, we meet with all sorts of complications; but that the symptoms appertaining to the stomach are not urgent may be inferred from the fact that patients do not seek advice for their relief, but for the Vertigo, and some steadily refused to admit there could be any disorder of the stomach, when remedies addressed to that organ afterwards cured the Vertigo. Additional symptoms of functional disease of other organs are, of course, in such a class of patients common, but they are found to be independent of, and to have little influence on, the Vertigo.

[There is excellent reason for believing that disorder of the liver has a part in the production of Vertigo in some cases. It is not uncommon to find an attack of dizziness associated with a yellowish tongue, bitter taste in the morning, and yellowness of the conjunctiva. Here *cholæmia* may be inferred; or, as it is preferably designated by Dr. A. Flint, Jr., on the basis of his elaborate investigations, *cholesterœmia;* the ingredients of the bile, especially cholesterin, being present in excess in the blood, and affecting the brain with Vertigo as a symptom.—H.]

Very usual combinations of symptoms run thus: Vertigo, pyrosis, leucorrhœa. Vertigo, menorrhagia, leucorrhœa, anorexia. Vertigo, weight of the entire head; relieved after food. Vertigo, vertical headache, nausea both before and after food. Vertigo, clavus, obstinate constipation, amenorrhœa. Vertigo progressive, weakness of sight, formicatio. Vertigo, tinnitus aurium, and partial deafness. In all these combinations the collateral diseases may be cured, and yet the Vertigo remains. There are several points of interest connected with chronic stomachal Vertigo which serve as a means of diagnosis from the graver forms of it. Thus it is never associated with loss of consciousness. There are intervals of hours in which the patient is perfectly free from it. It is made worse by excitement, by long fasting, and almost always the severe attacks occur when the stomach is empty. A stimulus in the form of wine or brandy relieves it; so also does food taken in small quantity. Closing the eyes to exclude objects in motion often relieves. During the attack a steady gaze on some fixed object mitigates the intensity of the sensation of giddiness. It is right to say that closing the eyes and the steady gaze are not invariably productive of relief, although subsequent treatment may prove Vertigo to have been stomachal.

In some cases the giddiness is slight, but almost constant; then it is usually associated with tinnitus aurium. More commonly it will occur several times daily, lasting from a few minutes to an hour, varying in degree, and accompanied by a singular general heaviness of the head, and a sense of heat at the vertex, which latter becomes aggravated when the Vertigo ceases. With respect to the peculiar form of Vertigo, no special conclusions serving the purpose of diagnosis can be drawn. Almost always unevenness of the ground is spoken of, or an illusive opening of the ground under the feet. Objects race in the eye of the beholder, and the patient feels going round with the objects he looks at when confined to a limited space, as in a small bedroom.

There are two varieties of chronic stomachal Vertigo which resist ordinary treatment. I allude to those forms complicated with, in some cases caused by, changes of tissue and alterations of the structure of the minute arteries, such as are known to occur in the persons of hard drinkers, or in those who have suffered from delirium tremens, and also in those who have suffered from latent or slight and irregular gout. It is in these cases we find the rarer forms of vertiginous perception,—as, for instance, when objects in the street or in the room appear turned upside down. In such persons vertiginous perceptions and movements last for days, and are often so severe as to confine the patient to bed, incapable of the slightest movement in the upright position without assistance. Nausea and disinclination for food are the only stomachal symptoms present.

Vertigo of the Aged is often stomachal, but equally often has no reference to that organ. As years are added, arteries become atheromatous, and otherwise diseased and obstructed, the circulation in the brain becomes irregular, we may have congestion in one place and anæmia in the other—a varying condition, abundantly sufficient to explain the frequent slight attacks of Vertigo in the aged. The essential condition of the brain is always one of anæmia. (Maclagan.) It is to be remembered that the prognosis is always more or less unfavorable when Vertigo has commenced only in old age, on account of the known pathological condition of the nerve centres.

Essential Vertigo.—Some remarks have already been made on this variety. The following case will best illustrate its characteristics: A gentleman, aged thirty-four, of considerable energy of character and great bodily vigor, has for three years suffered from almost constant, for the last two years quite constant, Vertigo. He is in comfortable circumstances, and has been very free from the ordinary anxieties of life. He has led a temperate country life, and has never had syphilis, gout, or rheumatism. Excepting the Vertigo, he has enjoyed excellent health. He says the giddiness came on gradually, and was at the commencement so slight that he can hardly fix the time of its first appearance. In kind it was subjective. At first, he found himself giddy on dressing in the morning; he felt as if he had taken too much wine overnight, and his legs were weak, and his gait unsteady. After breakfast he was well. The attacks became more prolonged, and occurred at various periods in the day; and now he is rarely free from a sense of uncertainty rather than positive giddiness. Occasionally he becomes worse, and is obliged

to sit down to prevent falling. He has no confusion or muddiness of intellect, has never lost consciousness, has no complaint of headache, dyspepsia, or disorder of any other kind. After many examinations, I have not been able to discover even functional disorder in any of the abdominal viscera. He has a soft, small, compressible pulse. The impulse of the heart is not visible; the area of dulness enlarged laterally to the right; the sounds are feeble, close to the ear, and too clearly audible to the right of the sternum. This patient has undergone a variety of medical treatment in the hands of various practitioners, including strychnine, which was pushed to the verge of producing involuntary spasms in the limbs. He has tried the hydropathic treatment, has passed a season at Vichy, but has not been able to find the slightest benefit. Very careful diet has not altered his condition, but excesses of any kind make him worse. Although there is no evidence of valvular disease, yet I cannot help connecting the feeble heart, and perhaps enlarged right ventricle, with a disordered cerebral circulation, which is itself the proximate cause of the Vertigo. I have met with two cases of this kind which were apparently hereditary. The father of one of them is now seventy-one years of ag ; he suffers from spasmodic asthma, and has been the victim of Vertigo for the past thirty-five years.

Vertigo from Overwork ranks next in frequency to the stomachal variety. It occurs in young persons who are underfed as well as overworked, as in some sempstresses; in the middle-aged, who to spare diet add various irregularities, as well as in the temperate and well-fed, who are constantly subject to mental anxiety and excitement. The attacks of Vertigo are of short duration, occur at intervals of some hours or days, after prolonged exertion, or poorer diet than usual; it is only a sense of the abnormal appearance of external objects at first, and occurs only on movement; it becomes more frequent, and then assumes, in addition, the character of an internal feeling of dizziness; the recumbent position always relieves, but does not even temporarily cure it. It is often complicated with stomach disorder, as anorexia, rarely nausea, with constipation, and in the female sex with menstrual irregularities. But the simple stomachic remedies do not remove, they scarcely mitigate, the Vertigo. Patients complain of a want of clearness of intellect, an incapability of sustained mental exertion, together with occipital heaviness or headache. In the worst cases, irritability of temper, restlessness, a sense of impending evil, and more rarely insomnia, are added. Sometimes the Vertigo is so easily induced by the appearance of objects in motion, that the patients are unable to go into the streets. In such cases there are functional ailments of other organs, palpitations, and lumbar pains, accompanied by the passing of phosphates in the urine. Oxaluria is a not unfrequent complication. Indeed, there is a general lowering of vitality, a universal deprivation of nutrition, and corresponding diminution of power, of which the Vertigo is only one of the exponent symptoms. This is the form of Vertigo which most often amongst business men precedes softening of the brain. Vertigo from irritation of the auditory nerve has been noticed by my late colleague, Dr. Brown-Séquard. He mentions, in his Physiology of the Nervous System (p. 195), this result produced by injecting cold water into the ear, and also by the topical application of nitrate of silver; and he suggests that such applications act in a reflex manner on the bloodvessels, producing temporary anæmia and a disordered circulation and nutrition of the brain, resulting in the production of Vertigo.

Ménière, in 1841,[1] established before the Academy of Medicine in Paris, that certain affections of the ear produced a series of symptoms closely resembling those attending disease of the brain, as Vertigo, dulness, uncertain walk, occasional circus movement, and even falling down, accompanied also by nausea, vomiting, and syncope. He gives also a case of a young girl who, travelling one cold night in winter during menstruation, was seized with sudden and complete deafness. Her chief symptoms were continual giddiness and irrepressible vomiting, produced by the slightest movement. She died on the fifteenth day; yet no trace of disease was to be found in the brain, cerebellum, or spinal cords. The semicircular canals only exhibited traces of disease; they were filled with reddish plastic lymph. Other cases are on record, by French authors, of a similar kind, some having associated with the Vertigo dysæsthesia; that is to say, the slightest noise producing positive and even severe pain in the affected and deaf ear. It is remarkable that such cases may terminate fatally, without presenting one single symptom of feverish reaction, and without any extension of disease to the brain (Trousseau). It is well known that Vertigo in animals may be produced by puncture of the semicircular canals, as well as by wounding various parts of the base of the brain. Further information will be found in Dr. Brown-Séquard's Physiology

[1] Bulletin de l'Académie de Médecine, vol. xxvi. p. 241; Gazette Médicale, 1861, vol. xvi. pp. 88, 239, 597.

of the Nervous System, and in the works of Schiff, Flourens, Magendie, and Claude Bernard. These are, however, matters rather of physiological than medical interest. Cases of vertiginous movements arising from disease of brain are common, such as a tendency to gyrate, to fall forwards, to one side, or backwards; but we are not able in the present state of science to draw accurate conclusions as to the seat or nature of the disease, unless it be one of a group of symptoms involving paralysis, or having other special marks of disease in a particular locality. Vertigo accompanies, to a greater or less degree, almost every organic disease of the brain, and every acute affection of this organ. Its value as a sign of disease clearly depends on its association with other symptoms; and it can only be properly appreciated in connection with a study of those diseases of which it forms a minor part.

In a large number of hospital cases there is the association of Vertigo on movement with tinnitus aurium and partial deafness. The tinnitus and deafness appear first, and the Vertigo follows. I have never been able to trace anything like suddenness in the invasion of these symptoms; their accession is always gradual, and unassociated with pain in or about the ear, or with symptoms of fever. Persons in fair average health, and without any stomach or other obvious disorder, suffer most. There seems to be some mischief of a very slow kind going on, perhaps in the semicircular canals connected with the circulation, analogous to the more acute cases recorded by Ménière. The occasional value of counter-irritation, and of iodide of potassium and of small doses of mercury, confirms this view.

Treatment.—Stomachal Vertigo, in its acute and chronic forms, often yields to a very simple method of treatment. This consists in the exhibition of alkaline remedies and of alterative aperients continued steadily for some weeks, to be followed by bitters, and especially by the use of nux vomica or strychnia. The alkaline treatment is to be used after meals, so as to neutralize any formation of acid, and to excite a freer secretion of gastric juice; the tonics to enable the stomach and bowels below to perform completely their functions. [Magnesia answers an excellent purpose in acute cases.—H.] Stomachal Vertigo of the severest kind yields most readily to the influence of these remedies. At the same time food is to be taken in small quantity, to be carefully masticated, at regular periods; and, for drink, Vichy water mixed with a small quantity of brandy acts most efficiently. All kinds of malt liquors are to be forbidden, whilst general hygienic measures are to be adopted. The splash bath in the morning, early retiring to rest, sleeping on a mattress in a large airy bedroom, are great adjuvants to the treatment. Freedom from the cares and anxieties of business are not less necessary. In all varieties of Vertigo it is wise to commence the treatment as if the case were stomachal, not simply because the case may turn out one of this variety, but because stomachal disorder may complicate any variety of the malady. The chronic forms of the complaint are more difficult of cure, but the same principles apply, and the treatment must be varied according to the peculiarities of the individual case, always remembering, however, that it will be wise to attack and remove the complications which are associated with it, before making a special treatment of the Vertigo. In more obstinate forms of disease connected with tissue degeneration, intemperance, or with chronic gout, measures adapted to these several conditions will of themselves relieve the Vertigo, and prepare the way for the restoration of tone and improvement of nutrition, on which any hope of a great amelioration or cure must depend. [When the attack is attended by a yellow fur upon the back part of the tongue, with a bitter taste on rising in the morning, and a yellowish tinge of the conjunctiva, it is a matter of frequent experience to find the use of a few grains of blue mass followed by improvement. The dose need not be larger than two grains, at bedtime, for two or three nights in succession.—H.]

The Vertigo of the aged demands wine, and any plan of treatment which the case may demand must be associated with stimulants, unless (a very rare occurrence) the Vertigo be premonitory of meningitis, and is accompanied by heat of the scalp and some congestion of the conjunctiva. A most effective combination for Vertigo of the aged consists in very small doses of the bichloride of mercury, with tincture of iron and cantharides. In Vertigo from overwork, in the well-fed there are usually present restlessness, insomnia, depression of spirits, and a vague feeling of unhappiness or impending evil, for the relief of which I have found great help in bromide of ammonium, given in an effervescing form, with the addition of cascarilla. Amongst the poor, where scanty food accompanies overwork, this remedy is not of such value; we shall gain more from measures calculated directly to improve nutrition, and from slight stimulants frequently repeated. Brandy or wine, under these conditions, is a better tonic for a time than bark or quinine,

which will be found most appropriate afterwards. The solutions of the hypophosphites are also especially valuable.

Essential Vertigo is most benefited by a long course of citrate of iron and strychnia, given in an effervescing form, alternating month by month with tincture of larch and small doses of digitalis. The local application of belladonna does good, although there may be neither pain nor palpitation to suggest its use. I believe it is a direct tonic to the muscular tissue of the heart, in which respect it resembles the preparations of larch, and perhaps also of digitalis. The usual conditions of rest, freedom from care and anxiety, are, of course, as essential as in the other varieties of the disease. The treatment of Vertigo arising from grave disease of brain, from softening of its structure, from aneurism, or tumor, must be involved in the treatment of these diseases.

CHOREA.

By C. B. Radcliffe, M.D., F.R.C.P.

Chorea is the disease known as St. Vitus's dance in this country, as the dance of St. Guy in France, and as the dance of St. Weit in Germany, St. Guy being the name which is the French equivalent of St. Vitus or St. Weit. It is chiefly characterized by irregular clonic movements of the voluntary muscles, and by weakness more or less approaching to paralysis in the same parts.

1. Symptoms.—Chorea is sketched for the first time in the writings of the English father of medicine. "St. Vitus's dance," says Sydenham, "is a sort of convulsion which attacks boys and girls from the tenth year until the time they have done growing. At first it shows itself by a halting, or rather an unsteady movement of one of the legs, which the patient *drags*. Then it is seen in the hand of the same side. The patient cannot keep it a moment in the same place; whether he lay it upon his breast or any other part of the body, do what he may, it will be jerked elsewhere convulsively. If any vessel filled with drink be put into his hand, before it reaches his mouth he will exhibit a thousand gesticulations like a mountebank. He holds the cup out straight, as if to move it to his mouth, but has his hand carried elsewhere by sudden jerks. Then, perhaps, he contrives to bring it to his mouth; and if so, he will drink the liquid off at a gulp, just as if he were trying to amuse the spectators by his antics."

The symptoms of the fully developed disorder, as the following case will serve to show, are marked enough and characteristic enough.

Case.—Mary C——, aged 11, admitted into the Westminster Hospital, under the care of the writer, on the 12th of March, 1864.

She is suffering from pains in the limbs, slight feverishness, and some tenderness and fulness in the right wrist, the pains in the limbs being chiefly in the right arm. The day before, she got drenched to the skin in a shower, and was obliged to remain in her wet clothes for some time. She is a bright-faced, good-looking, exceedingly pale child, the reverse of dull and stupid in every way, never strong, but never ill, except with severe convulsions when cutting her first teeth. Her mother had four or five epileptic fits about the time of puberty.

March 14.—The pains in the limbs are better; but the tenderness and swelling of the wrist have somewhat increased in the right wrist, and extended to both the ankles. The pulse is 100, and slightly irregular; the action of the heart is a little excited, and there is a slight systolic bruit at the apex. The skin is moist, and the perspiration has a sourish smell. The appetite has gone altogether, and there is some thirst. The medicine ordered contains iodide of potassium and bicarbonate of potass.

March 20.—The fulness and tenderness of the joints have disappeared, and so have the thirst and want of appetite; but the cardiac murmur is more, rather than less, marked. Ordered to get up, and to have cod-liver oil.

March 27.—She has just been greatly frightened by seeing a patient close by die suddenly, and is now crying and sobbing bitterly. Previously to this she had been playing with another child in the ward, and was to all appearance quite well.

March 29.—A marked change has taken place since the last visit. There is now great restlessness, and impatience, and fretfulness, with curious wriggling, fidgety movements in the right arm. Her sleep has been much disturbed, and twice in the night she got up and went to the sister, crying and saying she was frightened. Four ounces of wine were ordered.

March 30.—The restlessness is much increased; but, instead of impatience and fretfulness, there is now evident dulness and listlessness. The right arm is continually jerking about, and in attempting to walk the right leg both jerks and drags. Though right-handed, the left hand is used in feeding, and on inquiry it is found that the right hand is useless for this purpose. The speech is thick. All the joints feel strangely loose. There is some difficulty in swallowing, and the food is rolled about in the mouth some time before disposing of it in the usual way. The features twitch and twist a little, but not much, the tongue is put out and kept out without difficulty, and it is not particularly unsteady. All the disordered movements are much increased by trying to be still, and during fits of crying and fretting, which fits are not uncommon. The pulse is quick and small, the hands are cold and rather damp, and there is a constant wish to huddle over the fire; the bowels are very sluggish; the urine is neutral, and rapidly becomes offensive. Ordered to have hypophosphite of soda and cod-liver oil thrice daily, and a single dose of castor oil.

March 31.—The restlessness is much increased, and the tossings and jerkings have become almost general. Standing and walking are barely possible, partly from the jerks and tossings of the limbs, but chiefly from the weakness of the right leg. The grasp of the right hand is also much weaker than that of the left. The right foot is a little more sensitive to pinching than the left. The features are almost continually being twisted into the oddest grimaces, but when at rest they are so wanting in expression as to give the idea of extreme silliness. Indeed, the expression is so changed as to make it difficult to identify the patient as the bright-faced, intelligent girl she was when admitted into the hospital. The speech is quite inarticulate, her only question or answer being "um," with a snort. Saliva dribbles from the mouth, and even food, which she can only now get by being fed, is scarcely kept from falling out of the mouth. Mastication and swallowing are both matters of much difficulty. The tongue is unsteady, but it can be put out and kept out by an effort. The pupils are dilated and sluggish—the left especially. The pulse is quick and weak, but not irregular; the hands are cold and moist. The movements are suspended during sleep, but sleep itself is only in comparatively short catches. The same medicines to be continued, with brandy and milk at short intervals, in addition four ounces of brandy being given in the twenty-four hours.

April 5.—No very material improvement. The grasp of the right hand a little stronger perhaps, and the right leg dragging and jerking not quite so much.

April 14.—A marked improvement. The gait is much more firm and steady; the features are less vacant and less discomposed; the sleep is comparatively sound; the appetite is better; the hands are warmer; the pupils are now fairly sensitive and equal in size, but the speech remains inarticulate, and the child has still to be fed. No change in the treatment.

April 21.—The irregular movements of the arms and legs are nearly at an end; the features are comparatively at rest, and the expression of intelligence has returned; the speech is distinct now, but the voice is low, and the articulation slow; the gait is slouching, but there is no dragging in the right leg; the power of self-feeding has returned, though the left hand is still made use of rather than the right, and the sleep is sound and refreshing. No change in the treatment.

May 1.—Nearly well.

May 14.—Discharged well, except that there is still a systolic bruit at the apex of the heart.

In this case the salient points are, the age and sex, the movements, the paralysis, the numbness, the dulness and listlessness, the relations to rheumatism and heart-disease, the absence of fever, and the neutral urine; and the noticing of each of these points in turn will serve to bring out the general features of chorea in its ordinary form.

Age and Sex.—Sydenham states that chorea, for the most part, attacks children between the tenth and fourteenth year of their age, who have not reached the time of puberty. Sir Thomas Watson considers these limits to be too narrow, and extends them to the period of the second dentition on the one hand, and to that of puberty on the other; nay, he extends them still wider, for he states that now and then, but only exceptionally, cases occur as early as 4 or 5, and as late as 20 or 25 years of age. Up to nine years of age the two sexes appear to be equally liable; after this age females become much more liable than males, in the proportion of nearly 5 to 2.

Of 422 cases treated as out-patients at the Children's Hospital in Great Ormond

Street, and tabulated by Dr. Hillier, the numbers of each sex at different ages were as follow :—

	Males.	Females.	Total.
From 3 to 6 months . .	1	2	3
" 6 " 12 " . .	1	4	5
" 12 " 18 " . .	1	1	2
" 18 " 24 " . .	1	3	4
" 2 years to 3 years .	3	3	6
" 3 " 4 " .	6	5	11
" 4 " 5 " .	4	16	20
" 5 " 6 " .	7	23	30
" 6 " 7 " .	18	30	48
" 7 " 8 " .	17	34	51
" 8 " 9 " .	17	41	58
" 9 " 10 " .	23	57	80
" 10 " 12 " .	23	81	104
Total . .	122	300	422

At the Children's Hospital patients are not admitted above the age of 12, so that these statistics do not include cases from 12 to 15 years of age—that is, about the age of puberty; but judging from other statistics, as Dr. Hillier says, "it does not appear that the period of puberty is more prone to the disease than the period between the second dentition and puberty." Thus, of 100 cases occurring at all ages, and tabulated by the late Dr. Hughes in the Guy's Hospital Reports, 29 were between 12 and 15 years of age, 9 being males and 20 females; at 15 years of age there were 5 females and 1 male. After puberty, chorea is comparatively rare. In 96 cases, of which the statistics are given by Dr. Ogle, 19 were above 15—the ages being 2 at 16, 6 at 17, 2 at 18, 1 at 19, 2 at 20, 2 at 21, 1 at 23, 1 at 24, 1 at 26, and 1 at 43; and of these 19, 16 (including the one at 43) were females. And of 17 cases of chorea during pregnancy which proved fatal, which cases form the basis of an excellent paper by Dr. Barnes on chorea in pregnancy, the ages range between 17 and 24, with one exception, in which the age was 47.

The Movements.—These are the most characteristic feature of Chorea. They are clonic spasms, unattended by pain, and, as Dr. Hillier says, "something like the restless movements of a child put out of temper." Usually they are more marked on one side of the body than on the other, and at first they may be confined to one side. Not unfrequently they make their appearance first in one arm, then in the leg of the same side, then in the face, then in the arm and leg of the other side. They are always increased by any attempt to exercise the will, or under any emotional excitement: and they are, for the most part, put a stop to by sleep. As a rule, it is enough to see the movements to recognize at once the nature of the disorder; and some of the movements of the face are very characteristic. Still it does not do always to reckon upon finding movements which are looked upon as characteristic. Thus, in the case which has been cited as a text, the tongue was not put out with a sudden jerk after a pause, and retracted with equal suddenness, after the manner which is described as specially characteristic of Chorea; and most certainly this case is by no means exceptional in this respect.

Paralysis.—Want of muscular power is shown by the readiness with which the patients become tired, and by the slowness with which they recover from fatigue, as well as in the soft, flaccid, and wasted condition of the muscles when the disease has continued for some time. But this is not all which may be noticed, especially in those patients in which the choreic movements are confined to one side; for, in these cases, there is usually a want of power in the affected muscles which must certainly be spoken of as a slight degree of paralysis. This want, as Sydenham said, "often shows itself by a halting, or rather unsteady movement of one of the legs, which the patient *drags:*" or still more frequently, it may show itself in the helpless way in which the arm almost immediately *falls* when it is held out, for it is easy to see that this falling is a phenomenon which has much more to do with paralysis than with choreic movement.

As positive evidences of paralysis, must be reckoned the loss of speech, the loss of facial expression, the loss of the power of swallowing, the inability to use the hands for the purposes of feeding, the looseness of the joints, all of which symptoms were present in a marked degree in the case which has been given, and one or other or all of which, in a greater or less degree, are usually present in all cases. Paralysis, indeed, is seen to be a marked feature in Chorea, if only the attention is not allowed to be entirely absorbed by the contemplation of the movements; and in some extreme cases it may be so marked as to lead to incontinence or retention of urine, or to involuntary stools. Usually, also, the muscles which are most affected by the movements are those which are most paralyzed. Nor is the connection of Chorea and paralysis altogether out of order; for in many cases of paralysis properly so called, the paralyzed parts are affected by movements which, without question, are not remotely akin to those of Chorea.

Numbness.—This symptom is sufficiently marked in many cases to be detected without difficulty, especially in the parts in which the movements are most marked, but it is never as prominent a symptom as in hysteria; and the same remark applies to the opposite condition of oversensitiveness. Trousseau says that numbness, when present, is usually accompanied by tingling.

Dulness and Listlessness.—The vacancy of expression resulting from the semi-paralyzed condition of the features, which may be so extreme as to suggest the idea of idiocy, must not be taken as the gauge by which to measure the mental condition of the patient. Matters mentally are certainly not so bad as they look; still there is always more or less dulness and listlessness—dulness and listlessness rather than fretfulness, and undue excitability, as in the ordinary hysterical condition. It is to be remembered, also, that the children attacked by Chorea are commonly distinguished by vivacity and restlessness of disposition.

Rheumatism and Cardiac Diseases.—In his excellent digest of 300 cases of Chorea, occurring in Guy's Hospital, the late Dr. Hughes ascertained that, "out of 104 cases in which special inquiries were made respecting rheumatic and heart affections, there were only 15 in which the patients were both free from cardiac murmur, and had not suffered from a previous attack of rheumatism." Nor is it possible to get over this fact by imagining that the pains of the supposed rheumatism may have been simply neuralgic, and the cardiac murmur merely anæmic, for in 11 out of 14 cases of death from Chorea recorded in this paper, there were actual vegetations upon the cardiac valves. Dr. Romberg says, "The rheumatic predisposition, noted by English medical men, was rarely traceable in the cases presenting themselves to my observation;" but this opinion is not that of other German writers. Dr. West, who once had doubts as to the frequency of the connection between rheumatism and Chorea, now believes that the rheumatic diathesis is a powerful predisposing cause of Chorea. M. Rogers says, "The coincidence of Chorea and rheumatism is so common a fact, that it ought to be regarded as a pathological law, just as much as the coincidence of heart disease and rheumatism;" and again, "The child affected with rheumatism is, after a longer or shorter interval, threatened with Chorea; and the child affected with Chorea is sooner or later menaced with rheumatism." It may also, as Dr. Tuckwell points out, explain why it is that in adults rheumatism and Chorea do not go together as they do in earlier life, that in earlier life rheumatism is far more frequently complicated with heart disease. "The younger the patient," as Dr. Hillier remarks, "the more frequently is rheumatism accompanied by endocarditis." Cardiac disease is also very common in Chorea. Thus, in 37 cases of which notes were taken by Dr. Hillier, there was probably organic disease of the heart in 25, and of functional derangement in 4, whilst in 8 only was there no sign of cardiac disturbance. Heart disease, however, does not necessarily point to rheumatism in children. On the contrary, it may follow scarlet fever or measles, and in some cases it may come on without any obvious reason. But, be the cause of the heart disease what it may, heart disease is a common accompaniment of Chorea, in the refractory cases especially.

Absence of Fever.—Fever does not figure among the necessary symptoms of Chorea. In fact, Chorea is essentially a feverless malady. Not unfrequently, also, there are signs which point to a condition of circulation the very opposite to that which is met with in fever, such as coldness and clamminess of the hands, a disposition to chilblains if the weather be at all cold, pastiness or puffiness of certain parts of the skin, anæmic vascular murmurs, and the rest. In some instances, it is true, the temperature is increased; but such increase, according to my experience, is only met with in mixed cases of Chorea, where delirium is a marked feature, and where the movements point to delirium rather than to Chorea, and then only exceptionally, and therefore it may have nothing to do with the Chorea. Moreover, increase of temperature is not always a sign of fever in the ordinary sense of the word, for it is a fact, not unfrequently verified, that the temperature often rises remarkably in the moribund state, and that for some time after death the corpse may give a disagreeably *hot* sensation to the touch. Nor is an argument to the contrary to be found in the relation of Chorea to rheumatism. Chorea may occur before or after rheumatic fever, but not along with rheumatic fever. This is the plain fact. Indeed, the very connection of Chorea with rheumatism, when properly understood, may be only one other proof that Chorea is associated with a state of wanting vigor and activity in the circulation, for most assuredly a weak circulation and a lymphatic habit generally is the state of things which is likely to be present in persons who are prone to rheumatic fever. Moreover, it not unfrequently happens that the symptoms of Chorea are suspended by the accidental development of scarlet fever or some other febrile disorder, and that they return again when the state of feverishness passes off.

Neutral Urine.—In the case which serves as my text the urine was neutral, and readily becoming offensive; and so far as my experience goes, this is the case generally. In some cases, however, the urine seems to be of unusually high specific gravity, as has been shown, first of all by Dr. Walshe, and afterwards by the late

Dr. Todd and by Dr. Bence Jones. In a case of acute Chorea, of which Dr. Walshe gives the history in detail, lithates were deposited in large quantities during the first few days, then urea was found to be present in great excess, then oxalates made their appearance, and last of all there was a copious precipitation of oxalates; and in another case, given in the Clinical Lectures of Dr. Todd, the specific gravity of the urine was never below 1·019, and often as high as 1·030 or even 1·035, and as a rule urea and oxalate of lime, but especially lithates of ammonia, were present in considerable excess. The state of the urine requires to be more carefully inquired into. In two cases I found, for a short time only, some excess of urea, and a thick deposit of lithates on cooling; but this state of things soon changed, and what I noticed chiefly was the rapidity with which the urine lost its acidity, and threw down phosphates.

The case of Chorea which has served as the text for these comments is a little more marked in its symptoms than the average of cases. Usually, indeed, the speech is thick and confused, not lost, and the use of the hands is not so completely taken away. Usually, also, paralysis is a less prominent phenomenon. The symptoms are, in fact, infinitely varied; and as they are toned down on the one hand, or exaggerated on the other, Chorea may be a most trifling disorder or a very grave malady. In its most trifling form Chorea may be nothing more than a grimace, or a shrug of the shoulders, or a catch in the speech, or some other odd or awkward involuntary movement, which in many instances appears to be little more, or no more, than an unchecked bad habit. In its gravest form, on the contrary, few diseases are more distressing to witness—the patient tossing ceaselessly to and fro, unable to walk or even stand, turning, writhing, dashing about, and only kept in bed by being strapped down or fenced in; without speech, perhaps with the lips torn, chapped, and bleeding, by being, in spite of all we can do to prevent it, continually drawn into the mouth and munched between the teeth, which themselves, in some instances, are actually ground down and even forced from their sockets; with the elbows and hips and other prominent points made raw by constant rubbing against the bedding—a sight which is forced upon one, for no care can keep the bedclothes in their place—sometimes raving, and never sleeping, until death comes to the rescue. Once seen, indeed, it is not easy to forget a scene so sad as that presented by Chorea in its gravest form, a scene than which there is none sadder in the whole range of diseases, hydrophobia itself not excepted.

2. Exceptional Forms of Chorea.—Allied more or less closely to Chorea in its severest form is a disease which was first described by Dr. Dubini, of Milan, about twenty years ago, under the name of *electric chorea*. This disease seems to be peculiar to certain districts of Lombardy. Its symptoms are:—(1) Certain choreic or convulsive shocks in the limbs, repeated with a certain regularity of rhythm, persisting with scarcely any intermission for days, or even weeks, and followed by paralysis, and, it may be, atrophy of the affected parts; (2) certain tonic convulsions of great violence, affecting the muscles in which the choreic convulsions are manifested, and occurring in not unfrequent paroxysms; (3) epileptiform attacks, sometimes general, sometimes partial; (4) certain head-symptoms, such as cephalalgia, delirium, and coma. One or the other of these groups of symptoms may be predominant in different cases. Electric chorea may be either acute or chronic, and in either case its termination is almost always in death. As a rule, it begins quietly, and is in no great haste to assume its serious characters. As a rule also, a delirium, lasting for some days, and ending in coma, ushers in the fatal termination; but not unfrequently death is brought about more speedily and suddenly in an epileptic paroxysm. The electric shocks which form so conspicuous a feature in the disorder, occur very frequently, as often as thirty, sixty, or a hundred times in the minute, and they are often, if not always, accompanied by feelings of pain, tingling, or cramp in the same parts, by vertigo, and by humming or singing sounds in the ears. At first the digestive organs are but little affected, but after a time the appetite fails, and gastralgia and frequent vomiting add to the distress. Fever, indeed, is not seldom present. The mean duration of the disease is from forty to seventy days, if we except a few acute cases in which death happened in a few days with urgent cerebral symptoms.

Electric chorea was the name chosen for this disease by Dr. Dubini; *typhus convulsivo-cerebralis* was the name selected by Dr. Frua, a colleague of Dr. Dubini's in the great hospital in Milan, who saw many cases, and whose description of the disease immediately followed that of this last-named physician; and *myelitis convulsiva* was the name made use of by Dr. Hörtel, in his account of the disorder. This difference of nomenclature shows how differently various observers were struck by what they saw, and proves, at the same time, what is plain from their description, that electric chorea has not, perhaps, the strictest claim to be admitted into the category of choreic affections.

St. Vitus's dance, however, is the very

Proteus of diseases, and many strange maladies have to be passed in review before the description of all its various forms is complete.

The disease to which the name of *St. Vitus's dance* was originally given was of an epidemic character. It broke out at Strasburg in 1418, close upon the heels of the black death. It was, in fact, a fresh outbreak of a dancing epidemic called the *dance of St. John*, which made its appearance at Aix-la-Chapelle in the summer of 1374, and then spread like wildfire over the whole of Germany and the countries to the northwest. This dance of St. John appears to have been characterized chiefly by paroxysms of extravagant dancing and leaping and howling and screaming. In some cases the head was filled with ecstatic visions in which St. John was a prominent object; in others the most frantic excitement was produced by certain sights or sounds. Sometimes the dancing movements were ushered in by symptoms of an epileptiform character: usually they were accompanied and followed by the most distressing flatulency: almost always they were carried on until they came to an end from sheer want of strength. For nearly two hundred years society was disorganized by persons suffering from this demoniacal disorder, and by rogues who simulated it for sinister purposes. Dr. Hecker tells us that the feast of St. John the Baptist was always held as a day of wild revelry; and that at the time when this strange malady made its appearance, the Germans were in the habit of mixing up with this Christian ceremonial an ancient pagan usage—the kindling of the "nodfyr." It was the custom on these occasions to leap through the flames of this fire, and to consider that a year's immunity from the disease was gained in this way; and in this leaping run mad, Dr. Hecker thinks, we have the origin of the dance of St. John.

In its main characteristics the dance of St. Vitus does not appear to have differed from the dance of St. John. The difference of name was owing to this—that at the first appearance of the disease in Strasburg, the sufferers, real or pretended, were so numerous that the city authorities divided them into companies, and appointed persons whose duty it was to conduct them to the chapels of St. Vitus near Zabern and Rotenstein, as well as to protect and restrain them by the way. They were taken to these chapels in consequence of a legend, invented conveniently for the occasion, which represented that this St. Vitus, when suffering martyrdom under Diocletian, A. D. 303, had, in answer to prayer, received power to protect from the dancing mania all those who observed the day of his commemoration and fasted upon its eve. At any rate to the shrine of St. Vitus these people went, and there priests were ready to sing masses, and to perform other services fitted for the occasion; and thus the name of the disorder became changed from the dance of St. John into the dance of St. Vitus.

Attention was first prominently directed to these two dances, at the times which have been mentioned, but there is good reason to believe that they had been known a long time previously.

At the beginning of the sixteenth century, a change had taken place by which these disorders had become less unlike disorders which are now classed under the head of chorea. This is evident from the description given by Paracelsus and other competent observers. At this time these maladies were characterized by frequent fits of hysterical laughing or crying, by odd movements, and now and then by fits of dancing, but not by the howling or screaming or mental delusions or distressing flatulency of former days. In some instances, also, the propensity to dance was not irresistible. Still, now and then the disorders in question appeared in their old form, and Dr. Hecker tells us that so late as 1623 some women were in the habit of paying a yearly visit to the chapel of St. Vitus, in the territory of Ulm, in order that a dance at the altar there might save them from dancing elsewhere against their will, until the same time next year.

Almost contemporaneously with the dance of St. Vitus, a dancing malady, called *tarantism*, appeared at Apuleia, and spread from thence with great rapidity over the rest of Italy. This malady was attributed to the bite of a tarantula, or ground-spider, common in the country; but it is more probable that undue fears as to the evil consequences of the bite—fears arising easily in the gloomy and despondent temper of the times—had more to do in causing the malady than the bite itself. Those who were bitten remained dejected and stupefied, or else, becoming greatly excited, went about laughing, singing, or dancing. In any case, they were utterly unable to restrain themselves if acted upon by music of a certain kind. A bacchantic furor was excited by the first notes, and as the performance went on they would dance, and leap, and shout, and scream, until they fell down from sheer exhaustion. Some colors appear to have excited them, others to have calmed them. Some had a strong disposition to rush into the sea; many were carried away by strong sensual passions into deplorable excesses. Some, again, were tormented by the flatulent distress which was a symptom in the dance of St. John. In this malady, music was looked upon as the only remedy, and the country everywhere resounded with the merry

notes of the tarantella. The favorite instruments were the shepherd's pipe and the Turkish drum. It was supposed that the poison of the tarantula was diffused over the system by the exercise of the dancing, and expelled along with the perspiration. It was customary for numerous bands of musicians to traverse the length and breadth of the land during the summer months, and the seasons of dancing at the different places were called "the women's little carnival," "carnavaletto delle donne," for it was the women, more especially, who conducted the arrangements, and defrayed the expenses. Tarantism continued in Italy long after the dance of St. Vitus had died out in Germany; indeed, the epidemic can scarcely be said to have been at its height until the middle of the sixteenth century.

It would seem also that the *tigrêtier* or dancing mania of Abyssinia, a malady occurring most frequently in the Tigré country, is, in some respects, not unlike the ancient dances of St. Vitus and St. John. Beginning with violent fever, this malady soon turns to a lingering sickness, in which the patient becomes reduced to the last degree of emaciation and exhaustion. This sickness may continue for months, and end in death if the proper cure be not sought after. The first cure, which is also the cheapest, is one in which a priest ministers. It is a kind of water cure, with a blessing superadded. If this fail, the aid of music is appealed to, and arrangements are made for a prolonged performance. The place chosen generally is the market-place. Under the influence of the music the patient soon bestirs herself, and begins to leap and dance in the maddest manner possible, and, having begun, she goes on in the same way until the day is nearly, and the musicians altogether, spent, and then she starts off, and runs until her legs refuse to carry her any further. Then a young man who has followed her fires a gun over her head, and, striking her on the back with the flat of a broad knife, asks her name, when, if cured (she had never uttered this name during her strange illness), she repeats her Christian name. After this she is re-baptized, and considered convalescent. The account of this extraordinary affection is by Mr. Nathaniel Pearce who lived nine years in Abyssinia, who saw what he describes, and who published the story about thirty years ago.

A place in this strange category of disorders must also be conceded to those extravagant leapings and dancings which have been met with at various times among certain sects of religious enthusiasts—the jumpers of this country and America, the "convulsionnaires" in France, and the victims of "leaping ague," who some time ago startled and shocked the grave people of Scotland. These latter enthusiasts complained of pains in the head and elsewhere, and soon afterwards they began to suffer at certain periods from fits of convulsion and fits of dancing. At these times they acted in the maddest way, distorting their bodies, springing to a surprising height, or running with amazing velocity until they fell down exhausted. When confined in cottages, a favorite practice was to leap up and swing about among the beams supporting the roof. The effects of music do not appear to have been tested.

The time for a general visitation of maladies such as these would appear to have passed by, at least in this country; but there are still to be met with, now and then, isolated cases which have some claim to be included in the same category—cases distinguished by involuntary leaping, turning, or rushing backwards, forwards, or sideways. One of these, often quoted before, is recorded by Mr. Kinder Wood; and this, with two which have fallen under my own observation, may serve as illustrations.

Mr. Kinder Wood's patient was a young married woman who had suffered for some time from headache, nausea, quick involuntary movement of her eyelids, and various contortions of the limbs and trunk. The paroxysms themselves were not always of the same kind. At one time she would be violently and rapidly hurled from side to side of the chair in which she might happen to be sitting, or else, suddenly gaining her feet, she would go on jumping or stamping for a while, or she would rush round and round the room and rap with her hand each article of furniture that lay in her course. Or she would spring aloft many times in succession, and strike the ceiling with the palm of her hand, so that it became necessary to remove some nails and hooks which had done her an injury. Or she would dance upon one leg, with the foot of the other leg in her hand. These movements always began in the fingers, and the legs were not affected until the arms and trunk had been first seized upon. Noticing a rhythmical order in some of her movements, as if they were obedient to the memory of some tune, a drum and fife were procured, and the result of playing upon these instruments was, that she immediately danced up to the musicians as closely as she could get, and continued dancing until, missing the step, she suddenly came to a standstill. On another occasion a continuous roll of the drum at once put a stop to the dancing movements. Afterwards, the drum was used in this manner with the happiest results, and at the end of a week these movements may

fairly be said to have been stopped and cured in this way. Unfortunately, however, the drum and the fife were alike found to have lost the power on two subsequent occasions when the dancing recurred. These strange paroxysms were generally accompanied by some headache and nausea, and followed by a feeling of great weakness and exhaustion, but the patient was always able to go about her household duties in the interval.

A young lady, between twelve and thirteen years of age, who had suffered for about three years from a choreic practice of "making faces," and bobbing her head forwards in a curious manner, was the patient in one of my cases. About three weeks before the date of my first visit (24th June, 1857) she suddenly began to suffer from the paroxysms which have now to be described, and a few months previously she had suffered for some weeks in a similar manner. In one of these paroxysms she would sink or rise into a sitting posture, with her legs folded under her, and then her head would be agitated by a violent, alternating, semi-rotatory movement, until the hair would stream out horizontally on all sides, like the strands of a mop when twirled over the side of a vessel. Then followed a movement in which the whole body was thrown round and round by a succession of rapid vaults. In making these vaults, the hands were placed upon the floor or bed, and the arms used as a kind of leaping-pole; and except at the instant of swinging round, when the feet and legs were thrown horizontally outwards, the half-sitting, half-kneeling posture was never abandoned. The movements of alternating semi-rotation of the head, and of circumvolution of the whole body, occurred separately and without any order, and lasted from a few minutes to half an hour. At their worst the paroxysms were only separated by short intervals; and it is difficult to say whether the movements themselves or the state which followed—a state in which the patient lay panting, dripping with sweat, and exhausted to the last degree—were most distressing to witness. Paroxysms such as these occurred several times a day during the first fortnight of my attendance, and then ceased suddenly. After this the patient rapidly improved in general health, and the choreic twitchings of the muscles of the face and the bobbings of the head became much less frequent. This improvement, however, was only temporary, and at the end of three months the fits returned, though in a modified form, and much less frequently. At this time, indeed, the alternating semi-rotatory movement of the head did not return, and the movement of circumvolution was varied by other movements. Thus, instead of turning, the patient would at times make a succession of leaps in a straight line, so that it was necessary to run in order to prevent her from rushing out at the foot of her bed; and now and then, after falling back exhausted at the end of such a paroxysm, she would roll over and over sideways for three or four times. During these strange attacks there was not the least trace of stupor, and she would often complain of pains in her head, or of being excessively tired even while the muscular disturbance was at its height. In some instances after the relapse, however, her mind was in a rapt or entranced state, and now and then words escaped which showed that she was absorbed by some alarming dream or vision. At those times the eyes had a fixed stare, and the cheeks were somewhat flushed. After the paroxysm she would be for some time in an intensely nervous and excitable state, starting at the slightest noise or the gentlest touch, and now and then bobbing her head with much violence; or if the mind had been entranced while the movements were going on, this state would continue for some time, and then pass off with a succession of sighs. Ordinarily, however, the mind was perfectly clear, and the first moment of rest was occupied in complaining of the feeling of headache and fatigue from which she suffered. In the intervals, the patient was nervous and excitable, but in every respect an acute, clever, accomplished, amiable girl. At these times her principal complaint was of a dull pain across the top of the head, or of a feeling of tingling in the back and limbs. In this case, the pulse was quick and weak, the hands and feet were habitually cold, chilblains were scarcely absent in summer, anæmic sounds were audible in the heart and great vessels, the appetite was very defective, and the digestion sluggish. There were no worms or any other evidence of derangement in the alimentary canal beyond a slight disposition to tympanitic distension of the abdomen. Nor was there the slightest evidence of uterine derangement; indeed, in this point of view, the patient was a mere child. Recovery was tedious, and more than once interrupted by a relapse, but it was complete in the end; so complete that there was no relapse when menstruation was established about twelve months later.

The next case is that of a young gentleman, Mr. E——, æt. 22, who came up from the country, about six years ago, to consult me for what he considered to be epileptic attacks. These attacks he had, but he also had other attacks, for the sake of which I now refer to the case. In the first place, he had a curious pursing up of the mouth, attended with frequent shruggings of the right shoulder, and frequent

tossings out of the right leg; in the next place, he had attacks of shuddering, which were so violent as to shake things out of his hand, or to pitch him bodily out of the chair in which he might be sitting, or even out of the bed in which he might be lying; in the third place, he had what he called a "fit of turning." He had scarcely told me this story, when, after two or three shudders, as if a shock of electricity had been passed through him, he got up from the chair on which he was sitting, and began to turn slowly on his heels upon the hearthrug. He turned round and round in this way perhaps twenty times and then sat down. Before getting up from the chair he told me not to be surprised at what I saw, and begged me not to attempt to stop him. He said, moreover, that the impulse to turn was not altogether irresistible, but that he could not resist the impulse successfully without being much agitated afterwards. This gentleman had gained honors at college, and there was no reason to conclude that his mental powers were at all impaired. He had suffered for some time from vertigo, and now and then from headache, but never distressingly so. His pulse was 60, and weak, and during one of the paroxysms which I have described it fell full 10 beats, and became much weaker. I noticed, also, that the breathings were slow and embarrassed, and that he drew several long breaths in succession as soon as the paroxysm was over.

It is also customary to regard as varieties of Chorea those distressing and not very uncommon cases in which the head is affected by semirotatory, oscillatory, bowing, or bobbing movements. These movements are very varied in character and degree: they may be combined in various ways; and not unfrequently one kind changes into another in no very regular or intelligible order. The contractions giving rise to these movements may take place suddenly or gradually; very often they recur with monotonous regularity so long as the patient is awake; in some instances they may now and then be suspended for a time by a strong effort of the will, or by holding the head firmly between the hands: not unfrequently they are accompanied by muscular contractions elsewhere, especially when the patient begins to be worn out by want of sleep and annoyance, and in some degree by bodily suffering also, for, after a time, the muscles affected become very sore, especially about their insertions, and the contraction is attended with a good deal of pain.

Nor does this exhaust the list of affections which have or are supposed to have some relationship to Chorea. On the contrary, it remains to mention certain movements which are, often at least, little more than bad habits or awkward tricks, such as semi-uncontrollable grimacings, winkings, and other movements, which are sometimes spoken of as tics-*non*-douloureux. Nay, even stammering, stuttering, giggling, sneezing, and some forms of hysterical coughing, are not excluded, nor yet the convulsive shakings which are often seen in certain paralyzed parts, or the jerks and starts which are not unfrequently met with in connection with epilepsy. In fact, the term Chorea is of the widest and loosest significance; for it is scarcely too much to say that it is made to include every form of disorderly involuntary movement, partial or general, which has not altogether the specific characters of tremor proper, or convulsion proper, or spasm proper.

3. PATHOLOGY.—During the last two years Drs. Hughlings Jackson, Broadbent, Tuckwell, Ogle, Barnes, and others, have done much to elucidate the pathology of chorea; the investigations of Dr. Kirkes, made four or five years previously, serving as the starting-point to these new inquiries.

Dr. Kirkes was of opinion that "Chorea is the result of irritation produced in the nerve-centres by fine molecular particles of fibrin which are set free from an inflamed endocardium, and washed by the blood into the cavities of these centres;" but he did not venture to fix upon the precise seat of the mischief thus done in these centres. He merely pointed to the vegetations on the valves of the heart which he believed to be constantly present in fatal cases of Chorea, and to the signs of heart disease during life in these and other cases, and drew his conclusions.

Adopting this theory of embolism, Dr. Hughlings Jackson goes further than Dr. Kirkes had done, and attempts to prove that the plugging of the vessels, which he regards as the cause of Chorea, is in the nerve-tissue forming the convolutions near the corpus striatum—a part supplied by branches of the middle cerebral artery; and that the tissue is thereby not destroyed, but rendered unstable from undernutrition resulting from a diminished supply of blood. And, without doubt, the clinical evidence adduced in favor of this view is very cogent. Taking Chorea of one side of the body, hemichorea, as the simplest form of Chorea, and putting it side by side with hemiplegia, the result of embolism, good reason is found for believing that the disorder of movement and the palsy both point to the region of the corpus striatum as the seat of mischief. If this be the seat of mischief in hemiplegia, why not in hemichorea? The muscles most moved in hemichorea are those most palsied in hemiplegia. In hemichorea, as in hemiplegia, the arm, as a rule, is more affected than the leg. In right hemichorea, as in right hemiplegia,

the speech is generally very much affected. Again, hemichorea is always more or less mixed up with, and sometimes ends in, hemiplegia; and, on the other hand, hemiplegia from various causes is not unfrequently attended by chorea, or movements of some kind or other. The fact that the face is involved in chorea shows that the seat of the disorder must be above the spinal cord. The facts which have been instanced point to the convolutions near the corpus striatum, rather than in any other part of the brain, as the part affected. In this way Dr. Jackson reasons, and reasons to good purpose; for most assuredly the difficulties which beset any attempt to localize the choreic lesion in the nerve-centres are not a little simplified by thus insisting upon the clinical relations between hemichorea and hemiplegia, as a ground for believing that the region of the corpus striatum is the part affected in both disorders.

Dr. Broadbent also accepts the same doctrine of embolism up to a certain point, and, not knowing that any one had gone before him, travels by the same way to the same conclusion as that which Dr. Hughlings Jackson had arrived at only just before. He is, however, inclined to localize the seat of the cerebral mischief in chorea *in*, rather than near, the sensori-motor ganglia, and he looks upon embolism of the fine vessels of these ganglia only as the chief cause of Chorea. As with paralysis, so with Chorea, he believes that the symptoms point to the *seat* of the mischief, not to its *nature;* and that, besides embolism, hemorrhage, softening, irritation, and other causes, may figure among the causes of Chorea; the difference between the mischief causing Chorea and that causing paralysis being this—that in the one case it is impairment of function only, and in the other case abolition of function—a view which is also insisted upon by Dr. Hughlings Jackson. In addition to embolism as a cause of Chorea, Dr. Broadbent instances local innutrition, reflex action from peripheral irritation, and direct action upon the sensori-motor ganglia, from shock, &c. He shifts his ground, in fact, considerably, from embolism as a cause, but at the same time he refers to the discovery by Dr. Bastian of the proximate cause of the delirium of febrile diseases in embolism by altered and cohering white blood-corpuscles, as bringing some of the causes which might be referred vaguely to local innutrition or blood disease within the category of the cases caused by embolism.

Much evidence to the same effect, at least so far as showing that the condition of the heart is favorable to embolism, is also supplied by Drs. Ogle and Tuckwell, though Dr. Ogle himself is not in favor of this theory of embolism.

Dr. Ogle reports sixteen cases of fatal Chorea occurring at St. George's Hospital since 1841, and all taken from the hospital books. In ten of these, fibrinous bands were present on the cardiac valves; and in eight of these ten, their seat was on the auricular surface of the mitral valves. In another case also, not included in these ten, the carotid artery was plugged up. Dr. Tuckwell has witnessed five fatal cases, in all of which the valves of the heart were affected in the same way—a way so constant as to lead him to speak of hearts thus altered as *choreic* hearts, the peculiarity being in the presence on the auricular aspect of the mitral valves, along the free margin of each cusp, of a line of numerous, bright, clustering, warty vegetations, some as large as a pin's head, others so minute as to be just visible to the naked eye, and that only in a certain light, but shining like little white beads when slightly magnified, which bodies might be easily detached by lightly brushing the part with a camel-hair brush or with the tip of the finger, and some of which had been detached, and were clinging to the chordæ tendineæ of the valve, ready to pass into the circulation at the next contraction of the heart. In order to find these evidences of valvular disease, as Dr. Tuckwell points out, it is not enough to open the heart in the ordinary manner, and look at the mitral valve from below: for, looked at in this way, the valve may appear quite healthy. It is necessary to slit up the left auricle and look at the valve from above; and because this is not always done, no doubt the disease has been often overlooked when it only wanted looking for to be detected. [Dr. Dickinson has reported autopsies of 22 cases of fatal Chorea, in 17 of which recent vegetations were found on one or more of the valves of the heart. —H.]

The appearances met with, after death, in the nervous system, are more difficult to explain in accordance with what has been already said. In a few instances only do they tend to confirm the notion of the choreic lesion being caused by embolism, and localized in the region of the sensori-motor ganglia. In one of two fatal cases, of which Dr. Tuckwell gives the details, mania was the most prominent symptom during life, and the post-mortem examination discovered an extensive red softening of the convolutions—"a consequence of embolism;" and in the other, in which there was no mania or delirium, there was no superficial softening of the gray matter, but a deeper seated softening of the right, and in a less degree of the left hemisphere, in that part which lies outside and beneath the sensori-motor ganglia, without any recognizable evidence of embolism, the corpus striatum

and optic thalamus lying, as it were, embedded in a nest of softened cerebral matter. Dr. Tuckwell also cites a case of embolic hemiplegia, with choreic movements supervening upon the paralysis, in which the same parts were found softened after death.

"In the beginning of May, 1860, a girl, aged 19, was admitted into St. Bartholomew's Hospital, under Dr. Burrows, with complete hemiplegia of the left side, and a loud musical systolic murmur at the apex of the heart. The diagnosis made was 'plugging of some cerebral vessel by fibrin detached from a diseased mitral valve.' Within a fortnight from the time of her admission, while the paralysis of the left side was steadily improving, the right side became paralyzed, and both the right and left sides became affected with well-marked Chorea. On May 27, double pneumonia, involving the right more than the left lung, came on; and she died on May 29. The post-mortem examination was made by Dr. Harris, and I took down the following from his dictation:—The brain was found healthy at all points except at the under part of the middle lobe in either hemisphere, where there was a well-marked patch of softening, about as large as a hen's egg, larger in the left than in the right hemisphere. The brain tissue at the softened part had a reddish-yellow tinge, more marked on the left than on the right side. The middle cerebral artery, on either side, at about its third division, was found obstructed, at an angle of bifurcation, by a firm fibrinous deposit. The heart had its left ventricle hypertrophied, and the auricular surface of its mitral valve studded with numerous warty growths. The right lung was partly in the first, partly in the second stage of pneumonia. The upper lobe of the left lung was in the first stage of pneumonia. There were no deposits in the liver, spleen, or kidneys."

Dr. Bastian also refers to a fatal case of bilateral Chorea, with delirium, of which he promises to give the details presently, in which embolisms, consisting of masses of irregular shape and size, and evidently made up of an agglomeration of white blood-corpuscles, had led to ruptures and obliterations of small vessels throughout the corpora striata and the course of the middle cerebral arteries generally. So far the appearances in the nervous system after death from Chorea agree with the premises, but not so what remains to be stated. Thus, in fourteen cases of deaths from chorea, collected by the late Dr. Hughes, the brain was quite healthy in four, and only congested in three cases, while of the remaining seven cases the particulars are these:—In the first, serous effusion beneath the arachnoid and into the ventricles, slight effusion of blood beneath the right cerebral hemisphere, softened brain; in the second, arachnoid opaque, brain dark and soft; in the third, pia mater watery, cineritious matter, red, soft, and partially adherent; in the fourth, brain soft and vascular, much fluid in ventricles; in the fifth, arachnoid opaque in parts, cerebrum vascular, left thalamus rather soft; in the sixth, dura mater adherent very firmly to calvarium, more opaque than natural, cerebral vessels turgid; in the seventh, blood effused into arachnoid, fornix and edge of third ventricle soft, red, and tumid, brain softened. In the same fourteen cases, the spinal column was not opened in six: of the remaining eight, the cord and its membranes were quite healthy in three, and only a little congested in one; and of the four others, the particulars are these:—In the first, fresh adhesions of the arachnoid, gray matter dark; in the second, vessels rather large and numerous, serous surfaces opaque, old adhesions of the membranes, especially posteriorly; in the third, medulla slightly softened, rachidian fluid opaque, yellow, and densely coagulable by heat; in the fourth, softening of the cord opposite the fourth and fifth dorsal vertebræ. Nor is the information supplied by Dr. Ogle in the paper already referred to less vague, for the sum of it is only this—that the brain or cord, one or both, were more or less congested in six cases, that the central parts of the brain were much softened in one, and that the cord was softened in one and otherwise affected in another. Very possibly a different result might have been arrived at if these cases had been examined with special reference to the condition of the sensori-motor ganglia, especially if more men like Dr. Bastian were concerned in the investigation. As it is, all that can be said is that the facts of morbid anatomy do not supply *much* support to the notion that the choreic lesion is caused by embolism and localized in the sensori-motor ganglia. There is nothing in the facts to contradict the notion that the choreic lesion may *begin* in the sensori-motor ganglia; there is something to show that all parts of the nervous centres may become affected in the end—the cord as well as the brain. No doubt, as Dr. Reynolds remarks, the symptoms of Chorea point from, rather than to, the cord. No doubt the spasm should be tonic rather than clonic, as it is in Chorea, if the cord were specially at fault in chorea. No doubt the cessation of the spasm of chorea in sleep points to the brain, which does sleep, away from the cord, which does not sleep. Nor are these the only reasons which point away from the cord; but the fact remains that after death from Chorea the cord is often found to be affected, and

also that a particular part of the cord, the posterior columns, is especially affected in a disease which agrees with Chorea in this, that it is marked by inco-ordination of movement, namely, locomotor ataxy.

[The order of succession of the symptoms in some cases, where Chorea follows articular rheumatism, makes it not improbable that a subacute rheumatic meningitis may occur, bringing on the attack of Chorea by cerebro-spinal irritation. More acute and violent manifestations of cerebral rheumatism are, although uncommon, familiar to most physicians.—H.]

Neither do the teachings of experimental physiology help much towards exactly localizing the particular mischief which operates in the exceptional cases of Chorea. These teachings show that movements of a rotatory character may originate in various parts of the nervous system—in the thalami optici, corpora quadrigemina, crura cerebri, pons Varolii, crura cerebelli, in certain parts of the medulla oblongata, and also in the upper portion of the spinal cord; that choreic agitation may be caused by slicing away the cerebellum, and by puncturing one of the corpora quadrigemina; that the removal of the encephalon in front of the thalami optici *may* result in an impulse to go forwards; and that a deep wound in the cerebellum *may* be attended by an impulse to go backward. "The parts injured," says Dr. Brown-Séquard, "seem to be quite different from those employed in the transmission of sensitive impressions or of the ideas of the will to the muscles, at least in the medulla oblongata and pons Varolii. They constitute a very large proportion of these two organs, perhaps three-fourths of the first one: they are placed chiefly in the lateral and posterior columns of these organs: they seem to contain most of the vaso-motor nerves, by which, directly, or through a reflex action, they may act on other parts of the nervous system; and they can give rise to spasm on the *same* side of the body—a fact which shows that many of their fibres do not decussate." Moreover, another lesson to be learnt from experimental physiology is, that rotatory movements may have their starting-point in a *nerve* at a distance from the nervous centres. Thus, Dr. Brown-Séquard has made a rabbit turn or roll towards the injured side by puncturing the expansion of the auditory nerve within the ear; and M. Flourens has produced similar movements in a pigeon, by simply tying a bandage over one of its eyes. It would seem, indeed, as if the parts of the nervous centres which are concerned in the production of choreic movements may be affected from a distance by *reflex* action. Nor is this to be wondered at, seeing that there are facts without number which show that distant parts of the nervous system are continually being affected by reflex action, and that the varied consequences of a particular injury are only to be accounted for by supposing many of them to be reflex phonomena. Indeed, there is no lack of instances to show that any part of the nervous system may act on any other part, and the exact localization of many disorders of the system is a difficult if not hopeless task, for the simple reason that any given lesion in any part may be attended by a wide range of symptoms depending upon sympathetic disorder set up in other parts.

At first sight it may be supposed that the pathological facts which have been given, favor the idea that inflammation of the brain or spinal cord, one or both, has to do with the production of Chorea; but a moment's reflection is sufficient to dispose of this supposition. It is plain, in fact, that this inflammation cannot be regarded as essential to the Chorea, for in some of the cases there are no traces of inflammation. This inference is inevitable. Moreover, the clinical history of these very inflammations, apart from Chorea, leads to the same conclusion, for the symptoms of these inflammations are not those of Chorea. There are also on record many cases in which inflammation in other parts, as in the lungs, has been developed in the course of Chorea, and in which the choreic symptoms have been suspended during the inflammation. The case, indeed, is one which seems to justify the inference that the Chorea is connected, not with inflammation, but with a state which may issue in inflammation. The case is one in which all seems to be explained if it be supposed that the Chorea is connected with *irritation*, not with inflammation—with the state, that is to say, which precedes inflammation always, and which may or may not issue in inflammation. In this way, then, the cases which have been given, in which the traces of inflammation are absent after death, must be looked upon as cases in which the Chorea proved fatal before irritation issued in inflammation, and the cases in which the signs of inflammation were present, as cases in which before death the irritation had issued in inflammation. Nor is there anything contradictory to this conclusion in the clinical history of the cases of which the post-mortem appearances were those of inflammation, for there is nothing in this history to show that this inflammation may not have occurred *very shortly* before death, and that the true choreic symptoms may not have disappeared as the true symptoms of inflammation made their appearance.

And, certainly, there is little reason for connecting Chorea with fever. On

the contrary, there appear to be good grounds for believing that the maxim of Hippocrates holds good here as in other cases—*febris accedens solvit spasmos*. At any rate, there are many cases on record of measles, scarlet fever, rheumatic fever, or some other fever, being developed during the course of Chorea, and in which the choreic symptoms have been suspended during the fever. I have met with seven such cases. Indeed, so far as I have had the opportunity of judging, the constant rule appears to be, that the Chorea is aggravated in the initial stage of the fever—that is, in the cold stage, or stage of irritation—and suspended more or less completely when the stage of reaction, or hot stage, is established; and that, in relation to rheumatic fever, the place of Chorea is either before the fever (often a long time before) or after the fever (often a long time after).

The history of Chorea in relation to inflammation and fever, indeed, so far as I can see, would seem to be like that of disorders which are more or less akin to Chorea—namely, tremor,[1] convulsion, and spasm in their various forms. For what is this history? In an attack of common trembling, the circulation is greatly depressed, and the pulse does not recover itself until this paroxysm is over; and in paralysis agitans the paleness and chilliness of the surface of the body, and the decided relief afforded by wine, tell a similar story. In delirium tremens the cold perspirations, the quick and fluttering pulse, the moist and creamy tongue, are all significant facts. The initial rigor of fever, moreover, is coincident with defective surface-warmth, miserable pulse, sunken countenance, blueness of nails, cutis anserina, and other signs of vascular collapse, and subsultus goes along with the most utter prostration of the powers of the circulation. And in mercurial tremor, an inference as to the real state of the circulation may be drawn from the fact that the subjects of this disorder are not unfrequently in the habit of resorting to gin and other stimulants for the purpose of making themselves steady. There even appears to be something uncongenial between tremor and an excited state of the circulation. The state of the circulation in the delirium of which trembling is the distinctive feature—delirium tremens—is quite different from the state of the circulation in the delirium in which there is no trembling. In the latter case—in the delirium of acute meningitis, for example—the skin, especially the skin of the head, is hot and dry, not cold and damp; the pulse is hard and strong, not weak and fluttering; the tongue is parched and brown, not moist and creamy—the condition is one, in fact, of high fever, and not one which, as in delirium tremens, is more akin to collapse than to high fever. And it is not less certainly a fact, that delirium tremens loses its characteristic trembling if acute head-symptoms and high fever make their appearance in the course of the disorder. Moreover, it must be borne in mind, as pointing to the same conclusion, that the initial rigors of fever disappear *pari passu* with the establishment of the vascular reaction of the hot stage, and that they return in the form of subsultus when the state of reaction has died out, and the patient is left utterly prostrate and helpless.

Again, there is reason to believe that spasm is associated frequently with a depressed state of circulation. During the attack of catalepsy, the appearance of the patient is very like that of a corpse, and it may even be necessary to apply the ear to the chest to know of a certainty that the heart continues to beat. In tetanus, as all are agreed, there is no fever; and in the tetanus arising from strychnia, as Dr. Harley has shown, one effect of the poison is to prevent the blood from becoming properly oxygenated. In cholera the cramps are coincident with a state of almost pulseless collapse. In hydrophobia the condition of the circulation is as far removed from feverish excitement as in tetanus. And, certainly, a similar inference may be drawn with respect to the state of the circulation in cramp in the leg and elsewhere, for these seizures are met with, not in strong persons, but in those who are weakly, and especially in those who are elderly as well as weakly. Nay, there is reason to believe that spasm in its various forms is antagonized rather than favored by an excited state of the circulation. In tetanus it appears to be the rule for the spasm to gain ground almost in exact proportion to the degree in which the pulse loses its true power. In hydrophobia it would seem as if the same law held good, for on analyzing the histories of a considerable number of cases, I find that there was less agitation, less convulsion, less spasm, where the circulation was less depressed than it is in the ordinary run of cases. Nor is a different conclusion to be drawn from the history of spasm as it is set forth in whooping-cough. For what is the fact? The fact is simply this—that the whoop, which is the audible sign of the spasm, does not make its appearance until the febrile or catarrhal stage has passed off; that it disappears if pneumonia, bronchitis, or any other inflammation be developed in the course of the malady; and that it returns when the inflammation has departed. And most assuredly there is no clinical evidence to show that convulsion is asso-

[1 Choreic movement is, however, a phenomenon very different from tremor.—H.]

ciated with an over-active condition of the circulation. In the fevers of infancy and early childhood, especially in the exanthematous forms of these disorders, convulsion not unfrequently occupies the place which belongs to rigor in the fevers of youth and riper years. It occurs in the cold stage of the fever, when the powers of the circulation are greatly depressed in every way; and it is confined to this stage, except in those cases in which there are certain brain and kidney complications, when it may also take the place of subsultus, or rather of death itself, for when it occurs at this time the patient has all but ceased to strive in the "struggle called living." Nay, I am even disposed to think that there is something altogether uncongenial between convulsion and the hot stage of the sympathetic fever connected with inflammation, for it is a fact not unfrequently verified that fits of common epilepsy are often suspended for the time by causes which give rise to a state of sympathetic fever in the system. For example, I can call to mind four or five cases of epilepsy, in which high sympathetic fever was set up by a burn or other injury inflicted during a fit, and in which fits, which were of daily occurrence before the accident, and which recurred with the same degree of frequency afterwards, were altogether suspended so long as the fever continued. Nor is a contrary conclusion to be deduced from the history of the convulsion connected with teething, with worms, or with any other condition in which what is called "morbid irritability" is the prominent characteristic; for it is found, not only that fever is almost entirely foreign to the state of "morbid irritability," but also that convulsion, when it does occur, is associated with seasons of decided vascular depression. In a word, the result of bed-side study has been to convince me that the true place of convulsion, in connection with any form of febrile disorder, is in the cold stage before the hot stage, or in the cold stage after the hot stage, and not in the hot stage itself; that, in fact, there is something uncongenial between convulsion and an excited state of the circulation. And so also with ordinary epilepsy, the general history of the disorder appears to be that the convulsion is antagonized by an excited state of the circulation rather than favored by it.

As it seems to me, then, there is nothing unintelligible in the fact that Chorea, instead of being connected with a state of inflammation and fever, is connected with a state which must be looked upon as the very opposite of inflammation and fever. As it seems to me, indeed, there is nothing in this part of the history of Chorea but what was to be expected from the history of tremor, convulsion, and spasm in their various forms. Nay more, the antagonism between chorea and inflammation or fever is, as it seems to me, nothing but what is necessitated by the physiological as well as by the pathological history of muscular contraction. But these are topics upon which I may not dilate further in this place, and I therefore bring my remarks under the present head to a close, by simply saying, that those who care to know more of what I think on this subject will find the latest statement of my views in a book about to be published under the title of "The Dynamics of Nerve and Muscle."

4. CAUSES.—"The patients who suffer from Chorea," says Dr. Hillier, "are very impressible and emotional, and very liable to derangements of the nervous system." Often too, as in the case which has been given as an example of the disease, where there were fits at the time of teething, they have suffered from some other decided disorder of the nervous system, and quite as frequently; as also is illustrated in the case in question, where the mother had epilepsy at the time of puberty, an inherited disposition to disorder of the nervous system may be suspected. Thus, out of 48 cases in which I have inquired into the family and personal history of the patients, I find 27 cases in which father or mother, or brother or sister, had been, or was, subject to epilepsy, paralysis, apoplexy, hysteria, Chorea, or insanity; and 11 in which the patient had had infantile convulsions, Chorea, or epilepsy. As in hysteria, it might be expected that sympathy and imitation would figure among the causes of Chorea; but this anticipation does not appear to be borne out by the facts. "The disease," says Dr. Hillier, "is never induced by the assemblage of several choreic patients in a ward of children, nor does it appear that the symptoms are in any way aggravated by mutual association." Fright, on the other hand, is without question a frequent cause; it is distinctly stated to be the exciting cause in 31 out of 56 cases collected by Dufossé and Bird, in 34 out of 100 cases reported by Hughes, in 9 out of 31, and in 9 out of 38; related by Dr. Peacock and Dr. Hillier respectively. Still it is certain that in many of these very cases the *début* of the choreic symptoms is so long deferred as to make it difficult to believe that fright has had very much to do as an exciting cause, and not unfrequently also a doubt as to the operation of any sudden exciting cause is suggested by the very slow development of the choreic symptoms. Indeed, when the matter is strictly inquired into, but few cases are to be met with in which the patient was at once suddenly sent into a state of Chorea from any cause, emotional

or other. Some special cause of *irritation* may also be suspected in some cases, as worms in the intestines, a fœtus in the uterus, and especially unnatural irritation of the sexual organs; but here again the evidence is less conclusive than it might be supposed to be. One or two cases are on record in which choreic symptoms have ceased almost abruptly on the expulsion of a tape-worm by a vermifuge. Chorea in pregnancy has also been found to cease on delivery. Still it may be questioned whether Chorea in pregnancy is always true Chorea, and whether an altered state of the blood rather than irritation may not be the true cause in those cases where there is no good reason to be in doubt as to the nature of the disease; for in pregnancy there is a hyperplastic state of the blood which may favor embolism, from the direct deposit of white corpuscles in the minute vessels, as is pointed out by Dr. Bastian, if not by the floating into these vessels of minute vegetations detached from the cardiac valves. In 5 out of 16 cases of fatal Chorea reported by Dr. Ogle there were "proofs of congestion and other graver lesions of the genital system;" and most certainly I have in not a few cases found reason to know or suspect the existence of practices which might lay the foundation of such congestion, and give rise to any amount of irritation, and this too in cases where the age of the patient might be supposed to be a sufficient contradiction to the notion. Again, the causes of Chorea and rheumatism would seem to be closely allied, if not identical. "Chorea," says Dr. Tuckwell, "is a disease which is common among the poor and ill-nourished, rare among the rich and well-favored; and exactly the same holds for rheumatism." Chorea also resembles rheumatism in being more common in damp and cold than in warm and dry climates. At the same time, season does not seem to influence the development of Chorea very much. Thus, in 27 cases given by Dr. Hillier, 13 occurred in the six winter months and 14 in the six summer months—viz., 2 in January, 5 in February, none in March, 3 in April, 2 in May, 4 in June, 2 in July, 0 in August, 2 in September, 1 in October, 3 in November, and 2 in December.

5. Diagnosis.—In a well-marked form, Chorea cannot well be confounded with other maladies. It does not even suggest the idea of hysteria, and therefore there need be no confusion on this score. Nor need Chorea be confounded with inflammatory diseases of the brain and spinal cord. In the more aggravated cases there is, I believe, a tendency to run on into one or other of these diseases, and the moment of transition may not always be easily definable: but, as a rule, the accession of the new disease will be indicated, not by the aggravation, but by the cessation of the choreic symptoms proper, and the substitution for them of delirium, pain in the head, convulsion, paralysis, numbness, pain in the back increased by movement, and others, in groupings which will leave no doubt as to what their true meaning must be. I have seen two cases of Chorea in children which ended in cerebral meningitis, and in which the choreic movements ceased when convulsion and delirium made their appearance; and I have seen one case of severe Chorea in a youth which ended in inflammatory disorganization of a considerable portion of the spinal cord, and in which the choreic symptoms did not continue after the development of the numbness, paralysis, and other symptoms of myelitis. Indeed, it may be stated broadly that the symptoms of Chorea are *not* the symptoms of acute inflammatory affections of the substance of the brain and spinal cord, or of their meninges. Delirium is reckoned among the symptoms of acute Chorea in some cases. But I am very much disposed to believe that the case has changed from Chorea to some other disease of the brain, not always inflammatory, of course, when delirium makes its appearance; and that this case will be spoken of, not as Chorea, but as a consequence of Chorea, when more is known of the diagnosis of diseases of the nervous system, and when greater exactness of nomenclature is attained to. Nor need Chorea be confounded with any chronic affection of the membranes or substance of the brain or cord, the points of difference to be noted being always more numerous than the points of resemblance. In locomotor ataxy the disorderly movements are mostly in the legs, and in these parts only when attempts are made to stand or walk; whereas in Chorea the movements, which are chiefly in the upper part of the body, though aggravated by any attempt to use the will, continue at all times with little or no intermission so long as the patient is awake. In Chorea also there are none of the severe neuralgic pains which are so characteristic of locomotor ataxy. Again, Chorea is emphatically what locomotor ataxy is not—a disease of childhood. The history of Chorea is also sufficiently distinct from that of the jerks and shocks attending epilepsy and paralysis agitans. In the former case I have sometimes seen movements so repeated, and so like those of Chorea, that a mistake might have been possible if the fact of the fits had been overlooked; but usually the movements attending epilepsy are jerks and shocks, separated afterwards by wide intervals and extending over a long period, and not at all choreic in themselves. The history of paralysis agitans,

let alone the age of the patient, is sufficiently distinctive. And so is the history of those cases of paralysis complicated with choreic movements, in which paralysis is the primary disorder, and which may be spoken of as a sort of local paralysis agitans; for this history points to previous brain disease in a way not to be mistaken, and to a time of life which is in itself inconsistent with the idea of Chorea. In fact, it is scarcely possible to confound ordinary Chorea with any disorder of the bain or cord, acute or chronic, if only moderate care be used in the diagnosis. And this is all that need be said under this head; for with respect to the exceptional forms of Chorea, general or partial, it is more than probable that they ought to be taken out of the category of Chorea and placed with hysteria, or referred to some special disease or disorder of the brain or cord.

6. Prognosis.—The natural tendency of Chorea is, without doubt, towards recovery. Sooner or later, as a rule, the patient gets well; and too often, as it would seem, the treatment deserves very little credit for this result. The mean time occupied in recovery, according to Sée, is 69 days, or a trifle under 10 weeks; and Dr. Hillier, basing his calculations on 30 cases treated by himself, arrives at the same conclusion, the longest time occupied by these cases in recovery being 28 weeks, the shortest two weeks, and the mean 10 weeks. The disposition to relapse is considerable, and usually primary attacks are more protracted than relapses. Now and then, in the proportion of six per cent. according to Sée, Chorea takes an acute form, and is rapidly fatal. Local chorea, as exhibited in the muscles of the neck at least, is notoriously obstinate; and instead of wearing itself out, it is more likely to go on year after year until the patient is worn out by it. How far the occurrence of Chorea implies a tendency to other disorders of the nervous system, especially to epilepsy, is a question which has not yet been fully entertained, and I cannot supply an answer from actual statistics. But this I may say—that I have *frequently* met with epileptic patients who were choreic at one period of their life, and that the impression left on my mind from what I have seen is, that the chances of Chorea being followed, sooner or later, by some other disorder of the nervous system are too much made light of.

7. Treatment.—Nothing can be more perplexing than the statements made by various authorities respecting the efficacy of remedial agents in the treatment of Chorea. Few voices, it is true, are now raised in favor of the old-fashioned antiphlogistic ways of treatment, in which bloodletting and purgatives and low diet figured so conspicuously; but beyond this all that is uttered seems to be dictated by the spirit of contradiction or scepticism. Indeed, so little unanimity of opinion is there respecting the treatment which ought to be pursued in Chorea, that the only course is for each one to glance at the principal remedial agents recommended, to weigh the statements made respecting them as well as he can, and to take upon himself the responsibility of deciding upon his own course of action.

Sir Thomas Watson considers that the most suitable medicine in cases of Chorea is, as a rule, some preparation of *iron;* and this verdict is accepted by the great majority of English practitioners in medicine. Dr. Elliotson says that he cured forty cases in succession by the use of full doses of sesquioxide of iron, the time spent in the cure varying from six to eight weeks. I have not used iron much in the treatment of Chorea, and I have not seen it used to any great extent by others. Not unfrequently, however, I have known a person using this agent go on for a while with it, and then discontinue its use, apparently as if he were not satisfied that all the good was being done which ought to be done. Of the several preparations of iron which have been recommended, I am disposed to believe most in the syrup of the iodide, the use of which was first suggested by the late Dr. Barlow, of Guy's Hospital. I have certainly seen several cases in which the use of this preparation seemed to be followed by unequivocal evidence of improvement; but, on reflection, I find it difficult to refer this change for the better to the iron altogether, or even to the iron chiefly. On the contrary, I am disposed to think that the iodine is entitled to a fair share of the credit, to say the least; and that the iodine in the doses usually given is stimulating or restorative in its action rather than alterative, in the sense in which it is usually supposed to be alterative. I fancy, also, that there is a growing doubt as to the efficacy of iron in cases of Chorea, and that many would now be disposed to agree with the late Dr. Hughes, who says only that iron has been administered in numbers of the cases of Chorea recorded in his admirable report, and that it has "sometimes succeeded where zinc has failed."

Zinc is given very largely in the treatment of Chorea. In the cases of Chorea occurring in Guy's Hospital, the late Dr. Hughes says that "zinc in the form of sulphate has been the most frequently employed as a remedy, and has generally been most successful;" and forty-five cures out of sixty-three cases, or five in seven, are credited by him to this medicine. Dr.

Barlow says, "In ordinary cases, the exhibition of purgatives to keep the bowels freely open, and the sulphate of zinc in doses gradually increased from a grain to even fifteen or twenty grains, or even more, will effect a cure. When, however, the sulphate has been used in these large doses, its sudden discontinuance seems to be felt by the system, and a return of the symptoms ensues. The best rule, therefore, for its exhibition is as follows:—"the bowels being kept open, the sulphate of zinc should be given in doses commencing with a grain three times a day, and in the case of a child about twelve years old the quantity should be increased by the addition of a grain daily, until the medicine causes sickness, or there is an obvious diminution of the choreal movements. In the former case the dose should be diminished by at least one-half, and so continued for several days, with a view to establishing a tolerance; but if, on the other hand, there be a marked improvement, it should be no further increased, but continued without alteration until either the improvement ceases—in which case it should be again gradually increased—or the disease has altogether subsided. Whenever the latter is the case, we ought to diminish the dose day by day, rather than discontinue it suddenly, as by following the latter course we have less reason to dread a relapse." In continuation of these remarks, Dr. Barlow adds, "In some cases, however, especially those in which there is considerable anæmia, the iron seems to have more control over the disease than has the zinc, though these cases are rather exceptionable ones." The late Dr. Bright tells us that he found the sulphate of zinc answer where the carbonate of iron had failed, and that where iron succeeds, there the zinc had done no good; and Sir Thomas Watson, who repeats this statement of Dr. Bright, leaves us to infer that these words express his own experience in the matter.

Arsenic is another favorite medicine in the treatment of Chorea, especially in Great Britain and Ireland. Thomas Marten was the first to recommend it, now sixty years ago; and since his time it has been very extensively used. Dr. Romberg, speaking of the various remedies recommended, and alone deserving confidence as capable of arresting the disease in a short space of time, says, "The foremost among those an experience of several years has taught me to be arsenic." Dr. Begbie also writes, "In an experience of nearly thirty years I have never known arsenic fail." Nor would it be at all difficult to cite other authorities to the same effect. Dr. Begbie gives five drops of Fowler's solution twice a day, an hour after a meal, and adds a drop to the dose every day until the specific effects of the mineral upon the system are observable, and then he suspends the treatment for a while. He goes on with the medicine, that is to say, until he is warned to stop by itching and swelling of the eyelids, by redness of the conjunctivæ, by a white, silvery appearance of the tongue seldom accompanied by tenderness, and by nausea and uneasiness at the pit of the stomach. I have often used arsenic in the treatment of Chorea, and I have great faith in its efficacy as a medicine in the malady. At the same time I have often abandoned its use in consequence of the gastric disturbance which, do what one will to prevent it, was set up by it. It seemed, indeed, as if in these cases the stomach would not tolerate the medicine in doses large enough to produce a sufficiently rapid action in the cure of the disease. It did not follow, however, that this intolerance of the stomach was a sufficient reason for abandoning the arsenic in these cases, for the stomach is not the only channel by which this medicine could have been introduced into the system. Failing the stomach, indeed, the hypodermic or endermic method might have been tried, and that too, I have now reason to believe, with many chances of advantage to the patient. The case which suggested to me the hypodermic use of arsenic was that of a patient in the Westminster Hospital (Hallett Ward), Margaret S—— by name. This patient had suffered for nine years from a distressing choreal affection of certain muscles of the neck, by which the head was continually kept turning and bobbing. At different times various modes of treatment had been tried, including the hypodermic injection of morphia and atropine, without the least benefit. When first admitted under my care, and for the three weeks following, I gave her bromide of potassium and morphia, my chief object being to procure sleep and alleviate pain in the neck; for the muscles in the neck, which were the seat of the morbid movements, were very tender in many places, and the movements themselves attended with much pain; but harm, rather than good, seemed to be done by these means. The idea of injecting arsenic hypodermically occurred to me on the 12th of January, 1866, and was carried out on the same day. Fowler's solution was chosen, and the part selected was the most tender point over the contracting muscle. Three minims were injected on the 12th, ℳ v. on the 15th and on the 27th, ℳ vj. on the 19th, ℳ viij. on the 22d, ℳ vij. on the 25th, and again on the 29th, ℳ viij. on February 1st, ℳ ix. on the 3d, ℳ x. on the 6th, ℳ xj. on the 8th, ℳ xij. on March 1st, and again on the 10th, ℳ xiij. on the 12th, ℳ xiv. on the 14th. On the 21st the patient left the hospital almost well. Before the fourth

injection was practised, a marked change for the better had taken place; before the eighth the choreal movements were almost at an end, and the change for the better had gone on steadily progressing from the beginning. Between the eighth and the ninth injection there was an interval of three weeks[1]—the injections being suspended on account of the local irritation and inflammation which they had set up. When the patient left the hospital there was some stiffness in the muscles which had been the seat of the disturbance, by which the head was slightly twisted, and the voluntary movements of these muscles were not free; but every day there was a change for the better in these respects. In the hospital, the only treatment associated with the injections was a gymnastic one, the patient being made to move her head from side to side, and backwards and forwards, in time with a slowly moving pendulum, together with an occasional dose of morphia at bedtime, the drug being given less on account of the malady in the neck, though pain in this region was still complained of at night, than on account of a distressing habit of sleeplessness. Two months have now elapsed since the patient left the hospital. She occasionally presents herself for inspection, and her state continues very much the same as it was, just one step from being quite well, and not bad enough to make her wish to have the injections repeated. She goes on exercising the muscles of the neck with the pendulum, and having them shampooed, and for medicine she has now and then had some cod-liver oil. In this case the object in introducing the arsenic hypodermically was, not to escape gastric irritation, but to produce some local change in the nerves of the parts which were the seat of the disorder, as well as to bring about some more general change in the system. I have employed, with results more or less satisfactory, the hypodermic injection of arsenic in several analogous cases, and also in certain cases of neuralgia, epilepsy, and other affections of the nervous system; and thus the case which I have given is not the only case which furnishes to my mind reason for believing that this mode of treatment may be of use in the treatment of certain cases of Chorea. I have also used arsenic endermically as well as hypodermically in a few cases of Chorea. In order to this I have dropped from fifteen to twenty drops of Fowler's solution upon lint moistened with water, and applied this, under oil-silk, night and morning, to a raw blistered surface. This application gives rise to considerable local irritation; indeed, it generally, before the week is over, has the effect of covering the blistered surface with a thin, dry eschar, and of causing a zone of angry pimples to crop up in the skin immediately surrounding the part which has been blistered. Owing to this irritation, indeed, it is generally necessary to make pauses in the treatment after going on for six or seven days at a time. As yet, however, I have little practical experience of the effects of this mode of treatment in actual cases of Chorea. I have tried it in two cases of average severity, in one of which the patient was well in twenty-eight days, in the other in thirty-two days; and this is all that I can say respecting it, except this, that as with the hypodermic method before mentioned, so also with this, I have given it a trial in certain cases of neuralgia and epilepsy, and that the results arrived at in these cases lead me to hope that this mode of giving arsenic may prove to be a not unimportant addition to the *armamenta therapeutica*. With respect to the comparative merits of the hypodermic and endermic methods of introducing arsenic into the system I cannot yet speak. I incline to give the preference to the former method, both as least distressing to the patient and as most efficacious; but I have, as yet, no sufficient practical experience to justify the expression of a definite opinion.

During the last twenty-five years *strychnia* has been employed somewhat extensively in the treatment of Chorea, especially in France. Dr. Trousseau was the first, or among the first, to do this, and after an experience of a quarter of a century he is still disposed to give the preference to this practice. The preparation employed by this physician is a *syrup of the sulphate of strychnia*, made by dissolving 3 grains of the sulphate in ℥x of simple syrup; and the manner of giving it, which is peculiar, is as follows:—In children from five to ten years of age, the treatment is commenced by giving a *teaspoonful* of this syrup (containing $\frac{1}{8}$th of a grain) twice or thrice a day—one dose in the morning, another in the evening, and the third, if there be a third, at noon. On the next day these doses are repeated. On the following days, each day an additional teaspoonful of the syrup is given until six teaspoonfuls are given, care being taken to distribute these four, five, or six doses at equal intervals through the day. Having arrived at this point, if the

[1] Up to the eighth injection undiluted Fowler's solution was used; when the injections were resumed, and after this time, this solution was diluted with an equal quantity of water. In other cases, also, where the same mode of treatment has been carried out, I have employed a mixture of equal parts of this solution and water, for I found that the solution diluted to this extent produced very much less local irritation than the undiluted Fowler's solution.

physiological effects of the dosing are not yet produced, *dessertspoonful* doses are substituted for teaspoonful, and the same rule is observed with these larger doses as with the smaller. Beginning with two or three dessertspoonfuls in the course of the day, and giving three on the next day also, the doses are increased by a dessertspoonful each day, until six dessertspoonfuls are taken in the course of the day, care as before being taken to distribute these doses, few or many, with intervals between them as wide as possible. If the desired effect be not yet produced, a still bolder practice is pursued, and a tablespoonful of the syrup is substituted for *one* of the desertspoonfuls; and you are to go on, still augmenting, but in a way which is not very clearly laid down. "En augmentant progressivement," M. Trousseau says, "avec la même prudence, avec la précaution essentielle de distribuer le médicament à des intervalles sensiblement égaux dans le courant de la journée, vous arrivez à donner aux enfans de cinq à six ans 50, 60, 80 et jusqu'à 120 grammes [5 grammes go to the teaspoonful] 25 milligrammes de sirop; 3, 4, jusqu'à 6 centigrammes de sulfate de strychnia." In persons older than ten years, Dr. Trousseau begins with large doses, with dessertspoonfuls in place of teaspoonfuls, and goes on until he reaches 200 grammes of the syrup—a quantity containing no less than 10 centigrammes, or 1½ grain, of the active principle. The object is to produce the full physiological effects of strychnia, and to maintain them for a while, and the duration of the treatment is said to be thirty-three days for girls and seventy-four days for boys. When the medicine begins to tell upon the system, the symptoms are, twenty minutes after taking it, or thereabouts, slight stiffness in the jaw or neck, some headache, confusion of sight, and giddiness, and some disagreeable "démangeaisons" in the parts of the skin covered with hair. Afterwards as the system becomes more deeply impressed, the stiffness extends from the jaw and neck to the limbs and elsewhere, especially to the limbs most affected with Chorea, which limbs are also in all probability more or less paralyzed, the itching of the skin is no longer confined to the hairy parts, and painful jerks or shocks, or still more obvious tetanic symptoms, make their appearance. The tolerance of strychnine varies not only in different individuals, but in the same individual at different times, so that the dose which was not more than enough one day may be poisonous the next. In fact, the treatment is one which requires to be most carefully watched, and which cannot well be watched with comfort, especially by the friends of the patient, however enlightened or forewarned they may be.

Another heroic treatment for Chorea, which has found some favor in France, is that by *tartar emetic*. Laennec has left on record three cases of Chorea treated by large doses of this medicine, and others have tried the same method with results, as they seem to think, more or less satisfactory, especially MM. Boulay, Gillette, and Henri Roger. M. Gillette's method, which is that adopted by M. Roger, is to give the antimony for three days, to withhold it for three or five days, and again to give and withhold it for the same period, as often as may be necessary, if the symptoms have not yielded to the medication of the first three days. On the first day of the first triple series of days, the dose given in the twenty-four hours is from 20 to 25 centigrammes (1 centigramme is = ·15, or nearly ¼th of a grain). This dose is doubled on the next day, and tripled on the third day; then the patient is allowed to rest from three to five days. On beginning again, if this be necessary, the dose given on the first of the three days, which is to be doubled on the next day, and tripled on the day following, is 5 centigrammes larger than that used on the day in which the treatment was commenced. If this be not enough, after waiting again for from three to five days, the dose for the first of the three days is 5 centigrammes larger than that used on the first of the last series of three days, for the second day the dose is doubled, and for the third day the dose is tripled; so that, if the dose given on the first of these three days was 30 centigrammes, the dose on the last of these three will be 90 centigrammes, *or nearly* 14 *grains!* In the majority of cases we are told the first doses are followed by nausea and vomiting of a glairy matter, but these symptoms soon pass off, and complete tolerance is established, especially if care be taken to withhold as much as possible all dietetic drinks. We are told also that diarrhœa is uncommon, that constipation is not uncommon; that the pulse becomes slower, that the skin moistens, and that the general health improves. Indeed, Dr. Bourguignon, speaking of certain children, patients of M. Gillette, under this mode of treatment in the Hôpital des Enfans Malades, at Paris, says: "Les enfans ne sont nullement abattus, ils conservent leur gaieté." Dr. Bourguignon, who is strongly in favor of this treatment, tells us also that in ten cases—whether in M. Gillette's practice, or in his own, he does not say—the patients got well in sixteen days, as an average, the shortest time being four days, the longest twenty-four.

Iodide of potassium is another remedy which has been tried somewhat extensively in the treatment of Chorea, and to a less extent so has *bromide of potassium*. This iodide was supposed to be indicated

by the probable existence of a rheumatic or lymphatic predisposition in the patient, or by the actual presence of some meningeal irritation or inflammation, and these indications have been carried out fully and frequently; but the practical results of this treatment, so far as I know, are unsatisfactory. Nor is a different opinion to be expressed with respect to the bromide. I have tried this medicine in several cases, and tried it fully, and from what I knew of its strange efficacy in epilepsy I was strongly prejudiced in its favor; but the result, as I have said, is that the bromide appears to be no more justly deserving of confidence than the iodide.

As might be expected, *opium* is a medicine which has not been overlooked in the cases in question, especially in the severer cases. As in tetanus, there appears to be a remarkable tolerance of this medicine in Chorea, and in several cases enormous doses have been given; indeed, in any case it appears to be necessary to give large doses, in order to procure what may be supposed to be the object in view, that is, sleep. I have seen opium employed in five very severe cases of Chorea, largely, and from what I saw in these cases I am not wishful to see the experiment repeated. I am speaking now of the free use of opium by itself, and not of opium in moderate dose along with other agents, with the free use of alcoholic stimulants especially. This, I believe, is quite a different matter. Nor does there appear to be sufficient reason for supposing that other narcotics, not excepting *cannabis indica*, are more to be trusted than opium in cases of Chorea.

The *inhalation of chloroform or ether* has been had recourse to in many severe cases of Chorea. I have seen three such cases in which chloroform was used in this way, and my impression was that harm, not good, was the result. I believe, also, that harm rather than good is likely to be done in these cases, unless alcohol is given in sufficient quantities before the inhalation. If this be done, the patient may remain asleep for some time, and awake the better; if this be not done, there is great danger, so far as my experience of the use of chloroform inhalations is concerned, of the patient waking almost immediately, and of being more unnerved and more agitated than he was before he was put to sleep. At the present time I am in attendance upon a case of Chorea, attended with much sleeplessness, until the practice was adopted of giving at bedtime a few whiffs of chloroform *after* a glass of hot negus. The chloroform had been tried for four nights without the negus, and harm rather than good had been the result. It has now been tried with the negus for a week, and, as it would seem, with unmistakable advantage. Nor is this an isolated case.

Antispasmodics, such as camphor, ether, valerian, assafœtida, and musk, have been tried extensively, and the general verdict appears to be that they are not useless. I am disposed to place considerable confidence in *camphor*, and also in *ether;* in camphor especially. I often give very generally this last-named medicine dissolved in cod-liver oil, and my impression, from what I have seen, is that this addition to the oil is a decided advantage to the patient.

Turpentine has been given for various reasons in Chorea—as an anthelmintic and purgative chiefly. At one time I gave it rather as a general stimulant, and, as it seemed, with benefit to the patient. I then tried *mineral naphtha* with the same view, and came to the conclusion that this medicine was more pleasant than turpentine, less trying to the system, and not less efficacious. During the last six or eight years, however, I have rarely given either one or the other of these medicines, and one chief reason for this seems to be that I have gradually come to prefer the treatment of which I have to speak in a few moments.

Ammonia is also a remedy which has some good claim to be mentioned in the present place. I have tried the sesquicarbonate in several cases, singly and in combination, and the trial has been to my mind eminently satisfactory. I am, for example, at present seeing a little choreic boy who had been for three weeks treated, without any benefit, with sulphate of zinc, and who has wonderfully improved during the last three days, by leaving off the zinc, and by substituting sesquicarbonate of ammonia in five-grain doses every three hours. In other respects there was no change in the treatment, and the patient is too young to allow it to be supposed that he was affected beneficially by the *change* of the practitioner.

For various reasons, theoretical and practical, the free use of *alcoholic drinks* has long seemed to me to be the foundation of a rational plan of treatment in Chorea, and the larger experience of the last few years has only served to confirm me in this opinion. I have seen enough to know that, as a rule, the change for the better is unmistakable when, after the carrying out of a contrary mode of practice, alcoholic drinks are given with a liberal hand. I have notes of three cases of great severity, where rapid amendment was brought about by giving, at frequent intervals, an egg beaten up with a large glass of sherry or with an equivalent dose of brandy, and I verily believe that this plan would rarely fail if carried out *in time*—if carried out, that is to say, before

the nervous system had become thoroughly exhausted and broken down, as it does do in the end. Indeed, in a bad case, where a dangerous degree of sleeplessness had to be dealt with, there is nothing in which I should have more confidence than in the free use of alcoholic drinks. I should look upon these means properly, that is freely, used, as the natural means of procuring sleep and all the beneficial consequences of sleep. I should be afraid of attempting to attain the same end by the use of *medicines* more or less analogous to alcohol in their action, because these medicines would all of them be more likely to disturb the action of the stomach, and so interfere with the restoration of the system by food. And for the same reason I should even be almost afraid of giving small doses of opium with the view of conciliating sleep, though I have no doubt that the proper dose of this drug at the proper time, in conjunction with the proper dose of alcohol, might be very satisfactory practice. In a word, I cannot but think that it is a perfectly rational way of dealing with severe cases of Chorea to push alcoholic drinks until they produce drowsiness—until, that is to say, they exercise a decidedly sedative action upon the system. At any rate I have carried out this idea in more than one case of the kind with what seem to me to be very satisfactory results.

In bad cases of Chorea, as a matter of course, the *recumbent position* is a necessary part of the treatment; and in cases of ordinary severity my own impression is that the patient would improve more rapidly if he were kept longer in bed. Indeed, it surely stands to reason that *rest*, properly used, is a right means of remedying a state of muscular disorder, in which muscular fatigue is an unmistakable element. Nay, it is not too much to imagine, that the persistency of many cases of Chorea may be not a little owing to the patient being allowed to be up and fidgeting about when he ought to be in bed.

Exercise, on the other hand, properly used, cannot well be dispensed with as a means of treatment in Chorea. In ordinary cases, indeed, it is difficult to overrate the importance of suitable gymnastics as a means of cure. This is no new idea. Darwin insisted upon it long ago; and from what has been done in this direction since his time, especially by Ling and his successors in the practice of the so-called "movement cure," it is perhaps not too much to say that Chorea may be one of the consequences of neglecting gymnastics as a means of education in children. Certain it is, that ordinary cases of Chorea get better rapidly—the average duration of the period of treatment being sixty days—under a properly arranged course of gymnastics, with little or nothing else. The practice of M. Sée, at the Hôpital des Enfans Malades in Paris, may be cited in support of this statement; and it would not now be difficult to find corroborative passages in the practice of others. For myself, I should think that I was omitting an important duty if I did not prescribe the use of some suitable exercise for a patient suffering from Chorea—the use of a skipping-rope or trapeze, if nothing else. Dancing has long been a favorite idea with me as a means of exercise in cases like these; and so have calisthenics regulated by music. More than one choreic patient I have known to be cured by learning to dance, and I think that music might be employed with advantage now, as it was in the case of the tarantula dance of old, in quieting severe cases of Chorea, anomalous or not. Indeed, there is more than one case on record in which music has been so employed. It may be supposed also that music will help the choreic patient in his gymnastic efforts in the same sense as that in which it nerves the acrobat to the performance of his wonderful feats. In a word, it is not necessary to think long before it must become self-evident that orderly movements, be they those of dancing, calisthenics, or more special gymnastics, and be they regulated by music or not, are natural remedies for disorderly movements such as are met with in ordinary cases of Chorea, and that a very important means of cure is neglected if they are not provided. Indeed it is to be hoped that the time is not far distant when a suitable gymnasium will be considered as much a part of the proper fittings of a hospital as the dispensary, and when medical men more generally will be alive to the importance of suitable gymnastics, not only as an educational, but also as a curative measure. Surely there is a lesson to be learnt from the results of the carrying out of the "movement cure,"—a lesson which the practitioners of orthodox medicine are not justified in continuing to decline to learn because it happens to have heterodox belongings!

Baths, of one kind or another, have been extensively employed in the treatment of Chorea. In this country the *cold shower bath* has been the favorite mode of bathing, and there are some good grounds for this preference. Part of the good result is ascribed to the shock; part—a greater part, perhaps—to the reaction. Still there are, unquestionably, many cases in which the shock is not tolerated, and where reaction is not easily established—cases in which the patient is rendered worse rather than better, so far as the Chorea is concerned, with the additional disadvantage of a bad cold, or actual rheumatism, or some other evil.

And these latter cases are by no means uncommon. Nay, it may even be suspected that all cases would come into this category if care were not exercised more or less. The same remarks apply also to cold plunge baths, and to other forms of cold baths. With respect to *hot baths* and to *warm baths*, the case is very different. A hot bath at bed-time has often seemed to me to have a marked calmative influence. I am also disposed to think that a good part of the benefit ascribed by M. Baudelocque to *sulphur baths* (each bath contains about four ounces of sulphuret of potassium) is to be ascribed to the high temperature of the water, or, at any rate, to this in conjunction with the counter-irritation set up by the action of the sulphuret upon the skin. The fact appears to be that baths of one kind or other are not sufficiently recognized as a means of cure, not only in Chorea, but in many other cases of disease, by the orthodox practitioners of medicine. With baths, indeed, it is very much as it is with "movements" as a means of cure, and hydropathy, like kinesitherapy, has a lesson to teach, which medical men ought to set themselves to learn if they would be fully provided with the means by which to contend successfully against disease.

Electricity is another agent which requires a passing mention in this place, though all that can be said respecting it is, that as yet there appears to be little or no reason for placing any confidence in it as a means of treatment. Whether this will be always the case—whether there are not modes of using electricity which will have the effect of quieting choreic and analogous movements (so long, at any rate, as they are used)—remains to be seen. I suspect that there are such modes, and that they will be beneficial, and that too not a little, in the case in question, but I have not yet the *facts* to justify the expression of a belief on the subject.

For the last seven years I have employed cod-liver oil in many cases of Chorea, and, so far as I can judge, I have good reason to be satisfied with the results. In adopting this practice my main object was to restore nerve-tone by improving the nutrition of nerve-tissue. I remembered that fatty matter was an essential ingredient in nerve-tissue; and, remembering this, I came easily to the conclusion that one natural way of attaining to the end in view was to take care that the food contained a sufficient amount of fatty and oily matter. Without a due supply of these matters, I reasoned, the nerve-tissue must be of necessity starved—that, in fact, to withhold these matters, or to supply them in insufficient quantity, would be as great a mistake in cases where the object was to improve the nutrition of the nerves, as it would be to withhold lean meat in cases where the object was to get more muscle. I argued in this manner, and be the theory right or wrong I think, as I have said, I have no reason to be dissatisfied with the results of putting it in practice.

For the last seven years also I have used phosphorus in the majority of cases of Chorea in which I have used cod-liver oil, and for the same reason. I asked myself whether the fact that phosphorus is present in large quantity in the great nerve-centres, and that the amount of this ingredient seems to have some direct relation to the activity of the nervous functions, being as much as 2 per cent. in adult life, and below 1 per cent. in infants and idiots, might not show that phosphorus is specially indicated as food for a weak nervous system—as much indicated, perhaps, as iron in cases where there is a deficiency of red-corpuscles in the blood; and this question once put seemed to require an answer in the affirmative. "In small doses," says Dr. Pereira, "phosphorus excites the nervous, vascular, and excretory organs. It creates an agreeable feeling of warmth in the epigastrium, increases the fulness and frequency of the pulse, augments the heat of the skin, heightens the mental activity and the muscular powers, and operates as a powerful sudorific and diuretic." In large doses, without doubt, phosphorus is a caustic poison; in proper doses, it produces the very changes that are to be desired in cases of Chorea and analogous forms of convulsive disorder. In proper doses and properly watched, it is quite innocent in its action, and may be very beneficial. Of this I am confident. The forms in which I first gave phosphorus in Chorea were the phosphorated oil of the Prussian Pharmacopœia and the ethereal tincture of the French Codex (forms containing 4 grains of phosphorus in the fluid ounce), but lately I have preferred the hypophosphites, especially the hypophosphite of soda, for the simple reason that these salts, which were originally recommended by Dr. Churchill of Paris as specifics in phthisis, are infinitely less nauseous than the oil or tincture, and not less efficacious. I have given for some time from 5 to 8 grains, three times a day, of the hypophosphite of soda to children, in cases of Chorea, without any harm certainly, and, as I think, with unmistakable benefit, and I have not yet found any reason to change this practice for another.

In an ordinary case of Chorea, the plan of treatment which I have now adopted as a rule for some time is to give cod-liver oil in conjunction with hypophosphite of soda, making the draught containing the latter salt the vehicle for the administration of the cod-liver oil. With these medicines, according to circumstances, I

have associated camphor or ammonia, one or both, adding the sesquicarbonate of ammonia to the draught containing the hypophosphite, and *dissolving the camphor in the cod-liver oil.* I have found that this latter solution is an excellent way of giving the camphor, and also that the camphor masks the taste of the oil not a little, and makes the stomach more tolerant of it. I have not kept notes of all the cases which I have treated in this manner, but I think I am quite within bounds when I say that the number now amounts to upwards of sixty, and that the average duration of the treatment was under one month.

I may also add that I have in three or four cases given arsenic along with hypophosphite of soda and cod-liver oil, and that the result, to say the least, was not such as to discourage a continuance of the practice.

If there be any special sources of irritation, as worms or carious teeth, these of course must be met and dealt with. If the agitation be so great that there is danger of the skin being excoriated, or of the patient falling out of bed, properly padded sides must be fixed to the bedstead, or it may be expedient to encase the body and limbs of the patient in cotton-wool. If the affection be confined to certain muscles of the neck or elsewhere, it may be expedient to use hypodermic injections of arsenic, as in the cases related in the text, to divide a nerve, as has just been done by Mr. Campbell De Morgan, or to use the actual cautery. If there be a morbid mental condition, as there too often is, moral means of treatment must not be neglected. In fact, each case of Chorea must be looked upon from a special as well as from a general point of view, and the success of the treatment will, in many instances, if not in all, depend upon the skill with which special means can be combined with those general means of which I have spoken, and upon which I have prosed at greater length than I ought to have done.

PARALYSIS AGITANS.

By William Rutherford Sanders, M.D., F.R.C.P.

Synonyms.—Paralysis tremens, tremula, jactitans, palpitans; Tremor artuum, T. coactus; Scelotyrbe festinans, seu Festinia (Sauvages); Synclonus tremor, S. ballismus (Mason Good); Schüttelähmung, Schüttelkrampf, Zittern; Tremblement sénile, Trémulence paralytique progressive; Chorea senilis, Ch. festinans; Pseudo-chorea, Pseudo-paralysis agitans; Dystaxia agitans; the Trembles, &c.; the Shaking Palsy (Parkinson).

Definition. — Idiopathic Paralysis Agitans consists of involuntary tremulous or shaking motions of the limbs, head, or trunk of the body, which takes place even when the parts are supported and unemployed. The voluntary movements are preserved, but their vigor is lessened in the affected parts. In certain, usually advanced, cases, there is a disturbance of equilibrium; most commonly a disposition to stoop, or bend the body forwards, and to pass, in locomotion, from a walking to a running pace. The senses and intellect are unimpaired.

The definition includes these principal characters: 1st, The shaking or tremors, of a spasmodic kind, which occur even when the parts are not in use (Tremor coactus, palpitatio, παλμός, agitatio, jactitatio, quassus); 2d, The diminished muscular power (Paralysis, paresis, pseudo-paralysis); 3d, The disturbed equilibrium, shown usually in the tendency to stoop and to move forwards with accelerated speed (Scelotyrbe festinans, festinatio, procursus). Of these characters the clonic tremors or shaking are the most constant and distinctive. The paralysis, on the other hand, is of a peculiar kind. As here employed, the term does not mean cessation or interruption of voluntary motion, which, on the contrary, persists; but it is intended to designate both the imperfection of the movements, which results from the interference of the tremors, and also the impaired strength and the slowness of muscular action, which are usually observed in the tremulous parts. Some writers, objecting to call this condition paralysis (a name apt to mislead), have spoken of it as "apparent but not real paralysis," or "paresis," or "pseudo-paralysis," or "dystaxia." Lastly, the disturbance of equilibrium does not always occur: it is often late in appearing, and it serves chiefly to mark a special form or

an advanced stage of the affection. Nevertheless, in fully developed examples of Paralysis Agitans, all these symptoms, the tremors, paralysis, forward stoop, and accelerating walk, are associated together; as Parkinson expressed it, there is a combination of Tremor coactus and Scelotyrbe festinans.[1]

Historical Notice.—From the definition, as explained, it will be apparent that the older descriptions of Paralysis Agitans are to be sought less in the history of palsies than in that of spasmodic nervous diseases. In fact, Paralysis Agitans has been overlooked principally from being confounded, 1st, with tremors in general; 2d, with chorea; 3d, with cases of motor palsy (hemi- or paraplegia) complicated with spasmodic and tremulous movements.

1st. As a symptom, tremors early attract attention. They were briefly noticed by Hippocrates and Celsus, while by Galen and succeeding writers their kinds, their causes, and their value as prognostic signs were fully discussed. At length, nosologists established the genus Tremor, dividing it into species, in some of which tremor was still regarded as a symptom merely, while in others it was recognized as a substantive or idiopathic disease. Accordingly, well-marked cases of simple Paralysis Agitans are found in many of the older authors,[2] by whom they are sometimes alluded to, sometimes described with graphic details, in illustration of the pathology of tremor or as examples of a distinct species of disease. Less notice was taken of the disposition to stoop and hasten onwards. The earliest mention of this curious symptom is probably made by Gaubius; it was first particularly described by Sauvages under the name of Scelotyrbe festinans. But Sauvages did not connect it with tremors; he, indeed, had seen only two cases of it. It is certain that the combination of persistent tremors and hurrying gait had not been recognized, and no adequate description of Paralysis Agitans existed previous to Parkinson's "Essay on the Shaking Palsy in 1817." His account still remains the standard authority. Succeeding authors have, in general, simply quoted it, or have (especially French writers) overlooked the disease altogether. Accordingly, although Parkinson drew attention to the imperfection of our knowledge, the original contributions made since his time have been few and fragmentary. A list of references will be found at the end of this article.

[1] Parkinson seems to consider the festination as essential to Paralysis Agitans, but it cannot be so regarded.

[2] Sylvius, Bonet, Juncker, Van Swieten, Sauvages, &c.

2d. In regard to the confusion of Paralysis Agitans with chorea, it must be remembered, that convulsive diseases have been imperfectly discriminated, owing partly to the difficulty of their pathology, partly to the superstitions with which they have been associated. The disease now commonly called chorea was not so named originally, nor was it confounded with true St. Vitus's dance; it was regarded merely as a kind of convulsion (motus convulsivus) or species of epilepsy (epilepsia gesticulatoria), till near the end of the 17th century.[1] About that period, Sydenham, in the brief description which fixes the characters of the disease, unfortunately named it "Chorea Sancti Viti," a misnomer which it has since commonly retained, with the effect of confounding it with the dancing mania, from which it is quite distinct.[2] While older authors therefore may have described cases of Paralysis Agitans among the motus convulsivi extraordinarii (just as some authors have given definitions applicable to it under the name hieranosos),[3] it is only in recent times that Paralysis Agitans has been confounded with what is at present known as chorea. This confusion is due partly to a certain similarity in the diseases, partly to the unsettled state of medical nomenclature. Ordinary cases of the shaking malady are widely distinct from common chorea; but certain extreme forms occasion a violent irregularity of movement, resembling in a great degree the gesticulations of that disease; so much so, that it has been proposed to regard Paralysis Agitans as a more intense form of chorea,[4] and cases of Paralysis Agitans have been recorded under the title St. Vitus's dance.[5] Some cases even exhibit a combination of Paralysis Agitans and chorea.[6] Moreover, while the common gesticulatory chorea is well known, there are other rarer forms, of irregular and uncontrollable spasmodic movements, as yet imperfectly studied and classified, to which the term chorea is usually applied: such are the rotatory or spinning-top chorea, the saltatio or leaping ague, malleatio, &c. In some

[1] Roth.

[2] Authors distinguish the common chorea of Sydenham as chorea minor, the dancing mania as chorea major, choreomania, or tarantismus. By chorea or St. Vitus's dance, however, Sydenham's disease is now always meant, the tarantism from its rarity being left out of account.

[3] Linnæus, Gen. Morb., Upsal, 1763, p. 17, No. 144: "Hieranosos, Byting, Corporis agitatio, continua, indolens, convulsiva, cum sensibilitate." Also Vogel and Macbride.

[4] Eisenmann, remark on Dr. Haas's case, in Canst. Jahrb. 1852, iii. 92.

[5] Trousseau, case in 1843. See references.

[6] Maclachlan.

respects, therefore, it is not altogether inappropriate to designate Paralysis Agitans as a species of abnormal chorea; hence some recent authors employ the terms chorea senilis and chorea festinans for Parkinson's disease. The objection to such names is, that they tend to confound Paralysis Agitans with the ordinary St. Vitus's dance, from which it is entirely different.

3d. Lastly, the term Paralysis Agitans or shaking palsy has been applied, both before Parkinson's essay and since, to cases of ordinary motor paralysis (hemi- and paraplegia) complicated with tremors —a complication not uncommon both in diseases of the brain, and especially in certain cases of chronic myelitis and of locomotor ataxia. Etymologically the name of shaking palsy belongs perhaps to these, rather than to Parkinson's disease, but time has consecrated his use of the term. Parkinson's malady is *Idiopathic* Paralysis Agitans, in which the tremors or shaking are the chief and earliest symptom, and the paralysis entirely subordinate and peculiar, true hemi- or paraplegia being rare complications: while, in the cerebral and spinal affections just referred to, the loss of motion (akinesia) or sensation (anæsthesia) is the main feature of disease, and the tremors and spasmodic agitations are only concomitants (*i. e.* the Paralysis Agitans is *Symptomatic*). Hence the latter class of cases should be styled, not Paralysis Agitans, but hemi- or paraplegia, or spinal or cerebral disease *complicated with Paralysis Agitans: i. e.* with spasmodic tremors. This distinction, which is essential for the accurate definition of Parkinson's disease, has often been overlooked, and requires, therefore, to be specially insisted upon.

In the following description of Paralysis Agitans, besides some allusion to tremors generally, it is proposed for the sake of distinctness to recognize certain subdivisions of the disease, which experience accumulated since Parkinson's essay seems to require.

Divisions. — Idiopathic Paralysis Agitans is divided into I. General (including the bilateral and unilateral), and II. Local.

I. General Idiopathic Paralysis Agitans presents certain forms or varieties important to distinguish, as regards prognosis and cure:—

A. Senile forms. *Paralysis Agitans senilis*, occurring in advanced life, above fifty or sixty; usually incurable and with fatal tendency from senile decay: divided into 1st, *Simplex*, and 2d, *Festinans* or *Procursoria*. Varieties, *Unilateralis* or *Hemiplegica* and *Retrograda*.

B. Non-senile forms, occurring under fifty, without fatal tendency, sometimes curable. 1st, *Paralysis Agitans non-senilis, simplex* (*i. e.* sine festinatione), including also *hysterical* and *reflex* Paralysis Agitans, &c. 2d, *Paralysis Agitans toxica*, including, chiefly, tremblement métallique, mercurial palsy, &c.

The curable forms have been supposed to be *Functional;* the incurable, *Organic.*

It will be necessary to describe the senile forms in detail, as they are much the most frequent; a shorter notice will suffice for the others.

Description.—I. A. Idiopathic Paralysis Agitans senilis. 1st, simplex, when attended by the signs of senile decay only; 2, festinans, or procursoria, &c., when the disturbance of equilibrium is also present. These two forms will be described together.

Symptoms and Course.—Onset usually gradual; course slow, progressive, liable to be arrested at different stages; duration protracted, associated with senile decay. Several stages may be recognized.

1st Stage. Commencement. — The first symptoms are usually so insidious that the patient cannot tell precisely when they began. A sense of weakness and a disposition to tremble is felt in some part, most frequently the hand or arm, sometimes the leg or head. The tremors, at first slight and occasional, gradually increase; and at an uncertain period, seldom less than a year, the corresponding parts of the opposite side, more rarely the other limb of the same side, become affected. The tremors and muscular debility seldom extend beyond the arms during the first two years, which period may be said to comprise the first stage. Except for the inconvenience arising from the unsteadiness of the hand in writing or other manipulation, the patient would not consider himself the subject of disease. At this period, probably, remedies might be applied with success. In a few cases, instead of the ordinary gradual approach, the tremors have come on rapidly after a fright or exposure to cold.

2d Stage. Generalization of the Tremors. —Some time after the hands and arms have been affected, one of the legs, usually that on the side first attacked, begins to tremble and is more easily fatigued; and in a few months the other leg becomes similarly tremulous and weak. Walking becomes a task requiring considerable attention. The legs feel heavy as lead, and are not raised to the height or with the promptitude which the will directs, so that care is necessary to prevent frequent falls. At a later period, usually some years after, the tremors extend to the head, and finally to the whole body. The tremors of the limbs are usually in the direction of flexion and extension, sometimes of rotation, sometimes of

ab- and adduction (so that patients have had their knees padded to prevent them knocking together). In the head[1] and neck the movements are more commonly lateral (shaking negatively), then vertical (nodding). The lower jaw is affected with vertical, rarely lateral motions; and the tongue is tremulous, impeding speech: in many cases, however, these parts are not affected till near the end of the disease. The larynx is little, if at all, affected. Deglutition does not suffer till near the close. The muscles of the eyeballs and eyelids, and the facial muscles of expression, are nearly always exempt from tremors.[2] The thorax and trunk are later and less affected than the limbs, or head, or neck. Appearing chiefly during a general paroxysm of tremors, the spasmodic action of the respiratory muscles occasions a peculiar panting of the breathing and a jerking interruption of speech. As the tremors last, and become general over the body, they increase in intensity; from mere vibrations they become violent convulsive agitations. The limbs are jerked to and fro as if by the action of springs or by rapid shocks of electricity.

From the beginning, and throughout the whole course of the disease, mental emotion or agitation excites an attack of tremors, or greatly aggravates them; rest and quietude diminish or stop them. In general, a slow, firm, voluntary act, or the grasp of a heavy body,[3] stops the tremors for a time, and any change of posture has the same effect, affording the patient considerable relief. Parkinson mentions an artist, who, while his arm and hand were palpitating strongly, would seize his pencil, with the effect of instantly suspending the tremors and allowing him to use it for a short period.[4] On the contrary, when the limbs are quiescent, a voluntary movement usually starts the tremors, which continue for some time afterwards. The attacks of tremors are at first of short duration, and separated by intervals of complete immunity; they become more severe and the intermissions shorter as the disease proceeds. In certain examples the paroxysms have lasted so long as ten to forty minutes, and were followed by fatigue like that produced by violent muscular exercise.[1] The tremors cease entirely during sleep.

Parkinson does not seem to have noticed that frequently, in addition to the tremors, there occur tonic spasms (rigidity or contraction) of the muscles in the parts affected. The fingers or toes or the whole limb become rigidly flexed or extended. These cramps last for some minutes, and return at intervals; they are sometimes painful and followed by a sense of fatigue. They occur chiefly during the day, but sometimes in the night also. In a case recently observed by the writer startings of the limbs took place during sleep, in the form of powerful flexion of the knees, by which the legs were suddenly drawn up. On the relaxation of the spasms, the limbs were slowly let down to their previous position without awakening the patient.

Local deformities also sometimes result. From the hands being kept constantly supported to stay the tremors, the fingers become dislocated backwards on the metacarpals so as to form an angle with the back of the hand.[2] Sometimes the distortion is lateral; in a case lately seen the fingers were bent obliquely to the radial side, owing probably to the clonic and tonic contractions being more powerful in that direction. These deformities must not be confounded with the effects of rheumatism.

[A not uncommon symptom is a sense of great *heat* of the body; causing the patient to throw off the bedclothes, &c. This sensation is shown by the thermometer to be subjective only; there being no real excess of temperature.—H.]

3d Stage. Disturbance of Equilibrium.—The occurrence of this symptom is variable: sometimes it appears early, while the legs are becoming tremulous; sometimes it is deferred for ten or twelve years or more after the tremors; in many cases it is entirely absent (Paralysis Agitans simplex). It is therefore less a stage than a peculiar feature characteristic of one form, or of a special extension of the disease; its presence should accordingly always be indicated by some additional term, such as *festinans* or *procursoria*.

Owing to deficient power in the extensor muscles of the back, the patient becomes less able to preserve the erect posture; he bends forward while sitting, still more while standing. In walking, the centre of gravity being displaced forwards, while the legs can only be moved slowly, stiffly, and with some degree of spasmodic jerking agitation, he is in constant danger of falling; he stumbles over small obstacles in his path, and by taking short hurried steps he is impelled from a walk to a run,

[1] Erb and others assert that the head is seldom or never affected in Paralysis Agitans, although it is in cerebro-spinal sclerosis. See Erb on Multiple Sclerosis, Ziemssen's Cyclopædia, vol. xiii.—H.]

[2] In Oppolzer's remarkable case the tremors are reported to have extended to the muscles of the face; also in a few other cases.

[3] A patient we saw lately holds a smoothing-iron to keep his hand still; another steadies it by seizing a chair.

[4] Lebert refers to a similar instance.

[1] Trousseau, Clin. Méd. Ibid.

till he has difficulty in stopping himself. Persons in an early stage of this condition can sometimes march slowly with long measured strides, quite well; but so soon as they resume their shuffling gait, they must quicken their pace to avoid falling. There is no vertigo, as in cases of precipitancy from tumors or injuries of the cerebral peduncles and adjoining parts. The forward running is the usual form of this curious symptom, which has not yet been much studied, but exceptional varieties occur. Thus Romberg met with an opposite disturbance of balance. "Two patients, aged sixty, felt a constant desire to walk or fall backwards, and therefore carried the head strongly bent forwards; one of them in order to stand, separated his legs widely, at the same time crossing his arms on the back, with the view of offering some resistance to the overpowering tendency to move backwards" (Paralysis Agitans retrograda). Graves mentions a patient who had to be balanced to and fro before starting, and who, if arrested in his forward movement, immediately began to hurry backwards and could not stop himself. No case is recorded of a disposition to fall or move sideways.[1] There is a less degree of this symptom in which the patient stoops and shuffles in his walk, but has not the true festination.

4th Stage. Disease fully established.—When the tremors have become general, violent, and of frequent recurrence, the patient experiences great inconvenience, which increases with the progress of the disease. The limbs cannot execute the directions of the will in the common offices of life. The patient is unable to write or perform any manipulation: he cannot hold a book to read, and has the utmost difficulty or is quite unable to clothe or feed himself. Raising a glass of water to the lips is impossible; the fluid is spilled and the glass knocked to and fro against the mouth. Patients deprived of assistance, in order to allay their thirst, have lapped up fluids with the tongue, like the lower animals. It is painful to witness the struggles which the patient makes to control the agitation of his body and effect some desired movement; the more he tries the more extravagant the jactitations become. To increase his distress, paroxysms of tremors now often arise during rest; indeed, at times, the tremors become almost constant, with frequent aggravations. Commencing for instance in one arm, the wearisome agitation is borne until beyond sufferance, when by suddenly changing the posture it is for a time stopped in that limb, but commences generally in less than a minute in one of the legs or in the arm of the other side, often spreading over the whole body. Harassed by the tormenting round, the patient has recourse to walking, to which he is partial, both on account of the relief afforded by change of posture and because his attention is diverted from his unpleasant feelings by the care and exertion required for its performance. But if the procursive tendency has appeared, this relief is denied. The propensity to lean forward becomes invincible. Forced to step on the toes and fore-part of the feet, while the upper part of the body is thrown forwards, he is irresistibly impelled to take quick and short steps, and to adopt unwillingly a running pace, in order to avoid falling on the face.[1] On some days, however, the tremors are less severe; and the patient is always relieved by intermissions during the day and complete cessation of the tremors during the night. The disease, even at this stage, sometimes undergoes remissions for some weeks or months, during which the tremors greatly abate: unfortunately a relapse occurs and the disease resumes its progressive course.

5th. Advanced and Final Stages.—Hitherto the jactitations have been suspended at intervals during the day, and have ceased entirely at night. But in this stage tremors of the limbs occur even during sleep, and increase till they awaken the patient, often with much mental agitation and alarm. In addition, signs of failing strength and physical decay, which had previously appeared, rapidly increase. Unable to convey food to the mouth, the patient must be entirely fed by others. The bowels, previously torpid, require powerful stimulating medicine or mechanical aid for their relief. The trunk becomes permanently bowed, and the whole muscular power fails. The patient walks with great difficulty; a stick no longer suffices; he requires an attendant, who, walking backwards before him, prevents his falling forwards by the pressure of his hands against the fore-part of his shoulders. His words are scarcely intelligible, and the memory and intellect are weakened. The actions of the tongue and pharynx are so hindered by enfeebled action and perpetual agitation, that the food can hardly be masticated or swallowed; the saliva mixed with particles of food continually drains from the mouth.

[1] Sauvages relates of a painter, aged 50, that he was not only impelled forward in walking, but could not turn right or left till he stopped himself against an obstacle, supported by which he turned his body gradually round and then hurried straight on anew. This is given as Scelotyrbe festinans, without any mention of tremors; but similar conditions have been observed in Paralysis Agitans.

[1] In the words of Trousseau, "Il s'en va trotillant, sautillant,—*il est obligé de courir, pour ainsi dire, après lui-même.*"

Finally, amid increasing general debility and diminished voluntary power, the tremors become more vehement, and seldom cease for a moment. When exhausted nature seizes a small portion of sleep, the motions become so violent as to shake the bed-hangings, and even the floor and sashes of the room. The chin is bent down upon the sternum; the power of articulation is lost; the slops with which he is fed trickle continually from the mouth. The urine and feces are passed involuntarily; bed-sores form; and at the last constant sleepiness, and other marks of extreme exhaustion, usher in the fatal termination.

The senile forms of the Paralysis Agitans, as just described after Parkinson, represent, it should be observed, the most aggravated examples of the disease. And the subject of it being advanced in years, the effects of senile decay are necessarily mixed up with the other symptoms. Indeed, this kind of Paralysis Agitans seems to induce and to terminate in general failure of the system. But the course of the senile disease is not always so deplorable. Many cases of the simple or non-procursive form remain stationary for an indefinite period, and never reach the ultimate stages.[1] A few exceptional cases have been cured. The procursive Paralysis Agitans also occasionally exhibits an arrestment, or, at least, extreme protraction of its course. The fatal forms seldom occupy less than ten years. At the same time, Parkinson's account, drawn directly from nature, represents, without exaggeration, the slow, continual progress and the fatal results of the senile Paralysis Agitans in its full development.

One very important fact, observed in nearly all cases of Paralysis Agitans, is that the cutaneous sensibility is not affected, either in regard to pain, touch, or temperature. The sensory powers, indeed, persist remarkably even amid the general failure of nervous energy in the later stages of the disease.

The *Unilateral*[2] or "*Hemiplegic*" variety of Paralysis Agitans, first noticed by Marshall Hall, presents no essential difference from the bilateral (paraplegic) disease, just described. The limbs on one side are agitated with violent chronic tremors, while those on the opposite side are entirely unaffected, or exhibit only a slight and occasional tendency to tremble. The affection is not less severe than the bilateral, into which it probably passes. There is no complete case of this form, from beginning to end, on record. In one instance, lately under observation,[1] there was no disturbance of equilibrium, no festination, and it does not appear that this symptom has been met with in the unilateral disease. The progress is, probably, the same as in the other senile forms.

To sum up: Paralysis Agitans senilis occurs in advanced life, past fifty, usually past sixty years of age; it is combined with and appears to hasten senile decay. Two forms of it are distinguished, the *Simple* and the *Procursive* (festinans): it is usually very protracted, lasting ten years or more, and is, with rare exceptions, incurable.

We pass now to those forms of the disease which occur earlier in life.

I. B. Non-senile forms of Paralysis Agitans, occurring under fifty, without fatal tendency, and sometimes admitting of cure. They are much rarer than the senile forms of the disease.

1st. *Paralysis Agitans simplex, non-senilis (sine festinatione).*—This form resembles the senile disease in regard to the tremors, differing chiefly by the absence of the signs of senile decay. The jactitations affect the same parts, the limbs, head, and trunk, exempting the muscles of the eyeball, and usually also the facial muscles of expression.[2] They come on in paroxysms excited by attempts at voluntary movements, or by mental emotion; they subside or disappear during rest, and they cease entirely during sleep. In severe cases they are extremely violent. The limbs and the whole body quiver and shake convulsively in the most extravagant manner. The patient cannot stand without support; in walking he jerks and staggers as if moved by broken springs, and is like to be pitched to the ground at every step. He cannot dress or feed himself; if his limbs are approximated, they knock against each other; and if his hand

[1] Dr. Maclachlan, out of a large number of instances among the inmates of Chelsea Hospital, found that the affection often had little or no influence in shortening life. None of his cases had occurred below 55, the majority between 65 and 70, yet in many the disease lasted upwards of 30 years. An in-pensioner, in his 107th year, had been affected since he was 60.—Page 213.

[2] "Uni-" and "bilateral" are preferable to "hemi-" and "paraplegic," being less apt to lead to confusion with ordinary motor paralysis.

[1] Patient of Dr. Warburton Begbie, to whom the writer is indebted for an opportunity of examining it.

[2] Marshall Hall mentions a male, aged 28, with Paralysis Agitans of right arm and leg, who presented a "peculiar rocking motion of the eyes, and a degree of stammering and defective articulation." Certainly, however, tremors of the oculo-motor muscles are very rare in Paralysis Agitans; singularly so, since nystagmus by itself is not uncommon

is brought near the chest or the face, it strikes upon them in a series of quickly repeated blows. When the shaking arises unexpectedly, the patient may hurt himself, knocking his head against a wall, &c. It is rare, however, for the tremors to exhibit such extreme vehemence; more commonly they exist only to the extent of rendering the execution of regular movements impossible, interfering completely with the patient's usefulness. It is further observed that, although sometimes as severe, or more so, than in the senile forms, the tremors are less continuous and never occur during sleep, although they may come on as soon as the patient awakes or turns in bed. The special distinctions of the non-senile Paralysis Agitans are therefore: 1st. There is no disturbed equilibrium; no disposition to fall or hasten forwards or backwards.[1] 2d. The diminution of voluntary muscular power is slight; sometimes none is observable. Tested by the grasp of the hand, by the dynamometer, or by the ability to lift weights or bear burdens, the muscular force is often found equal to the normal standard; sometimes the shaking arm appears stronger than the sound one. Yet the patient himself usually complains of diminished strength, and he has less ability to sustain prolonged exertion. If the disease progresses, the muscular debility increases. This is an important sign, for increasing muscular weakness is of unfavorable prognosis. 3d. There is no fatal tendency. The affection is extremely obstinate, often incurable, but the general bodily health is not impaired, and the duration is indefinite if no complication supervene. A patient, lately seen, aged sixty-six, was first affected at twelve years of age; the tremors have entirely unfitted him for labor during his whole life; yet even now his appetite and bodily health are excellent. Similar cases are not very rare; but, being regarded as examples of incurable infirmity,[2] they were not brought under the notice of the physician, and probably suffer irremediably from neglect of care at the earliest stages. 4th. Occurring in middle life (twenty-five to fifty), however formidable in appearance, it is susceptible of amelioration, and sometimes of cure. A case was cured by Elliotson by the use of carbonate of iron (1827), and several examples of recovery have been recorded under different methods of treatment.[3] Others, however, have resisted treatment altogether. To account for the fact that some cases are curable while others are not, it has been supposed that the former are functional and the latter organic.

To the slighter and more curable forms of Paralysis Agitans belong the *Hysterical Paralysis Agitans*, which exhibits the usual tremors (sometimes an approach to the festination), and is accompanied by hysterical symptoms and usually some disorder of the general health. Though often obstinate, it is entirely free from danger, and is usually cured when the general hysterical condition is removed by judicious treatment.

The *Intermittent Paralysis Agitans*, in which tremors of the limbs, lasting five to six minutes, recur twice or thrice in an hour, appears frequently to depend on intestinal worms in young subjects, or is a variety of the hysterical or reflex forms. It is curable.[1]

Reflex Paralysis Agitans may depend upon disordered primæ viæ, and be cured by appropriate remedies (Sauvages, Tremor a saburrâ). Perhaps derangements of other internal organs may exert a similar effect; as also external wounds and injuries. An interesting case, caused by the latter, was observed by Dr. Door, and related by Dr. Haas (1852). A healthy girl of nineteen received a splinter under the nail of her right thumb, on the extraction of which violent pain, and soon after Paralysis Agitans, came on in the right leg, subsequently spreading to the right arm and the whole body. The tongue and speech became affected; the general health suffered; the face had a stupid expression; and she dragged the legs in walking. She recovered completely.

Lastly, it appears from the important cases described by Dr. Hennis Green (a few similar to which are mentioned in older authors) that nervous tremor of the nature of Paralysis Agitans may occur *in children* (age, eleven to thirteen), and is in them speedily curable. Of the three cases which he reports, two recovered in about a month; the third died of pulmonary consumption, and no trace of lesion was found in the brain and spinal cord.

The non-senile Paralysis Agitans is particularly apt to be mistaken for chorea; it sometimes assumes the unilateral form.

---

[1] At least no case of non-senile Paralysis Agitans, accompanied by festination, is known to the writer.

[2] Often objects of charity, or paupers in and out of workhouses. The disease, however, affects the rich as well as the poor.

[3] Trousseau, case of St. Vitus's dance (properly Paralysis Agitans) in 1843; Russell Reynolds; Handfield Jones; Sanders, case of dystaxia or Pseudo-paralysis Agitans. This patient, after a year, was able to return to light work, the tremors having nearly ceased. Dr. Alexander Turnbull, R. N., has recently communicated to the writer two cases which came on after ague at Panama—the one recovered after a year, the other was still under treatment.

[1] See Gowry's case.

2d. *Paralysis Agitans Toxica.*—Various poisons occasion debility and tremors. When these symptoms arise from the abuse of alcohol, tea, coffee, tobacco, or opium, they rarely occur except when the parts are used, and hence are simple tremors only; but if they take place also during repose, they belong to this sort of Paralysis Agitans. A strongly marked and very characteristic form of the curable Paralysis Agitans is brought on by inhaling fumes of mercury, and, though less frequently, by certain other metallic poisons (tremor metallurgorum—tremblement mercuriel). This will be described elsewhere. (See Tremblement Métallique.)

The other kinds of tremor mentioned by medical writers are either unimportant or symptomatic of other diseases; they present an interest, as related pathologically to the morbid condition probably existing in idiopathic Paralysis Agitans. Such are the tremors from bodily weakness and mental emotion: tremor senilis, which is evidently closely allied to and may pass into Paralysis Agitans senilis; febrile tremors and rigors (attended by a sense of cold, which is never present in Paralysis Agitans), analogous probably to the toxic forms of Paralysis Agitans; tremor or subsultus tendinum, which exhibits the same spasmodic jerking of the muscles as Paralysis Agitans; lastly, the tremors in diseases of the brain and spinal cord (hydrocephalus, parasites in cerebrum, myelitis, ataxia, tumors, &c.) are symptomatic, and, as previously explained, distinct from idiopathic Paralysis Agitans.

II. *Local Paralysis Agitans* attacks a single part, most frequently the head and neck, or the arm, or the lower jaw, and remains limited to the region affected. The tremors occur occasionally, seldom constantly, during the day; they cease at night; they are excited or aggravated by exertion or emotion. Usually free from danger, local Paralysis Agitans is regarded, like the spasmodic tics, as an infirmity or bad habit rather than a disease. It is at the same time very obstinate, in fact usually incurable. It is important to distinguish the idiopathic Paralysis Agitans which continues local from that which is the precursor of the progressive general disease, or which may be symptomatic of a tumor or other lesion of the nerve centres. At first the distinction may be impossible; but the history and course of the affection determines the diagnosis. Whenever the tremor has continued for some time unattended by any concurrent serious symptoms and strictly confined to one part, experience warrants the conviction that the morbid action has been exhausted in a circumscribed area, and that no extension of the disease need be feared. It is, indeed, singular that after a few years the local exhibits no tendency to pass into the general disease.

A few remarkable cases have occurred of Paralysis Agitans restricted to the lower jaw and tongue; in some distressing instances all remedies proved unavailing. The pathology has not been ascertained, and probably the severer cases were not idiopathic, but were symptomatic of some grave disease of the nerve centres. Of the latter kind an interesting case is recorded recently by Leyden, in which Paralysis Agitans of the right arm was found associated with sarcoma in the left optic thalamus. The more serious symptomatic kinds are distinguished from the idiopathic by the more dangerous character of the symptoms, among which are the signs of centric nervous lesion, such as motor and sensory paralysis, &c. In respect of pathology and treatment, the local resembles the general Paralysis Agitans.

CAUSES. — These are frequently obscure, but it is probable that conditions productive of debility or atrophy of the motor nerve centres occasion the idiopathic Paralysis Agitans. The results of experience are as follows: *Predisposing Causes.*—1. Age is of primary importance, both in causing the disease and aggravating it. 2. Hereditary and parental influence is indicated in some cases.[1] 3. The male sex is almost exclusively the subject of general Paralysis Agitans; the hysterical forms and local tremors of the head being met with in the female. *Exciting Causes.*—1. Violent muscular exertion is a frequent cause, as also—2. Injuries, especially falls; also wounds, &c. 3. Excessive mental exertion, and particularly—4. Violent emotions, as terror or fright, which have sometimes produced the disease suddenly. 5. Venereal excesses have been alleged. 6. Exposure to cold and wet, as lying on damp ground, especially when giving rise to—7. Rheumatism, which was noted by Parkinson, and has a decided causal relation to Paralysis Agitans; also—8. Ague.[2] In certain cases Paralysis Agitans appears to have followed—9. Fever (typhoid and various exanthemata) and—10. Syphilis. 11. Intestinal worms sometimes give rise to it in young subjects, occasionally modified

[1] Sauvages: "Mulier gravida, quæ maritum subito peremptum exhorruerat, genuit filium tremore miserando correptum." Most knew a whole family in which it was hereditary. Lebert refers to females who, in successive generations, being otherwise in good health, presented tremors of the head at the climacteric age.

[2] Romberg, Maclachlan, Turnbull. No relation to gout has been alleged.

into a periodical or intermittent type. 12. Disordered primæ viæ (tremor a saburrâ). 13. Suppressed itch will hardly now be admitted.[1] 14. Alcohol, opium, tobacco. 15. Certain poisons, particularly mercurial vapors, cause the Paralysis Agitans toxica. While these are the causes of the idiopathic disease, the symptomatic Paralysis Agitans, as already stated, may occur, combined with other characteristic signs of nervous disease, in various lesions and tumors, &c., of the brain and spinal cord.

Diagnosis.—Idiopathic Paralysis Agitans is sufficiently characterized to be of easy recognition; but its relations to allied affections are important. 1st. It is distinguished from the other species of the class tremors, because in Paralysis Agitans the trembling occurs not only during action, but even when the parts are not in use and are supported (spasmodic, tremor coactus). Tremor senilis, which most resembles it, may pass into Paralysis Agitans, when the tremors which begin during action continue after it has ceased: the tremors usually at the same time increase in intensity from trembling to jactitation. 2d. The different kinds of Paralysis Agitans are distinguished: the *simple senile* form by occurring in advanced life, by its progressive course, association with general decay of the system, and fatal issue; the *procursive* (festinans) *senile* form presents in addition the disturbed balance in locomotion; the *simple non-senile* form occurs in middle age or under, is often stationary in its progress, may be ameliorated or cured, and is not accompanied by disordered equilibrium; the *hysterical*, *remittent*, *reflex* forms, and that occurring in children, &c., are known by their special circumstances; the *toxic Paralysis Agitans* is recognized by the cause, and by the concomitant effects of the poison—in the mercurial tremors the tongue and mouth are usually and early attacked, which is not the case in ordinary Paralysis Agitans. 3d. The relations to common chorea, both of difference and resemblance, are instructive. Chorea occurs chiefly in the young before puberty; Paralysis Agitans attacks the middle-aged, and especially the old. The gesticulations in chorea are jerking, irregular movements, changing frequently, and dissimilar on the two sides; the tremors or jactitations in Paralysis Agitans consist of to-and-fro oscillations of the part, due to the brief alternate action of antagonist muscles; they continue long unchanged, and are usually the same on the two sides. Chorea specially attacks the female, Paralysis Agitans the male sex. With attention, therefore, the diseases are not difficult to distinguish. But their points of resemblance are striking. Both consist of involuntary, spasmodic movements, rapidly repeated, and not under the control of the will, while the voluntary motor power persists in the affected parts, although it is often enfeebled, the debility sometimes amounting to paralysis.[1] Both are often caused by fright, and by rheumatism (although no relation seems to exist between Paralysis Agitans and heart disease); in both, when fatal, no visible lesion may be discovered. On the other hand, chorea is nearly always curable in a comparatively short period; while Paralysis Agitans, although susceptible of cure in younger individuals, is a peculiarly obstinate disease, and is incurable in old persons, in whom it associates itself with senile decay. 4th. The irresistible movements forward or backward in Paralysis Agitans present great affinity to the like symptoms met with in "leaping ague," and certain forms of tarantism and abnormal chorea, and which are also observed in connection with lesion of the cerebral peduncles or other parts of the encephalon.[2] But these affections are not usually attended by tremors, and the history and special concomitant symptoms are sufficiently distinctive. The difference of Symptomatic from Idiopathic Paralysis Agitans must always be kept in view. 5th. The same remarks apply to the discrimination of idiopathic Paralysis Agitans from certain cases of locomotor ataxia and chronic myelitis, &c.—affections which it often closely resembles in the progressive character of the symptoms, and in the spasmodic nature of the movements. But these spinal diseases are, in addition to their clinical history, especially distinguished by the presence of decided motor, and mostly also of sensory paralysis; while in idiopathic Paralysis Agitans the sensibility is remarkably exempted, what is there called paralysis being only a failure of vigor. The diagnosis is very important, and only difficult because the occurrence of tremors as a complication in various organic nervous diseases may mislead, if the difference of Symptomatic from Idiopathic Paralysis Agitans be not attended to. 6th. In a similar manner, Paralysis Agitans is distinguished from beriberi, raphania, &c.

[1] Mentioned by Canstatt.

[1] Both are sometimes unilateral.

[2] Marshall Hall remarks the similarity of certain symptoms in Paralysis Agitans to the effects observed by Serres in diseases of the tuber annulare and tubercula quadrigemina. The irresistible movements—forward, backward, lateral, whirling, rolling, somersault, &c.—in experimental lesions of the brain in animals (Fodera, Magendie, Flourens, &c.) have thrown much light on the subject.

Complications. — These are rare in idiopathic Paralysis Agitans; the health usually continuing good till senile decay begins. Apoplexy, hemi- and paraplegia sometimes occur, but not often. Common chorea has, in some cases, been found associated with Paralysis Agitans;[1] usually, however, the disordered movements called chorea have been only exaggerated examples of the shaking disease. On the other hand, as already remarked, symptomatic Paralysis Agitans may complicate many diseases of the brain and spinal cord.

Pathology and Morbid Anatomy. —*Tremors* are generally admitted to be a sign of weakness in the nerve centres, and are ascribed to defective and interrupted discharge of nervous stimulus. But two kinds of tremors have been distinguished:[2] *first*, simple or passive tremors, which occur during a voluntary act, and cease with it, being evidently due to want of power only (τρόμος); *second*, spasmodic, clonic, or active tremors, which take place even during rest when the parts are supported and unemployed (παλμός, tremor coactus, palpitatio): these are short, alternate, clonic convulsions of antagonist muscles, and imply some irritation in the motor nerve centres. Although these two kinds are allied and pass the one into the other, yet the distinction is important, and furnishes the ground of diagnosis; the spasmodic, not the passive tremor being characteristic of Paralysis Agitans. *The disturbance of equilibrium* is no doubt owing to an affection of a different part of the nervous system from the tremors, since these may exist, even generalized, without it. The cerebral or cerebellar peduncles or the pons Varolii, are most probably the seat of lesion. But while the locality is different, the association with tremors shows that the morbid action is probably the same in both. The general opinion is that the tremors are due to an affection of the spinal cord, the disturbed equilibrium to an extension of the morbid action within the cranium. Hence some writers speak of *Paralysis Agitans Spinalis*, consisting of tremor and muscular debility, and *Paralysis Agitans Cerebralis*, in which the disturbance of equilibrium is superadded.[3] There is no vertigo nor distortion of the eyeballs, as in lesion of the base of the encephalon. The disturbed equilibrium seems due to weakness of one set of muscles (*e. g.*, extensors), and perhaps spasmodic action of their antagonists (*i. e.*, flexors).

Morbid Anatomy, which formerly gave only negative, has lately afforded indications of positive results. The facts are as follows:—

1st. In many instances of idiopathic Paralysis Agitans no lesion of the cerebro-spinal axis can be discovered by our present means of investigation. In these cases, therefore, the disease may be regarded as *functional* or *dynamical;* and it may be presumed to depend, (*a*) on impaired generation of nerve energy, due to some unknown conditions; (*b*) alteration of vascular supply, either congestion, or, as late researches on the pathology of convulsions suggest, anæmia, *i. e.*, deficient or interrupted vascular supply;[1] possibly also an œdematous condition of the nerve centres might cause the symptoms; (*c*) molecular physical or chemical changes, which we may certainly assume in the toxic forms (mercurial tremors). The functional are especially the curable forms of the disease.

2d. In more inveterate, especially senile cases, Paralysis Agitans appears to depend on a discoverable lesion; namely, an *atrophic* condition of the spinal cord, pons Varolii, crura, or medulla oblongata (*atrophic* or *organic Paralysis Agitans*). This atrophy has been found in several careful dissections, and it certainly coincides with and would explain the chief features of the disease—namely, its obstinacy or incurability, without immediate danger to life; the progressive course; the impaired strength and muscular debility (paralysis); the occurrence in old age, after violent exertion and emotion, under conditions of premature senility, &c. In addition to simple atrophy, with serous accumulations, autopsies have revealed in different parts of the spinal cord, medulla oblongata, and pons an indurated condition (sclerosis), with patches of gray or gelatinous degeneration, due to the new formation of connective tissue, which compresses and atrophies the proper nerve structures.[2] Since a similar condition, involving extensively the posterior columns of the cord, is the chief lesion found in progressive locomotor ataxia (tabes dorsalis), some relation is established between it and Paralysis Agitans.[3] It is curious that Parkinson (from the report of a case not seen by himself) drew attention to the induration and enlargement of the upper part of the medulla spinalis, oblongata, and pons, as the probable morbid condition in Paralysis Agitans, and supposed it might be due to simple inflammation, or rheumatic

[1] Maclachlan, p. 216.

[2] Distinction first clearly drawn by Sylvius, previously indicated by Galen.

[3] Remak.

[1] Marshall Hall, Kussmaul and Tenner, Brown-Séquard, &c.

[2] Bamberger, Skoda, Oppolzer, Lebert, &c.

[3] Also with tetanus, probably chorea, and with progressive paralysis of the Insane. See Rokitansky, Ueber Bindegewebs-Wucherung im Nervensysteme. Wien, 1857. Cruveilhier, &c.

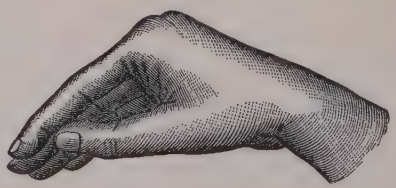
or scrofulous affection of the nervous substance or membranes. The sclerotic atrophy does not seem to be of inflammatory origin, although, according to Rokitansky, it is preceded by congestion. In the early stages, there may be softening instead of induration. Rheumatic and other morbid diatheses may probably dispose to it. Degeneration of the blood-vessels may possibly be connected with it.[1] The pathogenesis of atrophy of the nerve centres, however, has not yet been fully investigated; and, although highly probable, it cannot yet be positively affirmed, that Paralysis Agitans depends upon atrophy, simple or sclerotic, of certain parts of the cerebro-spinal axis. In regard to symptomatic Paralysis Agitans, the tremors are ascribed to the atrophy of the nerve substance surrounding the tumor or other principal lesion.

But whether the disease be functional or organic (atrophic) in its nature, it clearly affects the motor centres only, exempting the sensory and the intellectual; and, further, the morbid state of the nerve centres implies not only diminished power, but some condition of spontaneous irritation, giving rise to the spasmodic jactitations even during rest. Probably the degenerative molecular changes in the nerve structures may occasion a disturbed equilibrium and consequent irregular discharge of nerve stimulus. The morbid process is presumed to begin usually in the cervical portion of the spinal cord, since the arms are apt to be first affected, and the disease presents the bilateral type. But the occurrence of the unilateral form, as well as the fact that the limbs are much earlier affected than the trunk, shows that the possibility of the cerebral centres of motion being sometimes attacked should not be overlooked. The disturbed equilibrium probably ensues when the parts in the vicinity of the pons Varolii are involved, and the extension to the medulla oblongata is indicated by the impaired speech, deglutition, &c., which supervene in the advanced stages of the disease.

[Since this article was written, the distinction urged by Charcot has been generally accepted, between Paralysis Agitans and disseminated (multiple, cerebro-spinal) sclerosis. Of the two kinds of tremor above described, which may be called, the one passive and permanent, and the other volitional, the former is peculiar to Paralysis Agitans; the latter occurs in disseminated sclerosis. That is to say, in the first named disorder there is trembling at all times, whether the patient be moving or reclining, so long as he is awake. In the second disease, tremor occurs only when some muscular action is attempted; then the movement of the muscles used becomes irregular, with a coarser tremulousness than in shaking palsy (see Multiple Sclerosis, under INDURATION, in this volume).

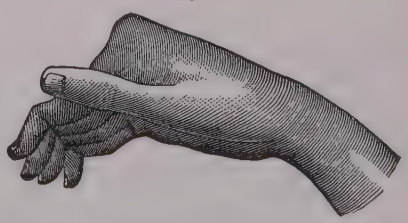
Charcot analyzes[1] the morbid appearances of the cases above referred to, reported by Bamberger, Lebert, Skoda, Parkinson, and Oppolzer; and believes that some of them were examples of disseminated sclerosis, not of Paralysis Agitans. Yet, in Charcot's account of disseminated sclerosis, it is admitted that morbid anatomy has not made clear what location of disease explains the trembling which is one of its characteristic symptoms. Erb,[2] also, observes, that the two kinds of tremor, in some cases, "exist side by side." This pathologist, while preferring to regard such a combination as indicating the presence of two different diseases, rather than as evidence of the non-essentiality of the distinction between them, adds: "We must wait for more accurate observations, however, before passing finally on the correctness of this view."

Fig. 38.



The writing hand. Habitual attitude of the hand at a somewhat advanced stage of Paralysis Agitans. (Charcot.)

Fig. 39.



Digital deformation, simulating that of primitive chronic articular rheumatism. (Charcot.)

In three cases examined *post-mortem* by Charcot,[3] the appearances common to all

[1] In Skoda's case the nerve elements were destroyed in some parts of the brain, the pons, and medulla, by embryonal connective tissue; the *vessels were obliterated;* the muscles were in a state of fatty degeneration; the neurilemma of the nerves of the upper extremity was thickened. The thickened neurilemma has been observed in other cases.

[1 Lectures, &c., Lect. v.]

[2 Ziemssen's Cyclopædia, vol. xiii. Multiple Sclerosis.]

[3 Joffroy, Société de Biologie, 1871.]

were the following: *a*, obliteration of the central canal of the spinal cord by proliferation of the epithelial elements which line the ependyma; *b*, proliferation of the nuclei which surround the ependyma; *c*, pigmentation of nerve-cells, most marked in Clarke's (posterior gray) columns. Two of the cases had also a multiplication of amyloid corpuscles; one, a sclerosed patch on the posterior surface of the bulbus rachidicus.[1] The language of Charcot appears to be still appropriate: "The special lesion of Paralysis Agitans remains to be discovered."—H.]

Should future researches confirm the results above indicated, there would then exist a secure basis of morbid anatomy for the distinctions which authors have indicated clinically, of idiopathic Paralysis Agitans into functional and organic: the latter with, the former without, atrophy of the nerve centres; the latter mostly incurable, the former admitting of cure.

The *Prognosis* is unfavorable, but depends upon the age of the patient and the particular form of the disease. When fully established, idiopathic Paralysis Agitans is an obstinate,[2] and in the aged, with rare exceptions, an incurable disease. But, unless mixed with signs of senile decay, Paralysis Agitans does not endanger life, and its course is often indefinitely protracted. As a rule, it is obstinate in proportion to the age of the subject, and is fatal only in the old. The distinction of curable and incurable, functional and organic (atrophic), has been already sufficiently indicated. It need only be added that mere violence of the jactitations is no evidence of incurability; slight tremors are frequently the most obstinate;[3] it is the persistence of tremors during absence of voluntary effort, and especially during sleep, the occurrence of disturbed equilibrium, and symptoms of senile decay which are of serious import. Disturbed equilibrium is apparently incurable in itself, as well as of bad augury for the disease generally. The supervention of convulsions, apoplexy, motor or sensory paralysis, indicates more immediate danger to life.

In the Registrar-General's Reports for England and Wales, from 1855 to 1863 inclusive, 205 deaths from Paralysis Agitans are recorded, 129 being males, and 76 females; on an average about 14 males and 8 females annually. Of these 205, 189 were above 55 years of age; nearly half, viz., 91, occurring at 65 years; only 16 below 45 years, one death taking place at 20 years. It may be doubted, however, whether the cases fatal below the age of 45 were true idiopathic Paralysis Agitans; more probably they were examples of spinal or cerebral disease accompanied by tremors, *i. e.*, by symptomatic Paralysis Agitans.

Of the *Modes of Termination*, the principal in the senile disease is by general decay of the system. Sometimes life is cut short by the intercurrence of the usual diseases of old age. Trousseau states that in three cases which he traced to the end the patients all died of pneumonia. The non-senile forms of Paralysis Agitans are not known to have any special mode of termination.

TREATMENT.—The modes of treatment and remedies employed are numerous, but few have been attended with success. Allowance must be made for the form of the disease; the senile being mostly incurable, the non-senile obstinate, but susceptible of relief or cure. The physician must keep in mind the propriety of abstaining from remedies in inveterate cases, after a fair trial has been given to them: a fruitless perseverance would only injure the general health and excite false hopes. At the same time, general hygienic measures are always beneficial, and by their means alone, the symptoms may be alleviated, and life prolonged to advanced age.

The methods of treatment are:—

1. *Antiphlogistic.*—This used to be commonly practised on the supposition of the congestive or inflammatory nature of the affection of the spinal cord. In some cases, in an early stage, it seems to have done good; purgatives, indeed, in judicious moderation, are useful in all cases, and they cure the forms depending on disordered primæ viæ (tremor a saburrâ). The means employed were: Venesection general or local, purgatives, diaphoretics, mercury; blisters, setons, cauteries actual and potential, moxas, &c., to the spine; frictions, stimulant embrocations, hot baths, &c. In the majority of cases, however, this practice did no good or did positive harm. The treatment now preferred, especially in chronic cases, is—

2. *Tonic*, general and nervine. Hygienic regimen, nutritious but not stimulating diet; little wine; rest or moderate

[1] *Bulbe rachidien*, the lower part of the medulla oblongata.]

[2] "Morbus valde pertinax," Juncker. Comparing it with apoplexy and motor palsy, &c., he says it is inferior to them in danger to life, but equals them in reference to treatment.

[3] Dr. Russell Reynolds, in a letter, June 1, 1865, says: "From what I have seen of a large number of cases, I am led to believe that there is a most important difference between those cases in which there is trembling only and those in which there is clonic alternate spasm. In the latter the prognosis is very much more favorable than in the former." The age of the patient and stage of the disease being, of course, taken into account.

exercise, light gymnastics. Excessive exertion is injurious, and many cases of simple Paralysis Agitans are aggravated by the patients when poor being compelled to work, or, when rich, endeavoring by forced exercise to overcome the debility in which they suppose their disease exists. *Subcarbonate of iron* has been a noted remedy in consequence of Elliotson having cured cases by it in persons under 50; it failed in older patients. Quinine, zinc, arsenic, nitrate of silver, chloride of gold; *strychnine*, which has apparently cured some, and failed in other cases; *ergot*, said to have been beneficial; iodine and bromide of potassium, balsams, *oil of turpentine*, sarsaparilla, quassia, colchicum, &c.; Iceland moss; mineral-waters, sulphurous or chalybeate; sea or mountain air, the milk cure, &c.

3. *Narcotics and Calmants.* — *Opium*, *belladonna*, *henbane* (ʒss of Tinct. thrice daily, used successfully in functional Paralysis Agitans by Dr. Handfield Jones); stramonium; *chloroform* stops the tremors at the time, but does not appear permanently beneficial; ether, musk, camphor; *veratrin*, externally or internally, reported successful in a case by Volz; valerian and valerianate of zinc; Calabar bean, tried without success by Dr. J. W. Ogle.

4. Baths have been much resorted to, sometimes with success, often without effect. Warm sulphur baths, especially of liver of sulphur, has been specially recommended. Simple warm baths with cold douches (Romberg). Vapor baths, Russian baths, and baths of gelatine, firtops, mud, even animal baths, have been used in Germany. The water-cure, cold affusions; sea-bathing, which rendered one case stationary (Lebert); brine baths, ice baths, first tepid, gradually made colder. It should be remembered that some of these, especially the cold-water cure, are not free from danger, and require proper caution in old persons; sometimes they aggravate the disease.

5. *Electricity and Galvanism.*—Partially successful in previous experience, electricity was found to produce no improvement in four cases observed by Gull. The interrupted galvanic current appears also to have been of little service; but the continuous current recommended by Remak proved successful in his hands with a patient aged 60, and others were benefited by it. In a man, aged 57, the disease, well marked though recent, was cured in this manner by Dr. Russell Reynolds. After five applications of Pulvermacher's chain of 120 links, daily, for one hour, the spontaneous jactitations completely ceased; the same treatment, continued every second day, completed the cure in about a month. In other instances, this means has not produced such favorable results, but it deserves a persevering trial in all cases.

On the whole, good hygienic rules, attention to any special indications, gouty, rheumatic, &c.; regulation of the primæ viæ; the administration separately or combined of general and nervine tonics, and calmants, and the judicious use of the continuous galvanic current, are the measures chiefly to be recommended. Depletion and counter-irritation are seldom required, and would in most cases be highly injurious. Time must in all cases be allowed, for the affection is obstinate. In the confirmed senile forms, we may be satisfied with arresting or mitigating, but must not expect to cure the disease.

References in chronological order. When marked °, the originals were not obtainable.

Hippocrates, Coan prognost. and Prorrhet., Ed. Kühn, i. pp. 159, 161, 246, 288; Epidem. iii. § 3, 4th case, p. 298, &c. *Celsus*, lib. iii. c. 27, and lib. i. c. 9. Galen, on Tremor, Palpitation, Convulsion, and Rigor, Ed. Kühn, vii. 584; Scelotyrbe, Definitions, xix., 427, § 293. *Oribasius* (A. D. 360), Ed. Bussemaker and Daremberg, iii. 209, On Trembling (from Galen). *Paul Ægineta* (6th or 7th century), Book iii. sect 21, On Trembling, Syd. Soc. transl. i. 407. *Diemerbroeck* (1652) Disp. de Paral. et Tremore. *Tulpius*, Obs. Med. i. 12, Tremor periodicus, 1672. Sylvius de la Boe, Op. Med., Ed. Alt. 1680; Prax. Med. l. i. c. 42, p. 291; De Spirit. Animal. per nervos. motu læso, § 5, and 25, Coactus Tremor, &c. *Bonet*, Sepul. Ed. Alt. 1700, l. i. sect. 14. On Stupor, Torpor, Tremor, &c. Obs. 6—11, pp. 346–9. Juncker, Conspect. Med. 3 Ed. 1737, Tab. 115; De Tremore artuum, p. 886. Gaubius, Inst. Path. Med. 1758, Spasmus, § 751; Paralysis, § 757. Van Swieten, Comment. 2d Ed. 1749, ii. § 627, Tremor febrilis; cases of Paralysis Agitans at p. 181, Vidi in hac urbe virum, &c. Sauvages, Nosol. Meth., Ed. ult. 1768, i. p. 557, § xiv. Tremor, and p. 590, 4 xxi. Scelotyrbe, festinans et instabilis. *Linnæus*, 1763, Gen. Morb. 144, Hieranosos. °*Vogel*, Def. Gen. Morb. 1764, Hieranosos. *Macbride*, Theory and Pract. of Physic, 1772, pp. 558–9, Hieranosos. *Sagar*, Syst. Morb. Sympt. 1779, Tremor, pp. 430–2. *Cullen*, Synops. Nosol. Meth. 1785, Gen. 43, Paralysis-Tremor. *Kirkland*, Comment. on Apop. and Parol. Affect. 1792, pp. 102 and 122. *Heberden*, Comment., Ed. Alt. 1807, c. 91, De Tremore, p. 371. Parkinson, Essay on the Shaking Palsy (Paralysis Agitans), London, 1817. *Art. Scelotyrbe* in Dict. des Sc. Méd. t. l. pp. 134, 1920. *Cooke*, Nerv. Diseases, 1821, ii. p. 207. Mason Good, Study of Med. 2d. Ed. 1825, iv. Synclonus Tremor, p. 458, and Syncl. Ballismus, p. 473. Elliotson, Med.-Chir. Trans. xiii. p. 240, 1827; Lancet, June 4, 1831, p. 290; Ryan's Lond. Med. and Surg. Journ. 1832, ii. 605; Lond. Med. Gaz.

xi. p. 532, Jan. 1833; Prin. and Pract. of Med. by Rogers and Lee, 2d Ed. 1846, ch. xi. p. 689. Cruveilhier, An. Path. t. ii. 32, Liv., Pl. 2, fig. 3, p. 19, 1829. *Gowry*, Case of Par. Agit. Intermittens (cured), Lancet, 1831, p. 651. Todd, in Forbes' Cyc. of Pract. Med. 1834, iii. 259, and Clin. Lect. 2d Ed., Lect. 45, p. 764, &c. *Most*, Encyc. d. Med. Prax. 1837, ii. 555. *Gibson*, on Spin. Irrit. case 5, Lancet, ii. 1838–9, p. 568. Romberg, Nerv. Diseases, 1st Ed. 1840; 3d, 1857; Syd. Soc. transl. 1853, ii. 233. Marshall Hall, Dis. and Derange. of Nerv. Syst. 1841, p. 320. *Thompson*, Secale cornutum in Chorea, Par. Agit. &c. Lancet, Jan. 29, 1842, p. 616. Graves, Clin. Med. 1843, 1st Ed. p. 714; Ellen Davis's case, &c. *Canstatt*, Pathol. or Handb. der Med. Klinik, 3d Ed. Bd. iii., 1 Abth. pp. 444–5, 1843. ° *Trousseau*, Des. Prép. de Noix vom. dans la Danse de St. Guy, Journ. de Méd. par Beau, June, 1843, p. 161, reported as Par. Agit. in Canstatt's Jahrb. 1843, Bd. ii. p. 99, § 35. *Watson*, Prin. and Pract. of Phys. Lect. 38, 1st Ed. 1843. Hennis Green, cases of Nervous Tremor in Children, Prov. Med. Journ. No. 178, Lond. Feb. 24, 1844. ° *Volz*, in Heidelb. Annal. xii. 2, 1846, reported in Schmidt's Jahrb. liii. 37. ° *V. Brunn*, Chron. Zittern, in Caspars Wochensch. No. 40, rep. in Canstatt's Jahrb. 1846, ii. 70. ° *Rudder*, Chorea with Parox. of Scelotyrbe, Ann. Soc. Méd. de Gand, 1848, ii. rep. in Canst. Jahrb. 1848, ii. 48. Roth. Hist. de la Musculation irrésistible ou Chorée Anormale, Paris, 1850. ° *Basedow*, Stabilitats-neurosen. Casp. Wochensch. No. 33, rep. in Canst. Jahrb. 1851, iii. 79. ° *Seitz*, Deutsche Klinik, No. 46, and ° *Haas*, Nassau Med. Jahrb. Hft. ix. rep. in Canst. Jahrb. 1852, iii. 92. Gull, Value of Electricity as a remedial agent, Guy's Hosp. Rep. 2 Ser. viii. 134–6. Paget, case of involuntary tendency to fall precipitately forwards, with remarks, Med. Times and Gaz. Feb. 24, 1855. ° Bamberger, Beob. üb. Hirnkrank, in Verhand, d. phys. Med. Gaz. z. Wurtzb. Bd. vi. 283, rep. in Canst. Jahrb. 1855, iii. 73. Hasse, in Virch. Handb. d. Spec. Path. u. Ther. 1855, Bd. iv. 1 Abth. pp. 301 and 306–7. Russell Reynolds, Diag. of Dis. of Brain, &c. 1855, p. 163; Case of Par. Agit. removed by continuous Galv. current, Lancet, Dec. 3, 1859, p. 588. Remak, Galvano thérapie, Berlin, 1858, pp. 219, 248, 447. *Copland's Med. Dict.* 1858. Cohn, Beitrag. z. Lehre der P. Agit. Wien Wochensch. Nos. 18—26, rep. in Canst. Jahrb. 1860, iii. 73. ° Oppolzer, remarkable case of P. Agit. with Autopsy, Spital-Zeit. Nos. 17, 18, rep. in Canst. Jahrb. 1861, iii. 78; also quoted fully in Trousseau, Clin. Méd. 2d Ed. ii. 219; also ° *Oppolzer* in Wien. Med. Zeit. 1862, No. 52, rep. in Canst. Jahrb. 1863, iii. 39. ° *Stofella*, case of P. A. with Autopsy, Wien Wochensch. xvii. 37, 1861, rep. in Schm. Jahrb. 113, p. 39, and in Syd. Soc. Yearbook, 1862, p. 82. ° *Skoda*, in Wien. Med. Halle, iii. 13, 1862, rep. in Schm. Jahrb. 119, p. 294, and in Syd. Soc. Yearbook, 1863, p. 100. ° *Alfred Louis*, De la Trémulence paralytique progressive, Strasb. 1862, rep. in Canst. Jahrb. 1862, iii. 66. *Lebert*, Handb. d. Pract. Med. 1863, ii. 590. Maclachlan, Dis. and Infirm. of Advanced Life, 1863, p. 212. Leyden, case of P. Agit. of right arm, with develop. of Sarcoma in 1 Opt. Thal. Virch. Arch. xxix, p. 202, 1864. Handfield Jones, Functional Nerv. Disorders, 1864, 263. *Topinard*, De l'Ataxie, Loc. Prog., forme Paral. Agit. pp. 103, 114, 117, &c. Paris, 1864. Trousseau, Clin. Méd. 2d Ed. ii. 213. Sanders, case of Dystaxia or Pseudo-paral. Agitans with remarks, Ed. Med. Journ. May 1865, p. 987. *J. W. Ogle*, Calabar bean in Par. Agit., Med. Times and Gaz. 1865, ii. 256.

[ATHETOSIS.

By Henry Hartshorne, M.D.

This term (from ἀθέτος, without fixed position) was first applied by Dr. Hammond, in 1871, to an affection of which he reported two cases in his work upon Diseases of the Nervous System. The characteristic symptom is a constant, involuntary, and more or less regular movement of the fingers or toes, on one side or both. As an occasional phenomenon, Charcot had noticed this in 1853, and Heisse in 1860.

Charcot, however, regards it as a variety of chorea, following hemiplegia; corresponding, therefore, in part, with what S. Weir Mitchell has designated as *post-paralytic chorea*. In some of the cases reported, however, no paralysis whatever has existed.[1]

Oulmont published an account of his study of several cases in the Salpétrière.[2] Other cases also have been reported; among the latest of which are one in the

[1 London Med. Record, March 15th, 1879.]

[2 Étude clinique sur l'Athétose, Paris, 1878.]

Revue Méd. Tr. et Etrangère, January, 1879, and another in the London *Lancet*, March 15, 1879. In the last-named instance, and at least once previously, post-mortem examination has been made.

The following is the description given of the phenomena in the last case, which was under the care of Dr. Sturges, in the Westminster Hospital, London :—

"The movements referred to were confined to the left side, and almost exclusively to the upper extremity. They were continuous and involuntary. When the hand was extended with the palm downwards, the index and middle fingers were slowly and gradually flexed, the ungual phalanges being first bent, then the middle, and finally the proximal. The thumb was also adducted, and either closed over the first phalanx of the index finger, or passed under the index and middle fingers, so that the ungual phalanx protruded between the middle and ring fingers. The hand was then supinated, the fingers again extended and the thumb abducted. Pronation of the hand completed the cycle. This was the type of the movement, but it was subjected to some variation, and there was at times great irregularity. When the fingers were flexed considerable force had to be exerted to extend them, but the forcible extension did not increase the subsequent involuntary movements. Patient had always to keep his nails short, or they would indent the palm of the hand. He was able by a great effort to partly control the movement, but his power in this respect was very slight. On requesting him to close his hand, the fingers being at the time extended, he was unable to do so, and the effort, although directed exclusively to the left hand, often resulted in the unconscious closure of the right. The only time the hand was really quiet was during sleep. The movements were ordinarily so constant and so little under the control of the will that the patient was not able to use his left hand for any of the ordinary purposes of lifting; even his food had to be cut up for him."

In this case there had been two fits during infancy, followed by hemiplegia of the left side, which, however, disappeared before the age of ten years, and did not return. The man died of phthisis in the hospital, and a careful autopsy was made. The whole right hemisphere of the brain was found to be distinctly smaller than the left. Atrophy of some of the convolutions had occurred, especially those of the frontal and parietal lobes. The whole of the gray substance of the right corpus striatum, and nearly all of its white substance, was destroyed. The optic thalamus, arteries and membranes of the brain, and the spinal cord, were healthy.

Oulmont has dwelt especially upon what he conceives to be the important difference between double or general Athetosis, and that which is unilateral, hemiathetosis. The latter, when there is motor hemiplegia, occurs nearly always on the paralyzed side. In the majority of cases, there is also hemianæsthesia of the same side. Permanent contraction, rigidity, atrophy, and laxity of the articular ligaments, may exist, as post-hemiplegic sequelæ, not necessarily connected with the Athetosis. This disorder is compared by Oulmont to hemichorea, and referred to a probable cerebral lesion, in the neighborhood of the fibres in front and outside of the sensory bundles at the lower part of the corona radiata (of Reil).

Gowers and McLane Hamilton[1] refuse to admit that there is clinical or pathological reason for separating Athetosis from other symptomatic or secondary affections of disordered movement, chiefly hemiplegic in origin. Notwithstanding its occasional independence of paralysis (as above seen), its place in nosology, at present, appears to be rather that of a symptom than of a disease]

WRITERS' CRAMP.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—A chronic disease, characterized by the occurrence of spasm when the attempt is made to execute special and complicated movements, the result of previous education; such spasm, not following muscular actions of the affected part when these special movements are not required.

The term "Writers' Cramp" is bad in one respect, because the symptoms it de-

[1 Nervous Diseases, p. 92.]

notes do not belong exclusively to the act of writing; it is good, and therefore retained in this "System of Medicine," because it points to the most frequent form in which the disease is exhibited, and because it has already passed into general usage.

A disease pathologically similar to Writers' Cramp may be found in the artist, and may prevent him from painting in oil; it may occur in the violinist or the pianist, and hinder the musical performances of either; it may be met with in the seamstress, or the smith, or the milkmaid, and may limit or destroy their powers of work. Wherever it is found it shows the same general features, expressed in the definition, viz. a limitation by spasm of a particular kind of movement, and of that movement only.

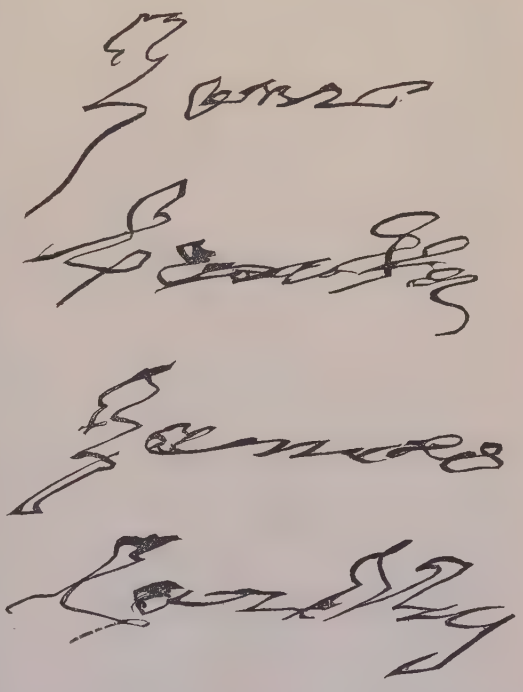
SYNONYMS.—Scriveners' Palsy; Mogigraphie; Schreibekrampf; Crampe des Ecrivains; Schusterkrampf; Melkerkrampf.

SYMPTOMS.—A slowly developed difficulty in executing a particular movement, such as that of writing or playing on a musical instrument, other movements of the same limb being perfectly easy of performance. Usually the patient feels at first some undue weariness after long exertion, a stiffness of the fingers, or an unsteadiness and uncertainty of movement, all of which immediately disappear on giving up the exertion. If writing, a man feels that his pen does not do what he intended that it should; that his handwriting looks unnatural; that he has to hold his pen more tightly than before, in order to keep it between his thumb and fingers; that it starts from its place, and often is pushed, by his first finger, over the nail of his thumb, and that he has some difficulty in getting it back to its place. A pianist makes blunders in striking chords, the fingers falling on keys they were intended to avoid: the violinist cannot control the movements of his left hand,—or can do so only by a painful effort,—the fingers running together and feeling stiff: the seamstress cannot ply her needle, but pricks her fingers, and makes her stitching irregular. In one case, under my own care, the bricklayer could not use his trowel.

At first the difficulty is slight, and may be overcome by strenuous effort; but, after a little time, if the attempt to continue or repeat the movement be persisted in, there is distinct cramp, jactitation, or tremor of the hand, and the particular performance is quite impossible. Other things may be done, but that one thing with regard to which the difficulty was first felt cannot be effected properly by any amount of exertion. A patient may not be able to write, and yet may feel no difficulty in fingering either the pianoforte or the harp.

The accompanying woodcut represents four different attempts made by one of my patients to write his name—

Fig. 40.



The moment that the attempt is given up the patient feels nothing abnormal; but the moment that he tries again to perform this special act the difficulty returns and increases. Sometimes the special symptoms are made worse by any exertion of the arm.

The cramp-movements, at first limited to the thumb and fingers, are sometimes avoided by the writer who adopts mechanical devices which leave them at rest, but make it possible to perform the act of writing by using only the muscles of the wrist and forearm; as soon, however, as he has trained himself to write in this awkward manner, the muscles of the forearm take on a spasmodic movement, and he is no better off than before. In one case, which I have recently seen, the patient could manage to write a few words by moving only the muscles of his arm and trunk—his pen was directed by the

muscles of his back and arm, the latter being pressed closely against his side; but, after a few seconds, spasm occurred in these, the whole body was contracted, the head being drawn downwards to the right shoulder, and the trunk contorted so as to render it concave on the right side.

In several cases that I have known the sufferers have taught themselves to write with their left hands, to do this with ease, rapidity, and neatness; but, shortly after having acquired the art, the left hand has become affected in a similar manner, and its writing-power has been limited more rapidly than was that of the other limb.

When the disease has existed for some time, the attempt to write often becomes painful; there is a feeling of "cramp," and much general distress, accompanied by spasmodic movements in the neck, and sometimes in the limbs not especially engaged in the effort; and yet, apart from the attempt to write, there is no spasm and no inconvenience.

After long persistence of the cramp there is sometimes feebleness in the general movements of the limb,—the grasp is not so firm as it used to be; but such quasi-paralysis is the exception, and not the rule.

In some cases the spasmodic movements have not been so closely limited, as they are in the majority, to the attempted performance of a special act; those movements which require no fine adjustment may be performed with force and propriety, but others—needing delicate coordination—may be difficult or even impossible. There has been tremor, or choreiform agitation of the limb, more or less persistent during the day, even when no voluntary effort is being made, but ceasing at night, during sleep, or after prolonged rest in one position.

There are—in some individuals, but not in all—abnormal sensations in the affected limb; and these may be noticed before any cramp appears. They may be increased by exertion, but do not entirely depend upon it. They are vague in character, such as a "feeling of weight," or of "tightness," "numbness," or "coldness;" a pain, but more often a "something not quite pain going up from the hand to the back." In some cases there has been actual anæsthesia of the fingers, and an "aching in the spine."[1] There is nothing peculiar to the disease now mentioned in the sensations that I have heard described, when description was possible, except this, that the attempt to control the spasm augmented the distressing or uneasy feelings.

In the majority of cases the special cramp exists by itself; but in a few it is associated with other disturbances of the nervous system. Those which I have met with have been torticollis, occasional strabismus, stammering, and palpitation of the heart, with some distress about the cardiac region, over and above the mere fact of increased force or frequency of beat.

The general health in some of the most typical cases has been excellent, and the physical strength equal to and even beyond the average. In a few individuals there have been weakness, a "nervous temperament," and some anæmia with impaired digestion and nutrition; but in no one has there been witnessed any modification which is not consistent with, and frequently encountered in, other diseases.

ETIOLOGY.—*Age.* Early life appears to be exempt; I have not met with a single case in which the symptoms appeared before the age of thirty. *Sex.* The male sex is much more liable to suffer than the female. *Occupation.* It is commonly held that the disease is caused by excessive exertion, but there are reasons for doubting the correctness of this statement. Thousands of individuals write, work, milk, or play musical instruments to the highest degree that is possible, without suffering from the least inconvenience of the kind now described; and, on the other hand, many cases occur in which there has been no excessive strain upon the muscles in the performance of these special acts; and, indeed, in some quite characteristic examples of the malady, there has been less than the usual amount of writing performed by gentlemen of the age and professions of my patients. It may then be convenient, but it is not scientific, to refer this form of cramp to over-exertion of a special kind:

Worry of mind and anxiety have been present in many cases before the outbreak of the symptoms, but so they have been in many other forms of nervous disturbance quite different from this; and in some persons affected with Writers' Cramp there has been nothing of the kind to which the patient or others could refer the symptoms. I have known the symptoms of Writers' Cramp to occur in one who had been much interested in their appearance in a friend. An *injury to the arm* has been supposed, in some cases, to have originated the disease.

DIAGNOSIS.—Scarcely anything need be added to the description already given. The special character of the difficulty is the diagnostic mark of true Writers' Cramp. A man may be unable to write from *lead poisoning*. But the presence of paralysis rather than spasm; the singling out of certain muscles not only for weakness, but for loss of nutrition and of irri-

[1] Solly, on Scriveners' Palsy; Lancet, Jan. 28, 1865.

tability to electricity in the induced form; the equal affection of the two upper extremities, although, when slight, it may be shown more conspicuously in the hand which writes, and has been educated to perform other complex movements; the presence of a blue line on the gums, and the general history of saturnine intoxication,—are sufficient to establish the diagnosis.

Wasting Palsy, which often commences in the muscles of the thumb, may be known by its characteristic feature, "wasting," and needs only to be mentioned in order to be distinguished from Writers' Cramp. In wasting palsy the loss of power is in direct proportion to the loss of nutrition; in Writers' Cramp, it is the spasm which interferes with the particular movement that is required.

Local Paralysis.—A few weeks ago a gentleman was sent to me with supposed "Scriveners' Palsy." He had been reading and writing much, and on one evening sat reading for some hours "in a draught;" his hand was weak, and on the following day he could not write. There was when I saw him nearly complete paralysis of the right hand and forearm, and the electric irritability was almost extinct, but in the course of a fortnight the power had returned, and the patient was well. The extent of the paralysis and the absence of spasm were the distinctive marks. Several cases of this kind have come before me, and have been thought to be examples of Writers' Cramp; but the fact of their having been mistaken for the latter is enough to put any one on his guard against a repetition of the error.

Prognosis.—If the case be seen when the symptoms have existed for only a short time, relief may be confidently expected, provided that rest can be given. If the symptoms have existed for many months, or if rest be impossible, the prognosis is extremely unfavorable. There is scarcely any malady which has resisted more obstinately all kinds of attempts—well-directed and ill-directed—which have been made for its cure. Many who were seriously threatened with Writers' Cramp are now free from the malady because they rested; many who could not and did not rest, are now, in the present state of therapeutics, incurable.

Bearing in mind what has been said with regard to the extension of the disease into other regions of the nervous system, some caution is required in stating the general prognosis of such cases, but, in the vast majority, it may be confidently expected that no such extension will occur. When there are signs of disease already present in other directions, such as strabismus, torticollis, weakness of the corresponding leg, and the like, the prognosis should be extremely guarded.

Pathology.—The exact locality of disease, and the precise nature of the change which constitutes it, have not been yet demonstrated with regard to Writers' Cramp. Its closest clinical affinities are with stammering, spasmodic wry-neck, and histrionic spasm, or "muscular tic" of the face. Analogous maladies, but moving in a yet wider range, are sometimes encountered, such as certain forms of rotatory movement, of chorea, and of locomotor ataxy; and beyond these there are anomalous cases, which every physician occasionally meets with, but does not know how to designate. One patient cannot make the attempt to walk without performing, or running in danger of performing, sundry rotatory movements, which terminate in a fall: another, a hard-working clergyman, can only speak, though he has the voice of a Stentor, when on a level with his audience; and this not from any fear, or shyness, or sham, but from definite aphonia. Some of the spasmodic movements induced in frogs by injury to the auditory nerve are of similar character;[1] and the experiments of Magendie, Flourens, Longet, and Schiff afford further illustrations of analogous disturbances in the physiology of motion.

In order to understand Writers' Cramp, it is necessary to remember what is, physiologically, involved in the education of the muscles to perform complicated acts, such as those of writing, speaking, or playing on musical instruments. The will does not pick out the muscles which are to be brought into play to hold a pen; it simply directs itself to the result. The boy who plays at marbles directs his movements in the same manner, and with as much accuracy and nicety, as the professor of anatomy directs his when he is writing a description of the muscles of the hand. The combination or co-ordination of muscular contractions is determined by the will, but is affected by another agency. Each is conscious of a wish to do a certain thing, and of a will to do it, but a knowledge of the mode in which the movement is brought about does not help, and may sometimes hinder, its production. Experimental physiology and clinical pathology combine to teach us that a certain portion of the nervous system, the cerebellum, has the power of effecting the co-ordination that is required; and they also unite in proving—what is often lost sight of—that this co-ordinating faculty is guided by sensations, and can act effi-

[1] Brown-Séquard, Lectures on the Physiology and Pathology of the Central Nervous System, p. 194.

ciently only when they are normal in kind and intensity. The production of a movement such as writing is therefore a very complicated process, requiring for its efficient performance the integrity of a great number of different parts: viz., that of the will and its immediate exponent in the cerebral hemispheres; that of the nerve-fibres between it and the muscles, together with that of the ganglia which exist on certain nerve-trunks; that of the muscles themselves; that of the cerebellum, as the centre of co-ordination, also that of all the "sensory" nerve-fibres which place it in relation with the organs of special sense and with the muscles themselves; and, lastly, integrity of the organs of sense, so that they, at the peripheral expansion of their special nerves, can receive impressions in a normal manner. It must be remembered, also, that not only the fact but the degree of contraction is under the control and guidance of the same organs or parts of organs. Failure in any one portion of this apparatus interferes with the production of the movement that is required; and the kind of failure is determined by the locality of the lesion; or, in other words, by the nature of the process or function which is lost or disturbed. If the contraction of a muscle be acutely painful, the man cannot write, the act would be impossible in some cases of rheumatism; if the muscle be wasted, it cannot be put into the same amount of contraction as in health; if the skin have lost its sensitiveness, all fine movements are awkwardly performed, and the finest are rendered impossible; they may be partially guided by the eye, but the guidance is defective for the most complicated acts; if the motor nerve be damaged, the muscle is *pro tanto* palsied; if the sensory nerves be injured, sensation is defective; if the sense of muscular condition be in abeyance, the power to control either the kind or force of contraction is without its guide: but locally, *i. e.*, so far as that limb is concerned, all other nerve, muscular, and sentient properties may be intact, and yet spasm or paralysis, or both, are present. If the will be deficient, and this from any cause, there is palsy, or irregular movement; if the fibres coming between it and the nerve-trunks be injured, there is paralysis in the ordinary sense of the word; if the cerebellum be diseased, there is loss of co-ordination, while power and sensation persist; if the spinal cord be injured, there may be, in relation to the nature and locality of the injury, almost any one of the conditions that have been enumerated.

In true Writers' Cramp, the will, the co-ordinating power in all directions but in one, the motor power, the muscular nutrition and activity, as well as the sensorial faculties, are uninjured; the individual is, or may be, "well" in all respects but one. A particular kind of movement is interfered with, by the occurrence of irregular and spasmodic, instead of regular and co-ordinated, contractions. It must be carefully remembered that the malady is special; the muscles which cannot be made to write can be controlled so as to fasten the most tiresome buttons, carve the toughest of pheasants, or pull a heavy boat. The pianist cannot play on the pianoforte, but he can write as well as ever; the bricklayer cannot use his trowel, but he can do everything else that he wants to do; and in order to understand this, we must revert to what is included in education, and what confers the dexterity which comes of special practice. Many movements are "automatic;" we adopt them without education and without effort; others are the result of laborious "practice." It would seem that the body is naturally endowed with certain paths or lines of nerve-action, along which all moves easily. The instinctive movements of the child or of the animal are examples of the mode in which, along these lines, impressions from without pass readily, and become converted into motor impulses, which are, in their turn, conveyed to muscles, which contract, and so perform these instinctive acts. But the process of education, so far as the performance of writing, playing, stitching, &c., is concerned, consisted in the frequent repetition, by an act of the will, of certain forced and complicated movements. The repetition makes them easy, until at length they are executed without effort, and almost unconsciously. It would seem that, by this education, new paths are forced, so that what was once difficult and required attention becomes day by day more easy, and at last "secondarily automatic." It cannot be doubted that some changes take place in the nutrition of the parts through which these lines of nerve-action run; and that their education involves structural alteration in the organs. The perfection with which complicated movements are performed in the lower animals appears to be associated with great keenness and remarkable development of the organs of sense; and in man a similar relation may be observed. No man writes well who has not keen sight and a quick sense of touch; no man plays well on the violin who has not an acute ear, and a delicate power of feeling in his fingers. In all instances of educated movement some "sense" is needed, and is an important element in the process by which the result is obtained.

In the present state of science it is not possible to say, for every act, what part of the nervous system is especially engaged in this educational development; but it seems probable that the association of

movement is effected by ganglia which are common to fibres passing through distinct but contiguous nerve-trunks, and that it is owing to some nutrition-change in them—the result of persevering and forced effort—that the perfection of movement is produced; associations at first caused by the will, are at last produced unconsciously. What happens, then, in such maladies as Writers' Cramp, is a perverted nutrition of these parts; a worn-out activity, or a degeneration which may arise without over-exertion, and destroy all that had been previously achieved.[1] Neuromata have existed in the arms of some patients. The disease, as it has been shown, passes readily from one side of the body to the other; and it must be carefully borne in mind that co-ordination of movement is a most complex process, requiring integrity of sensation as well as of motor nerve and of cerebellum. The real mischief may be some want of limiting and guiding influence ordinarily coming, through sensation, from external impressions. The spasm which occurs is very like that which Mr. Lockhart Clarke describes as taking place in the legs of ataxic patients who cannot regulate the *force* of their muscular contractions.

Treatment.—In an early stage absolute rest may do much; in a later stage it may accomplish something; but I know of nothing else which can be called a therapeutic agent. I have tried every form of general and nervine tonic, of sedative, and of local application, but no one of them has been of the least specific value. I have used hypodermic injections of morphia, atropine, and of arsenic, and have found them incompetent to cure the disease. The hypodermic injection of morphia appears sometimes to relieve the spasm for a certain period, and I have seen the writing become steadier within five minutes of the application, and increase in precision for half an hour, but the effect has then, or soon afterwards, passed away, and a frequent repetition of the process has been without any permanent result. It has, unfortunately, happened that several patients in whom I have used morphine hypodermically presented an intolerance of that medicine. I have employed galvanism and electricity in all their forms, and have seen no good results. But in many cases perfect rest has removed the symptoms, and it alone seems worthy of being regarded as a means of cure.

Mechanical contrivances for holding the pen may render occasional writing possible, but they do not affect the disease; and persistence in their use has been followed by an extension of the malady to the muscles of the forearm and arm.

CONVULSIONS.

By J. Hughlings Jackson, M.D., F.R.C.P.

It cannot be kept too much in mind that Convulsion is a symptom, not a disease. But it is the most striking member of the series of symptoms in which it occurs, and in many cases the only one about which we have definite knowledge. In other words, although we always believe a Convulsion to be symptomatic, we too often know very little of the condition of the system of which it is one of the symptoms; and this even after post-mortem examinations. Let us glance at the circumstances with which Convulsions may occur.

Convulsions occur in association with organic changes in the nervous system of the most varied kinds; for instance, with cerebral hemorrhage, and with intracranial tumors. They follow injuries to the head, either immediately or remotely: immediately (within a few hours), as when a blow leads to meningeal hemorrhage; and remotely (after weeks or months), when diseased bone, the consequence of a blow, causes cerebral abscess. They will occur in a healthy but parturient woman after severe loss of blood. They occur with diseased kidney. They come on as indirect results of syphilis, as in cases of gummatous tumors in the hemisphere. In children they are often associated with rickets. Some believe that Convulsions may be the results of disturbances of parts of the body at a distance from the central nervous system, the result of ec-

[1] Some of these points in the pathology of Writers' Cramp have been ably treated by Mr. Solly, in the Lectures already referred to in the Lancet of 1865.

centric irritations, such as the irritation occurring with dentition, or the irritation of worms. Finally, there are a large number of cases of convulsive seizures which (for want even of that approximative knowledge we have of such causes of fits as are mentioned above) we are obliged to speak of as essential, eclamptic, epileptic, or epileptiform. When we consider further that the symptom occurs at all ages and in many diseases, that there are many varieties in part of the body affected by spasm—it is unilateral or general—many degrees in severity—there may be local spasm without lost of consciousness, or general convulsion with profound coma—and in times of recurrence—there may be one fit a week, or fifty in a day—we are forced to the conclusion that we can only speak of Convulsion as a symptom.

The only things we can safely affirm of the symptom are certain truisms. It is the phenomenon of an occasional discharge of nerve tissue (no doubt of gray matter). It points not to destroying lesions, but to unstable nerve tissue—to "functional" changes. But the most careful study of the symptom (the paroxysm) tells us nothing of the *pathological* process by which such changes of instability are brought about; does not, for instance, enable us to say whether these changes are the result of "irritation," of tumors, of uræmia, or whether they are not minute changes (epileptic) to the pathology of which we have no clue. But clinical study of the *circumstances under which the symptom occurs*, tells us very much. Although it rarely leads us to a knowledge of the pathological condition of the nervous centres, it gives valuable information as to the treatment of the patient, and for the purposes of prognosis. There is a practical convenience in studying this symptom separately. Indeed, we are forced to this narrow study, as it is very often our only "way in" to a case; and its distinct consideration will not be hurtful if we use it as a point about which to group not only our positive knowledge for present action, but also, if such a phrase may be permitted, our positive ignorance for future research.

I will try to show what meaning we can give to this symptom under various circumstances; how we should investigate the condition of our patient who presents it, and what we should try to do for him. It may be well to say that I have only to consider epilepsy so far as diagnosis is concerned. It is justifiable to sacrifice some exactness to convenience by dividing the subject into (1) Convulsions in infants and young children, and (2) Convulsions in persons above seven years of age.

Convulsions in Children.

Convulsions may occur at any age, and this remark applies not only to general Convulsions but also to most kinds of convulsive movements. Still, Convulsion is *par excellence* the nervous symptom of infants and young children.[1] The tendency to Convulsions gradually decreases with increasing years. The following quotation from West shows this:—"In proportion as the brain increases in size, and its structure acquires perfection, and its higher functions become displayed, Convulsions grow less and less frequent, until, from the tenth to the fifteenth year, they cause less than three per cent., and above fifteen less than one per cent. of the deaths from diseases of the nervous system." The first line in the accompanying table (Dr. West adds in a footnote) shows the proportion per cent. of deaths from diseases of the nervous system at different ages, to the deaths from all causes at the same ages in the metropolis; and the second line the proportion borne by deaths from Convulsions, to deaths from diseases of the nervous system in general:—

Under 1 year.	From 1 to 3 years.	From 3 to 5 years.	Total under 5 years.	From 5 to 10 years.	From 10 to 15 years.	Total above 15 years.
30·5	18·5	17·6	24·3	15·1	10·6	10·4
73·3	24·9	17·8	54·3	9·9	2·4	·8

West says: "In a large proportion of cases of Convulsion in the infant, Convulsions answer to delirium in the adult;" and Trousseau says that there are children who have Convulsions as easily as some have delirium or even dreams.

We shall then, as a preliminary, speak briefly of the *physiological* peculiarities of the child's nervous system. We may affirm of it two things. (1) It is undeveloped. Besides the obvious fact that the infant has to acquire such movements as those of walking and talking, there is

[1] Meigs and Pepper write: "During the five years from 1844 to 1848 inclusive, 1729 children under fifteen years of age died in this city (Philadelphia) of convulsions, whilst, during the same time, 1611 died of infantile cholera, 1060 of marasmus, 1041 of dropsy of the brain, and 772 of pneumonia, showing that eclampsia was the cause of a

evidence from the special nervous diseases of children, that the parts of the young nervous system are not knit together as closely as in the adult—incomplete neurification analogous to incomplete ossification. The child is the subject of certain *limited* palsies and limited spasms which do not occur in the adult. Indeed, there is a form of talipes varus, Dr. Little tells us, which is always congenital. "Infantile paralysis" never occurs in adults. After hemiplegia in childhood, a well-known contraction (spastic rigidity) often sets in which does not follow hemiplegia in adults (p. 742). And lastly, coming near to our immediate topic, spasm of the glottis is a convulsion of a certain limited region which is rarely met with after the age of three or four years. Then there are minor symptoms which are almost peculiar to children, *e. g.* carpo-pædal contractions. As regards the last two symptoms, there are the significant exceptions that they occur in hysterical women.

(2) The nervous system is developing. It is in a state of active change. Its nutrition will be in considerable excess of its expenditure, whilst in adults the two will be more evenly balanced. For this reason the child's nervous tissue will naturally be more unstable than is that of the adult. It will more easily discharge from a slight cause, or, to use a common expression, it is more excitable. It is believed too that the equilibrium of the child's nervous system is more often upset by *nerve-transmitted irritations* than that of the adult is. Hence very severe convulsions are ascribed to irritation carried by the fifth nerve from the gums during dentition, or to the irritation of worms. The child's nervous system is even believed by some to be naturally so unstable that eccentric irritations so very local as those just mentioned will produce a general convulsion in a *healthy child*, *i. e.* will determine a sudden and excessive discharge of nervous tissue which is only *physiologically* unstable. Others will qualify this opinion by the supposition that the nervous system, or some part of it, is *pathologically* unstable prior to the action on it of the transmitted irritation. Thus the late Dr. Hillier says (I italicize some words): "It is very doubtful whether in a *healthy* child these causes can produce convulsions at all; in a *predisposed* subject they no doubt often *excite* them."

The above are physiological differences. Disease finds the child's nervous system undeveloped, and it finds it developing. But, so to speak, the attacking disease itself has peculiarities, at all events negative peculiarities. A child is much less likely to suffer from gross lesions in the brain, such as hemorrhage, syphiloma, and other new growths; he is less likely to suffer from uræmia excepting from acute changes of the kidney, especially of scarlatinal origin; or, putting it more simply, we usually *discover* no pathological changes in the nervous system of a child who has died of Convulsions. Wilks, speaking of diseases of children, says: "We meet with a large number of cases where the post-mortem appearances are absolutely nothing; and where, indeed, we could scarcely expect to find it otherwise. We allude especially to cases of *Convulsions* in children where no morbid changes are discovered; and when we consider that a child may have several convulsive attacks and speedily recover, which only a degree more severe shall prove fatal, it is clear that no very great change could occur in an organ which would have perfectly recovered itself had the fit been only one degree less in severity." Whilst it is true that, as a rule, no pathological changes are discoverable, we must not infer that pathological changes do not exist; the probability is that there are minute changes. Nor must we infer from complete recovery from a convulsion, or a series of convulsions, that there are no pathological changes. Adults recover even from hemiplegia, which subsequent post-mortem examination shows to have been due to obvious although very limited destroying lesions—small clots, for instance. Therefore recovery from Convulsions is no certain sign that there was no real impairment of structure. It is a sign only that no wide breaking up of structure has happened. There must certainly be local changes in those cases of Convulsions in which hemiplegia follows, however temporary it may be, since local symptoms of necessity imply local lesions. Still this is only a necessary inference, as we rarely *discover* any changes even in these cases. Nay, even in those rare cases where we find *gross disease*, a tumor for instance, we do not discover the *minute* and *secondary* changes on which the discharge producing the Convulsion depends. We must not say that the tumor was the direct "cause" of the Convulsion, but that it led to secondary changes in nervous tissue on which the Convulsion depended, and these secondary changes are inferred, not demonstrated.

It is true that there are found at examinations after death from Convulsion abnormal quantities of serum and blood in the head, but these differences are quite as

larger number of deaths than any other of the diseases just mentioned. It must be recollected, however, that a very large number of these cases ought, beyond doubt, to have been returned under other titles, as many of them must have been a mere result of organic disease of the cerebro-spinal axis, and of other acute local or general diseases."

likely to be results of the fits as their causes—the results of the sudden interference with respiration. Effusion of serum and congestion of the brain have not been shown, either in adults or in children, to have much to do in producing sudden and severe cerebral symptoms of any kind. Of course those cases in which, possibly from obstruction to the vein of Galen, as by tumors of the vermiform process of the cerebellum, there is immense effusion into the cerebral ventricles, are not in question.

To resume, we know nothing of the causes of Convulsions in children in the sense of knowing what the pathological changes are. This is so, however much we may narrow our consideration to groups of cases, either to those which occur singly and at intervals over a period of months or years, and which are often called epileptic, or to those in which the fits occur in considerable number for a limited period, and are often called eclamptic.

After these general remarks on the symptom Convulsion as it occurs in children, we have to consider what meaning we can give it in particular cases. Our task is twofold. We have first to note carefully the *kind* of *paroxysm*. For instance, is the Convulsion one-sided? Is it followed by hemiplegia? Secondly, to investigate the child's *general bodily health*. For instance, is he rickety? Is there bronchitis? Is there irritation from dentition?

Paroxysm.

Convulsions occur in all degrees. The Convulsion may be a twitching or clenching of the hands only, or an occasional grim smile in sleep, or the spasm may be general and so severe that the child dies of the paroxysm, even of the first. Attacks of slight occasional spasm, be it of one finger, have the same *general* significance as a severe convulsion has. They are both Convulsions, the proof being that, as in adults, we have very often first the local and quasi-trivial spasm, and later, a general convulsion. Each of them is a sign that there is an abnormal discharge of the nervous system or of some part of it. But the *effect* of the severe and of the slight discharges is different. We have to consider very carefully not only the "cause" of the convulsion, but also what effect single and repeated paroxysms produce on the child. The slight and partial fits do no harm, or little harm, for they do not spread to the respiratory muscles, and thus, as it were, they do not retaliate on the nervous system which "began it" by congestion of the brain, and they are too slight to exhaust the child by abnormal exercise of the muscles convulsed. Further, it is usually held that fits so slight and so partial point to slight and usually to transient causes, and that they often disappear when we obviate some condition of ill-health, such as wrong feeding and diarrhœa, or when we lance gums swollen during the eruption of teeth. But even granting that these slight symptoms signify that the nervous system is but slightly disturbed, or that they usually directly result from some removable condition, and that the seizure does little harm to the patient, they still demand serious consideration, for two reasons. In the first place, we think ill of that nervous system which is upset ever so little by slight causes, such as over-eating, and we have anxieties that if the child be afterwards exposed to severer exciting causes, such as fright, exhausting diarrhœa, &c., very severe convulsions may occur. Another reason for careful attention to slight spasms is, that they may not disappear under treatment, or that they disappear for a time only, and that they are often the premonitory symptoms of severer convulsions, the paroxysms of which *will* do much harm to the child by interfering with respiration and, when frequently repeated, by exhausting him. Therefore, although in strictness these slight symptoms are themselves miniature convulsions, it is convenient to consider them, along with other symptoms, as warnings. But it must be remarked that, like an adult, a child may be suddenly attacked by a severe convulsion in the midst of what seems to be perfect health. He may die in the first fit, or we may see him soon after the seizure playing about as if nothing unusual had happened.

Premonitory Symptoms.—It is proper to mention, to begin with, that Trousseau states that there are no premonitory symptoms. "Nothing," he says, "foretells the invasion of the attack; and, for my part, I have never observed the premonitory signs spoken of by Brachet, and repeated after him by others." Most authors, however, admit that there usually are warning symptoms. There are often symptoms before there is any local twitching, such as peevishness, want of sleep, and sleepiness. These symptoms show that the child's nervous system is suffering, but they cannot, of course, be taken as evidence that the illness is one in which the symptom Convulsion will be the sole or even the most striking event. In the child, as in the adult, want of sleep is associated with drowsiness. Adult patients will sometimes say what very young patients are not likely to tell us, that they always feel sleepy and never sleep soundly. Children who are about to have Convulsions will sleep with their

eyes partly open; their mouths will twitch; they will start in their sleep, grind their teeth, and may have night terrors. In the day they are dull, heavy, and peevish. When any twitching occurs on *one* side of the face or in one limb, or in both limbs of *one* side, however slight the cause, let us say over-eating, which seems to excite it, we fear the nervous system is seriously implicated, and that severe convulsions are setting in. When the slight symptoms occur during waking, especially if now and then a vacant look points to some loss of consciousness, however transient it may be, we fear severe convulsions are at hand. I may here quote, as a summary of the occasionally insidious march of the symptoms, what Churchill says of fits of dentition: "I have frequently observed a sort of gradation from simple irritation and restlessness to starting, surprise, wildness of look, partial or local convulsive movements, and, lastly, general convulsions."

Having spoken of partial or slight fits and incidentally of premonitory symptoms, we now come to consider varieties of severer convulsions. It is not denied that we may have any kind of occasional spasms in children, but we choose three types. It is a very important matter to note the variety of Convulsions, especially for prognosis: 1. Laryngismus Stridulus; 2. Unilateral Convulsions; 3. General Convulsions.

Laryngismus Stridulus.—A certain kind of Convulsion is called laryngismus stridulus because the muscles of the larynx being attacked by spasm, a noise results during inspiration from narrowing of the glottis. It is not a laryngeal disease, although one of its names, "false croup," seems to imply that it is. There is no continued fever in laryngismus. It is Convulsion affecting the muscles of respiration. Dr. Gee, in a most able article, very rich in clinical observations (Convulsions in Children, St. Bartholomew's Hospital Reports, vol. iii.), remarks: "In laryngismus (convulsion interne) we have a disease closely allied to epileptiform convulsion. Out of fifty cases of laryngismus of which I have notes, nineteen had had eclamptic fits." Niemeyer treats of it under the head of Nervous Diseases of the Larynx (Spasm of the Muscles of the Glottis). The larynx is found to be quite normal post-mortem. The obtrusive symptom, the crowing noise, is due to spasm of the glottis, but in many cases the muscles of the chest and abdomen, as well as those of the larynx, become involved.

The alliance of the laryngeal spasm—the local convulsion—with general convulsion is further shown by the fact that not infrequently the child has first laryngismus and then general convulsion. Occasionally we find in the intervals of the fits tonic spasm of the hands and feet—carpo-pedal contractions.

Yet it has certain peculiarities beyond those of limitation of range of the spasm. Age is one of the most important of these. Dr. West compares attacks of laryngismus to hysterical attacks, and remarks that both occur when processes of development are active. Out of thirty-seven cases of laryngismus, thirty-one, he tells us, occurred betwixt the ages of six months and two years. Vogel[1] says, "The age at which the disease occurs, fluctuates between one-half and three years; that is to say, it makes its appearance with the eruption of the first tooth, and disappears with that of the last." Niemeyer says that spasm of the glottis occurs almost exclusively during childhood, and especially in the first year of life. It is most frequent during the period of the first dentition. He makes a remark which is of considerable interest in connection with the one quoted from West: "Among adults none but hysterical persons suffer from spasm of the glottis, and those only exceptionally." Mackenzie, in his work, "Nervo-muscular Affections of the Larynx," treats of laryngismus under the head of Spasm of the Adductors of the Vocal Cords, and says that hysterical cases (in adults) are by no means infrequent. The observation of these cases is of very great interest, because in the adult the condition of the vocal cords in the attack can be seen, and we may plausibly infer that the condition of the glottis is similar in the laryngoscopy of children. Mackenzie writes of adults: "With the laryngoscope the vocal cords can be seen on inspiration to be spasmodically approximated. They may separate widely; but, instead of remaining apart for a few seconds, they are instantly and spasmodically adducted to the median line, or even beyond it, that is, against one another." Another peculiarity is, that boys are much oftener the subjects of this disease than girls, "a fact almost all authors admit" (Vogel). Of Gee's (op. cit.) forty-eight cases, thirty-four were males. Mackenzie (op. cit.) says, "The greater liability of the male sex, which occurs in other laryngeal diseases, holds good here."

The most striking feature of the disease is the crowing noise the child makes. This noise often begins insidiously, but there is great difference in this respect. The crowing noise is most frequently observed when the child awakes from sleep, and is very often noticed for the first time

[1] A Practical Treatise on Diseases of Children. By Alfred Vogel, M.D. (Translated by H. Raphael, M.D.) Appleton & Co., New York.

in the night. After several attacks of the crowing, which is generally at first occasional, and produces little inconvenience, very often exciting no alarm in the child's friends, a severe paroxysm may come on. A slight crowing noise may become almost continuous in the child's ordinary respiratory movements, and a severe attack may come on in the midst of this warning. The convulsion may be, it is believed, at first no more than spasm of the laryngeal muscles—a laryngeal convulsion; but in many cases the whole system of respiration is involved in the spasm, and sometimes the limbs—the convulsion becomes general. The severe attack is paroxysmal, and while the paroxysm is on, respiration is much impeded—sometimes indeed being quite, for a short time, suspended, as in severe convulsion in the adult. At the climax the face is flushed, the eyeballs start, the veins of the neck are distended, and the face wears an aspect of exquisite distress. The sign that this stage of danger is passing is a crowing or whistling noise made by air entering the now only narrowed glottic aperture. As before said, general convulsion may supervene. In the intervals, as in the other forms of Convulsion, if the attacks be not frequent, the child may be quite well, or only fatigued and peevish. If they are frequent (and they may occur thirty times a day), the child may be exceedingly exhausted, almost comatose. Occasionally, although this is a rare occurrence, the child may die in an attack, even in the first attack, just as now and then an adult may die in an epileptic attack. Nay, according to Niemeyer, in rare cases spams of the glottis in hysterical adults produces death by suffocation.

Without underrating the importance of studying particular convulsive seizures, due to spasm of certain groups of muscles which have especially important duties, we must, as regards treatment, consider the more general question of the state of the child's health or nervous system, which permits occasional spasm of muscles anywhere, whether these be of the limbs, of the thorax, or of the larynx. The inference is that the causes which give rise to laryngismus are essentially similar to those which give rise to other varieties of Convulsion. (See p. 744.) It is convenient, however, to say a few words here on causation.

The general belief is that this form of Convulsion is oftener than other varieties of Convulsion determined by the irritation of dentition, a belief which the facts as to age seem to justify. I say "seem," because the eruptions of the teeth are no doubt to be considered as marks of stages of development of the whole system, just as the occurrence of menstruation is later in life. Further, there is another fact of very great importance never to be lost sight of, viz., that, as Jenner, Elsässer, and Gee have pointed out, children the subjects of laryngismus are usually, almost always, rickety. Forty-eight out of Gee's fifty cases were unquestionably rickety, and in the two exceptional cases there was laryngeal catarrh.

We must at least modify the inference as to the influence of dentition, and say that the irritation of dentition produces laryngismus in rickety children. In none of Gee's cases (op. cit.) was there any reason to believe that the teeth bore any part in the causation of the fits. In accordance with these facts and opinions, whilst we must certainly endeavor to remove every source of eccentric irritation, we must also treat the child for rickets as well. "Laryngismus, when treated as if wholly dependent on the rickets, even if it be not so in fact, ceases to be a serious disease" (Gee).

Laryngismus has been attributed to enlargement of the thymus, but this view is not now entertained, one very good reason being that post-mortem examinations show that in many instances there is no enlargement of this organ. Moreover, in cases where a large thymus has been found there has been no laryngismus.

Unilateral Convulsions and Hemiplegia.—The Convulsions are limited to one side, or they affect one side first and chiefly. In such a fit, if it be severe, the face, arm, and leg of one side are in spasm; the head and both eyes turn to the same side, and next the chest becomes fixed. When severer still, the other side of the body becomes affected in the same way as the first, but to a less extent; the spasm may return to the side first affected. In this class of fits the spasm may be, for a while, very limited, *e. g.* a few jerks of the head to one side, or to spasm of one side, or it may be sometimes limited, and may at other times affect the whole of one side, or spread over the whole body. When the spasm is very limited, to the arm for instance, or even, when slight in degree, to one side of the body, there may be no loss of consciousness. The sources of danger to life from the paroxysm in this and in other forms of Convulsion are either that the spasm may fix the respiratory muscles, or that the frequency of the attacks may severely exhaust the child.

It is important to note this kind of Convulsion, because it is the one which is often followed by hemiplegia. We shall therefore anticipate what has to be said of the sequelæ of fits so far as this symptom is concerned. We should always carefully examine the child's limbs, after as well as during a fit; and when the fits of this kind are frequent, we may find the

arm and leg of one side paralyzed. If, however the child be deeply insensible, we may not be able to determine this, just as we cannot determine the existence of hemiplegia in some cases of cerebral hemorrhage in adults so long as the patient is very deeply comatose. We may first find out that the patient is paralyzed when he is recovering from the "status epilepticus." I believe hemiplegia is common, but the palsy usually passes off quickly. This is precisely what occurs so often in adults. We frequently see unilateral convulsions, or more strictly convulsions beginning unilaterally, in adults, followed by transient hemiplegia—the epileptic hemiplegia of Dr. Todd. However, the palsy in children sometimes does not pass off; and if it remains for many days *after convulsions have ceased*, and if, above all, it remains so long after but *one* severe convulsion in a child otherwise seemingly healthy, it is very likely to be permanent. The palsy may be only a little weakness, or there may be complete immobility. In most cases the leg at all events recovers so far that the child can walk. As the child grows up, the condition is often a mixture of palsy and spasm. There is either "contracture" or "spastic rigidity" of the hand and foot—the foot suffering very much less than the arm. More rarely the face suffers too in the same way. As the child grows up the paralyzed parts are smaller than those of the other side, the bones as well as the soft parts; the scapula is frequently strikingly smaller on the paralyzed side. The condition is not like that of limbs affected by infantile palsy. The muscles respond to the interrupted current, and when there is much spastic rigidity, the arm, although shorter than the sound limb, may be thicker. Here we may say a few words as to the cause of this symptom.

In the first place, unless the child's nervous system is altogether different from that of the adult, the symptom points to disease of the opposite side of the brain. (*Vide infrà* on Convulsions in Adults.) But such a symptom does not of course point to any particular pathological change.[1] It is rare to find atrophy of the optic nerves in hemiplegic children, and this is some evidence that there is no gross lesion, such as tumor, tubercle, &c. The changes are probably minute. The causation of the symptom cannot be clearly discussed. But this issue may be raised: Is it the result of the very same changes which caused the Convulsion, or is it the result of damage to the brain in the paroxysm, *e. g.* to excessive congestion, or even rupture of vessels from sudden stoppage of respiration? I have no doubt it is owing to the first cause. 1. Because the rule is that the spasm has been on the side afterwards paralyzed, or has begun on that side and affected it chiefly. 2. Because in the epileptic hemiplegia of adults, in whom we *do* sometimes find gross changes, syphiloma for instance, we find the disease in the cerebral hemisphere opposite the side of the body, first convulsed and afterwards paralyzed. To this may be added the argument that general congestion of the brain is not likely to lead to so local a symptom—to paralysis of *one* side of the body. The fact that the hemiplegia is often transient does not show that there have been no local changes, because in adults, hemiplegia, after a convulsion, is often transient, even when there is organic disease of the brain,—syphiloma for instance.

The Convulsions are general.—It is not meant that the Convulsion affects both sides together, nor both sides quite equally, but that both sides are nearly equally affected, and nearly at the same time. The chief point here is to consider the condition of the thorax in the paroxysm. A long stoppage of respiration is the worst symptom we can witness, and when a child dies in a fit he is no doubt killed by the prolonged fixation of his thorax. In some of these cases the limbs seem to be comparatively little affected, although all four are somewhat affected. The older the child, the more the limbs and the less the chest suffer. These fits vary much in degree of severity. If slight, the child may, as in other varieties of Convulsions, seem quite well shortly after. If severe he may remain exhausted and appear dull only; if very severe, he lies in deep coma. The frequency of the fits modifies his condition. He may have a second fit before he has recovered from the effects of the first, or as the nurse will say, "in and out of fits all day long."

The child may have an attack and never suffer again. He may die in his first fit. It is not very uncommon for a fit to occur in a child who is seemingly quite well. He is suddenly convulsed, and may as soon as the fit is over go to play again as if nothing unusual had happened. It is to be insisted on that, however well a child may seem before and after a convulsion, we cannot be sure that he will not

[1] I have made an autopsy on the body of a young woman, twenty-two years of age, who had been hemiplegic in the left side, after one series of fits, since the age of about three years, and subject to frequent convulsions from the age of five or six. The right cerebral hemisphere was much smaller than the left, and the left arm and leg were smaller than the right arm and leg. I found, however, no disease beyond what the unilateral atrophy implies. I have to thank my friend Mr. Norton for permission to see this patient.

have more. He probably will. Next day or next week he has another, and then perhaps thirty in the day. From a rapid succession he suffers in two ways: 1. The respiratory function is much interfered with, and it is suddenly interfered with. 2. There is very great exhaustion from the severe muscular "exercise," and want of sleep. These things will be particularly referred to under "Treatment."

SEQUELÆ.—The chief sequelæ may with great looseness be arranged as, (1) Paralysis; (2) Amaurosis; (3) Defects of Speech and Disorders of Mind; (4) Squinting; (5) Paralysis of cranial nerves. There are of course other defects after Convulsion: loss of smell, loss of hearing, and unsteadiness of gait. These, however, are less common; they have different significance, being more accidental than the other defects I have named, and often depend on organic disease.

(1) Now children are subject to two kinds of paralysis, one of which almost deserves the name of essential, and is well enough recognized when called Infantile Palsy. The other, which will occur at any age, namely hemiplegia, is the form of paralysis which most frequently follows Convulsion, and has been already considered. (2) Amaurosis will be considered elsewhere: it is a rare sequel of Convulsion in children. If we find double optic neuritis, or double optic atrophy, we fear there is a gross intracranial lesion, such as tumor, a lump of tubercle, &c. Recovery from a condition very like that occurring from meningitis does not contra-indicate the existence of gross organic disease, if there has been double optic neuritis. Atrophy of the optic nerves occurs with fits owing to chronic hydrocephalus. Under any circumstances we can do nothing for Amaurosis from atrophy of the optic nerves. (3) With loss of speech we may take in mental defects, because in children the two things often go together. (Deaf-mutism is not considered here. If deafness occurs from any cause before speech has been acquired, mutism is the result.) After attacks of Convulsion, children are liable to lose their speech, and this loss occurs without any notable lack of power in the articulatory muscles. The proof of this is that the child eats and swallows well, &c. After a while, a few months or a few years, he begins to utter some word or syllable, *e. g.* "do, do," "ta, ta," "mam, mam," and he utters such syllables very distinctly. This loss may be temporary or it may be permanent. There is rarely hemiplegia along with loss of speech in children, or at all events rarely persistent hemiplegia. Sometimes after Convulsion, before speech is acquired, the child never learns to talk. If the child is only beginning to talk when the fits occur, loss of so recent an acquirement is usually only temporary.

In most of the cases I have seen where the loss of speech is permanent, the children are spiteful, vicious, and they have nearly always uncontrollable tempers. Children may be left after Convulsion in this general condition without loss of speech. These are, I think, the saddest cases in the whole range of our practice. Trousseau says that idiocy very often supervenes on infantile Convulsion. But the condition of speechless children is often worse than mere imbecility. There are many varieties of this sad mental condition, from some vulgar coarseness of mind to a total absence of decency. No purely medical treatment is of any avail. In many cases a false sentiment induces the parents to let the enfeebled mind grow as it lists rather than put their child to the annoyance of vigilant discipline. Indeed sometimes a fond mother not unnaturally mistakes a kind of animal vivacity for cleverness, and will not be persuaded that her child is much different from others, even when he cannot or will not dress himself or cut his food. Strenuous efforts must be made to teach the child to talk, for Dr. Langdon Down tells me that idiots who have come under his care speechless have been taught to speak. Very strangely, the worst of such patients even when speechless have often a good ear for music, and will hum or sing tunes correctly. This is not an important sign of mental power. Dr. Down tells me that idiots often can sing. The younger the child, the more likely is loss of speech to be attended by general mental deterioration. I do not know how it is that there is loss of speech after some convulsions and not after others. It is not, I believe, the result of the Convulsion, but is another result of the disease in the brain, be it what it may, which gives rise to, or permits, the Convulsion. Loss of speech is indeed a rare sequel of Convulsions, and will occasionally attend a simple attack of hemiplegia—*i. e.* simple in the sense that it is unaccompanied by Convulsion, or other evident symptoms. I may just observe that the association of defects of speech with symptoms pointing to disease of the left hemisphere is not so striking as it is in adults, but it will be found that they more often occur with Convulsion and paralysis of the right than of the left side of the body.

(4) Squinting has long been considered a common symptom of affection of the nervous system in childhood. But Helmholtz has shown that the most frequent cause of strabismus is a congenital defect in the eyeballs. It is found in those whose eyes are hypermetropic. Of course a child may have strabismus, as an adult

may, from paralysis of one or more of the muscles moving the globe. It is generally held, however, that strabismus, when a nervous symptom, is a result of spasm of muscles. Yet neither paralysis nor spasm of one muscle—for instance, of one internal or of one external rectus—will properly account for the common form of strabismus. The kind of strabismus we meet with in children during or after Convulsions is not found during nor after Convulsions in adults. The presumption is that this difference is owing to differences in the development of the nervous system of the child and the adult.

(5) If the whole of the muscles supplied by any one of the cranial nerves are paralyzed during or after Convulsions in a child or in an adult, we are certain that there is disease of that nerve trunk, or of the part of the nervous system where the nerve issues. In these cases we are usually able to infer that there is organic disease. We should especially inquire for evidence of syphilis.

The Causes of Convulsions.

We have considered the several kinds of paroxysm, and now we have to speak of the diagnosis in cases in which a paroxysm of any kind occurs, or, as it is usually said, on the "causes" of Convulsions. However, we have already seen that we know scarcely anything of the pathology of children's Convulsions; we do not know what is the change in the nervous system which produces this symptom, because in most cases the brain appears healthy *post mortem*. It is scarcely fair, therefore, to use the word "cause." We can, however, group cases according to certain circumstances, and the following list will show, better than any exposition can, the limited way in which the word "cause" is to be used with reference to Convulsion. It is needless to say that the arrangement is a very arbitrary one, but it is convenient as guiding us in prognosis and treatment. We shall find that many a case will refuse to be placed altogether under one of the headings. For this reason it is impossible to discuss the points raised under each of the five following headings, quite separately from those raised under the others. When called to a case of Convulsion, we may find—

1. That an acute illness is setting in, as, for instance, an exanthem or acute cerebral disorder.

2. That the Convulsion is one of a series of symptoms, the result of organic disease of the brain or of its membranes, such as tumor, abscess, syphilis, &c.

3. That the fit is part of some chronic condition of ill-health, such as rickets, exhaustion and emaciation with diarrhœa.

4. That the child is suffering from some slight and temporary thing, such as an overloaded stomach, perhaps diarrhœa, the irritation of worms, or the irritation of teeth.

5. That the fits are of a kind which, for want of better knowledge, we are obliged to call essential, eclamptic, or epileptic.

(1.) There are symptoms of fever. Here we should fear that some acute illness, as pneumonia, smallpox, or scarlet fever, is beginning, of which the Convulsion is, if not the first, yet the first prominent symptom. It scarcely comes in my way to do more in this paper than urge the recognition of these rarer possibilities in the crowd of more probable causes. I must refer to special articles on the acute diseases of children. We should always make investigations for many acute diseases when there is heat of skin. The chest should be examined as a matter of routine, as not very rarely a child is first found by its parents to be ill when a Convulsion occurs, although when we examine we discover that there is bronchitis, or even that one pleural cavity is full of fluid. The history of scarlet fever in other members of the family would be a most important guide, and we must never fail to make inquiries for it, however suddenly the fit may have come on in the most robust child. Dr. West mentions a case, no doubt scarlatinal, in which Convulsions succeeded by coma destroyed in a single day a healthy boy two years of age. We should in all doubtful cases examine the urine. The presence of blood in the urine would help us to determine whether the fits were or were not the result of masked scarlet fever. We require more observations on the condition of the urine in convulsive seizures soon after the attacks. Sir James Simpson has found albumen in the urine of a child three days old who suffered from Convulsion.

Feverish symptoms may be thought to point to causes which primarily affect the head itself, tubercular meningitis for instance. The difficulty in diagnosis is only likely to occur in cases where Convulsion is the first symptom, or, in more strictness, the symptom for which we are consulted. It is to be remarked, however, that Convulsion is rarely one of the symptoms of the invasion of tubercular meningitis, and when it is we usually find that the child has for weeks, without obvious cause, been getting thin, listless, and peevish—that in reality he has been ill for some time. Moreover, facts as to age will often help us in the diagnosis of many of these cases. Tubercular meningitis rarely attacks children under two years, and very rarely indeed under one year of age. It is well to remark here that we must not mistake the status epilepticus—

the condition resulting from a quick succession of fits—for meningitis. If the convulsions be very numerous, the illness beginning by a convulsion, the disease is very unlikely to be meningitis.

(2.) Then it is possible that the Convulsion may be a symptom of organic disease, *e. g.* tumor. Such cases in children are very rare. There is usually a history of severe headache and urgent vomiting for weeks or months before the Convulsions, without any previous rapid emaciation to lead us to suppose there is tubercular meningitis. Moreover the symptoms are capricious, the headache and vomiting often ceasing for days. When these symptoms are not urgent we may be first consulted for a convulsion; we see cases occasionally in which a convulsion occurs from tumor of the brain in a child whose symptoms had been primarily so slight as not to prevent his going about, the severe headaches being put down by the child's friends to "biliousness," &c. In these cases we can only decide by considering the general evidence. The urgent, purposeless, often bilious, vomiting, possibly existing with clean tongue, and not infrequently with good appetite at intervals, days of comparative freedom from marked symptoms, point to the diagnosis of tumor. But the most conclusive evidence is the presence of double optic neuritis. If with headache, vomiting, and convulsion there is double optic neuritis, we may be almost certain that there is cerebral tumor. Double optic neuritis is rare under seven years of age, and very rare under five.

In all cases in which there is or has been discharge, especially offensive discharge, from the ear, especially when there is also pain in many branches of the fifth nerve; above all, if palsy of the portio dura nerve comes on, we should fear one of two things: (1) cerebral or cerebellar abscess, or (2) meningitis. It is not part of my task to enter fully into the diagnosis of these two conditions. We must not decide unless there are *general* symptoms also, *e. g.* increase of temperature, constipation, vomiting, and stupor, for discharge from the ear is not infrequently attended by *chronic* convulsions occurring at intervals for years (epilepsy), and the fit we are consulted for may be the first of such a series. What the connection of the ear disease with the fits in these chronic cases is we do not know, unless we adopt the explanation that they depend on irritation starting from the ear. The frequent repetition of fits, even twenty or thirty in twenty-four hours let us say, does not make us incline to the diagnosis of abscess or meningitis, because it is not at all uncommon for Convulsions both in children and adults to occur in batches when there is no ear disease and no evidence of organic disease in the brain. The stupor which is produced by a succession of fits is of little moment, comparatively speaking, unless there has been before the advent of the fits severe pain in the head and vomiting, and unless the respiration and pulse be irregular.

It is possible that the Convulsions may be owing to congenital syphilis; but we cannot so conclude unless, *besides* a clear history of syphilitic taint, there are such symptoms as palsies of cranial nerves, or paraplegia. The existence of hemiplegia with a mere *history* of syphilitic taint would not warrant the diagnosis. Of course if signs of active syphilis be present, especially nodes, and especially if the patient be above three or four years of age, we should treat for syphilis. Indeed, in chronic cases at all events, if the patient's brothers or sisters show signs of inherited taint, we should give iodide of potassium.

(3.) If we found the child emaciated (there being now, we are supposing, no signs of acute mischief and no signs of organic disease), or if he had diarrhœa or dyspepsia, we should hope that the fit was but a sign that the nervous system was temporarily suffering with the rest of the body, and that it would cease to suffer when the child was brought back to good general health. Convulsions not infrequently occur in infants improperly fed. In these cases there is often dyspepsia and diarrhœa, there is a big belly with general emaciation. The convulsions are then often ascribed to irritation from cutting the teeth, or, especially when there is diarrhœa, to the irritation of the intestinal canal. (The diarrhœa itself is sometimes ascribed to eruptions of the teeth.) These causes may have something to do in *provoking* the fit, but they cannot, I think, be solely to blame for it. Yet there can be no doubt as to the propriety of helping a tooth through if the gum be tense, swollen, or red; but to lance the gums as a matter of routine is not good practice. I have no belief that such irritations are the *sole* causes of fits; but I will now only deprecate exclusive attention to these supposed sources of eccentric irritation. If fits do sometimes cease when a vermifuge brings away worms, or when the gums are lanced, we know that in many cases they often continue when the system is freed from such cares, or at least when we have done all we can to get rid of them. I think with young practitioners, such "causes" are held to explain the occurrence of the fit so thoroughly that they keep in the background the evidence the child's general state will afford. We are, to say the least, unauthorized to ascribe children's illnesses solely to the irritation of teeth and the like, unless we have most thoroughly considered all other possible causes.

Above all, we must negative the existence of rickets. In Gee's article, several times referred to, the frequent, almost constant, association of Convulsion, including laryngismus, with rickets, is insisted on. He notes seventy-three cases of Convulsion, and of these fifty-six were dependent on the "general condition" of the child. These fifty-six cases were, he tells us, of that kind commonly called, *par excellence*, cases of essential Convulsions (eclampsy). "Now, of sixty-one eclamptic children, fifty-six were rickety. Saying this I fear that I shall incur the charge of exaggeration. It is necessary to explain that my experience is wholly derived from the children of the poor." He tries to show, and I think he shows conclusively, "that the existence of a constitution leading to rickets is the most important fact in the kind of Convulsion [that depending on the general condition of the child] in question." And even when no other causes are obvious, we must not infer that the relation between teething and Convulsions is one solely of irritation transmitted from the gum to some part of the child's nervous system. The coming through of a tooth must be thought of as an outward mark of a certain constitutional progress in development, as the occurrence of menstruation is later in life. It is far better to acknowledge that very often we cannot find out what causes a fit than to put it down to an orthodox cause for the want of a more real one.[1]

When diarrhœa is severe the child is often emaciated, and the fit is more likely to be due to exhaustion than to eccentric irritation starting from the intestinal canal. We should not adopt routine efforts at clearing out the bowels to get rid of "undigested irritating" matters. In most of these cases the child has been improperly fed, and careful dieting is of very much more moment than immediate treatment of the diarrhœa by drugs. A child is at once overworked and underfed when it has to take into its stomach large masses of food which it cannot properly digest. We must certainly not consider the most violent convulsion in a child who is thin or who has exhausting diarrhœa as a result of "congested brain." There may be stagnation of blood in the head, the result of the paroxysm, but not any "active" process requiring antiphlogistic treatment. We do not know what the intracranial changes are which cause fits, and we have no evidence that cerebral congestion occurs *before* the attacks. We have plenty of proof, from disease and from experiments on animals, that Convulsions will follow anæmia; but as in these instances the anæmia is *sudden*, perhaps the facts are not quite to the point. We have, however, clinical evidence that they occur in feeble children and in the course of exhausting diseases. Since the days when antiphlogistic measures were so freely resorted to in children's disorders, "we have learned," Vogel says (op. cit. p. 385), "that pale anæmic children are as liable to be attacked by Convulsions as robust and plethoric ones." Beyond question the *violence* of fits and their *frequent repetition* are not the clinical signs of active changes in the head. Heat of skin and vomiting, headache, and irregular pulse are the real signs of acute changes in the head—encephalitis and meningitis. But since the brain may become much congested as a consequence of the fits when they are severe and frequently repeated, it is intelligible that some advantage may follow the application of leeches by a reduction of the congestion, although there is no inflammatory process to relieve. I say again that severe and repeated Convulsions are not the signs which should make us deplete generally, apply blisters, or give purgatives largely. Treatment of this sort is admittedly a most fatal mistake in those cases where the fit is but one sign of starvation, as it undoubtedly is in many cases of diarrhœa, and in cases of wrong feeding.

(4.) It does occasionally happen that a child is attacked by Convulsions when in apparently good health. But before concluding that a child is in good health we must, I repeat, consider very carefully whether or not there are signs of rickets, and we "must bear in mind that active rickets and the preservation of a large amount of fat are by no means incompatible." (Gee, op. cit.)

These are the cases in which it is plausible that the convulsion is the result of some temporary cause, such as over-eating; of some removable cause, such as the irritation of teeth during dentition; or of some sudden excessive mental disturbance, such as fright. I consider it very doubtful whether any of these so-called "causes"

[1] "Much *éclat* has lately been made in England and France with the scarification of the gums. Some recommend a crucial incision; others, the removal of the whole cap which covers the head of the tooth. But, as an admonition, it is premised in all the reports and laudation, that the tooth has to be very near eruption, otherwise the scarification will be of no benefit. I have frequently performed this operation, but have always found that the lancinated wounds of an inflamed mucous membrane heal very badly, and ulcerate for a long time; that the nervous symptoms continue notwithstanding, till ultimately artificial or spontaneous diarrhœa supervenes. Indeed, if we have to wait until the tooth is 'very near' breaking through, then the process is in fact near its end, and any other simple remedy is as efficacious as this, which is attended by a considerable amount of pain." (Vogel, Raphael's Trans. p. 107.)

induce fits in children whose nervous systems are healthy beforehand. But it is very likely that they are exciting causes when it is not healthy. I have twice made autopsies on children who have died in fits after a meal, and in each case the stomach was full of food. In one case the child had had fits before, but none for three months before the fatal seizure; in the other the fit which the child died in was the first. If then we find that a child has had a fit soon after a meal which we have good reason to believe was large—say a basinful of soup—we should give an emetic. If we find that a tooth is coming through, we may justifiably lance the gum. But when we have done this we must bear in mind that a nervous system which has given way from such temporary and comparatively slight exciting causes, will be very likely to fail again when again tried by indiscretion in feeding or by eruption of teeth later in the dentition period. Indeed, we must, I think, conclude that the nervous system of a child cannot be healthy if a slight and temporary cause produces a convulsion, however healthy the child may look. We often find that the fits recur when we have done all we can to remove supposed sources of irritation. There are great differences of opinion with regard to the influence of dental irritation. Vogel says (op. cit. p. 387): "Eclampsia, originating from dental irritation, belongs to the serious forms, and often leaves behind it partial paralysis and imbecility." Meigs and Pepper, in their most valuable work on Diseases of Children, say: "As a general rule, the convulsions which depend solely on the process of dentition are slight, and last but a short time. In all the instances that we have seen in which this was the only cause to be detected, the attack was of this nature."

(5.) We will now consider cases in which Convulsion attacks healthy children without obvious cause of any kind, and cases in which they continue when we have removed all temporary sources of irritation. Once more having regard to the important researches of Elsässer, Jenner, and Gee, I would urge the consideration whether in these cases the "healthy" child is not rickety, although often slightly so. It is to be remarked that a child may suffer fits from blows on the head, and occasionally we see patients who have Convulsions after severe blows, followed by indentation of the skull. These cases it is not my task to consider. We are sometimes told by the friends of our little patients that palsy or convulsion followed an injury, but on inquiry we find there is not a shadow of evidence of a blow or a fall having occurred. The friends suppose very naturally that a fit must have a cause, and if their child has a convulsion in the midst of seeming good health, they infer that the child has had a fall. When we find a child the subject of Convulsions for which we discover no cause, or infer none from the condition of the teeth, bowels, and general health, we think of the terms eclampsia and essential. I do not, however, use these terms, for I know no means of distinguishing betwixt an epileptic fit in a child and an eclamptic fit. The practical point is this, and it is occasionally put to us by the child's friends: Is it epilepsy? I take this to mean: Is the illness one which will quickly, in days or weeks, run a course to death or to permanent recovery, or will the child recover from the fit or series of fits, but be liable to occasional attacks of convulsions for years or for life? This seems to me to be the practical question. Now, when we see a child in his *first* fit, we certainly cannot tell, whatever the age may be. The paroxysm is the same in all cases of general Convulsion. There seems to be great unanimity among authors that the eclamptic fit is quite like that of epilepsy. Niemeyer speaks of eclampsia as acute epilepsy. Vogel says it is impossible to distinguish the paroxysm of eclampsia from that of epilepsy. Then epilepsy will occur at any age. Vogel (op. cit. p. 411) states that Beau found, out of two hundred and eleven epileptics, that the disease was congenital in seventeen cases, and that it occurred from birth to the age of six years in twenty-two cases. Nevertheless, Vogel says, "Young children in general rarely suffer from true epilepsy, as we might expect, if the more frequent eclampsia be regarded as a distinct disease." And he adds: "Eclampsia is easily distinguished from the disease under consideration (epilepsy), by the fact that it almost always occurs at the breaking out of an acute affection only; that the general condition of the patient, after the termination of the Convulsions is not restored: and that it is often fatal, while epileptic attacks are almost always devoid of danger." I submit that at the best we can only deal in probabilities. The older the child the more seemingly causeless the fits, the slower the succession—say a fit every other day, or fits scattered at irregular intervals of days or weeks—and perhaps we may say the less rickety the child the more likely are the fits to be epileptic, *i. e.* the more likely is the child to continue for years or for life subject to fits. But I am convinced that we can give a prognosis in no case with anything like certainty. I do not exempt cases where a child has had one fit during the eruption of a tooth or during an exanthem. We can only say, even in these cases, that the child is very unlikely to suffer again. If the child be

partially hemiplegic after a seizure, he is very likely to suffer from Convulsions later in life.

Epileptic fits in adults not rarely date from Convulsions in infancy. The connection is shown now and then by straggling fits at intervals of months or years, or by uninterrupted continuity of attacks at fairly regular periods. I have no facts, and I know of none on record, to show how many children keep well after getting through an illness with severe Convulsions, but I am certain that attacks in infancy—from one which attracted little attention, "we thought," says the mother, "it was only the teeth," to a whole batch —are occasionally followed by epileptic fits near the age of seven, fourteen, or twenty.

In reference to this question, it is important to ask if the child's near relatives have had nervous symptoms. Every medical man can relate instances of fits, or of other symptoms of cerebral disease in different members of one family. I have had under my care a girl of eleven years of age, who has had fits from the age of six months. Her sister, three years of age, had had them from the age of one week: another sister, aged ten, from the age of four years; and a fourth, also a girl aged eight, from six years. Instances so striking are rare. The fact that several of the child's relatives have had hemiplegia or Convulsions from embolism, clot, syphilis, &c., has no bearing whatever on the question. If the child's brother, or sister, or mother had Convulsions in childhood, the evidence is perhaps stronger; but I confess that I have very little faith in the hereditariness of such *symptoms* as epilepsy or Convulsions. The occurrence of Convulsions in several of one family may be because they all suffer from rickets. We may believe that a "nervous temperament" is transmitted, but if so, there will be a predisposition to many nervous affections, and not to one symptom only. At the best it is very difficult to obtain certainty as to hereditariness, as Convulsions are so very common in children, and occur, it is presumable, from numerous pathological processes.

Prognosis.

The prognosis of a symptom with so uncertain a meaning must, of course, be very uncertain, and much has been incidentally said on prognosis. Here may be excluded from consideration the attacks which precede or occur in the course of acute disease. The writers of other articles will speak of seizures so occurring. When a child has had a fit, and appears to be in good health again, the question as to the cause of the fits, with a view to forecast the child's future, becomes again urgent. If there is clear evidence of some source of irritation, and we have got rid of it, we may hope the child will keep well; but I have already spoken of the uncertainty of our hopes in this respect.

By far the most important question in prognosis is whether or not the child is likely to get through an attack or a series of attacks of acute Convulsions with life. In the first place, a single attack may be fatal, but this is a rare event. Wilks relates two striking instances of rapid death with Convulsions. One was a small, delicate child, six weeks old, who was seized suddenly with diarrhœa and Convulsions, and died in a few hours. The other patient was a child four years old, strong and healthy, who died soon after being brought to the hospital. In neither case was anything wrong found with the brain; but in the child four years old the stomach was distended with food. I have mentioned two cases of a like kind. Such cases, however, are rare. The consideration of less acute seizures is more important. We have no generalizations, and can only deal in generalities. The younger the child, the more likely is the result to be fatal. The more frequent or violent the seizures, the more profound the coma; and the worse the state of health in which the fits began, the more likely is the child to succumb. To consider the manner in which fits are likely to bring about death, is the important matter. This will be considered with the treatment, to which I now come.

Treatment.

It would not be correct, as I have remarked, to speak of any purely *rational* treatment of a single symptom, as it means things very different. Our treatment is nearly altogether empirical. And, of course, we exclude entirely from present consideration cases in which Convulsion occurs in such diseases as scarlet fever, meningitis, &c. Even an empirical treatment of Convulsion would not be justifiable in these instances. Our thoughts or treatment go hand-in-hand with our investigations into the cause. Principles of treatment apply to Convulsions of all degrees, from rolling of the eyes to complete seizures.

If we find the child in a fit, we can do little during the paroxysm. We should see that every part of its dress is loosened, that it has a plentiful supply of pure air, and we should direct that it be laid down and kept quiet. Vogel says that by sprinkling the face and exposed chest with cold water, we may succeed in inducing deep spasmodic inspiration, by

which the danger of suffocation at least is lessened.

Then as to general treatment, in the intervals of the paroxysms. I begin with what may be called attention to immediate circumstances. We undress the child, and it is possible we may find that a pin or a needle is sticking in some part of the body, even in the child's head, penetrating the brain. A needle has been found in the liver of a child who died of Convulsion (see Trousseau's Clinical Medicine, vol. i. p. 343, Bazire's translation). Trousseau believes that blisters and mustard plasters are often the causes of fatal Convulsions.

If we find that the fit came on after eating, we should give an emetic of ipecacuanha. If a gum be swollen and tense, we may properly use the lancet. We should inquire after the state of the child's bowels; if they are constipated, we should give a purgative. But none of these things must be done as matter of routine. The presence of diarrhœa, especially, with tenesmus and expulsion of little but mucus, may show that there is irritating matter in the intestinal canal, and it is then proper to give a dose of castor oil. This must be, however, only to make a starting-point for careful dieting. The presence of abdominal pain and constipation in robust children may lead to the suspicion of retained feces. Diarrhœa, which is—paradoxical as it may seem at first glance—sometimes a sign of constipation, would not prevent the moderate administration of purgatives or enemata. For a child at the breast, an enema of an ounce of warm water or of thin gruel may be administered; at one year, two ounces. Very likely the diarrhœa is due to wrong feeding, and to diet the child would then be the most important thing to attend to. When the motions are very frequent, and if the child be thin and weak, we may try to check the diarrhœa by astringents, if proper dieting does not arrest it.

Warm baths are frequently used, and, when there is no great heat of skin, and no thoracic complication, the child may be put in a bath at the temperature of about 96° Fahr. for from five to ten minutes. Under any circumstances the feet and legs may be immersed in warm water. Then mustard plasters — a mixture of mustard and flour—may be applied to the calves of the legs for five, ten, or fifteen minutes.

When we have done all that immediate investigation prompts; when we have attended to the bowels, lanced the gums, ordered proper food, the Convulsions may persist, and may even increase in number and in severity. We are thus urged to do something more.

There is in attacks of Convulsion a tendency to (1) death by exhaustion, from the frequency of the fits and want of sleep; (2) death from asphyxia, from sudden and prolonged fixing of the chest walls, and from slow congestion of the lungs. The latter is often rather a way of dying than a cause of death. Adults die from convulsive seizures in each of these two days.

1. If the child were much exhausted by frequent fits, or if he were weak to begin with; if he were thin, if he had long had diarrhœa, we should look most carefully to his support: we should prescribe beef-tea or juice of meat in abundance. Nor should we hesitate to give stimulants. The circumstances that would guide me most on this point would be the great frequency of other abnormal muscular actions.

The main object in treatment of disordered function of the brain in general, *e. g.* sleeplessness, delirium, and frequent, slight Convulsions, is to produce sleep, and to accomplish this we should give nutriment liberally; and if this fails, stimulants freely. I have already spoken of the importance of recognizing that violent or frequent Convulsions do not depend on inflammatory changes. If the beef-tea or juice of meat were vomited, I should give milk with a little ice, and inject the tea and the juice.

Affusion with cold water has been advised, but this I should not adopt unless the child was robust. Of course I speak of cases in which there is no general fever, and no sign of inflammation of the membranes. A thin, delicate child I should keep warm. Vogel, however, says that affusions of the head with cold water, performed every hour or two, are useful against all Convulsions in children (op. cit. p. 107).

Then as to drugs. Antispasmodics have been given, but I think the best antispasmodics are nutrients and stimulants when these can be taken and digested. It is of course comparatively easy to get drugs into the stomach, but they may be vomited. It may then be desirable to give an enema of asafœtida, *e. g.* 20 to 30 minims of the tincture in an ounce of warm milk. In all cases—Convulsions in exanthematic and other acute diseases excepted—I should give bromide of potassium in large doses, by enemata, if necessary, if the fits were frequent, or if they continued several days. A mixture containing hyoscyamus, two or three drops for the age of six weeks, may be given with peppermint water. The great point, however, is, I repeat, to get the child to sleep, and to do this it is, I think, justifiable, simple nutrients failing, to give stimulants freely. I should, however, be most wishful to give as much nutriment and as little stimulant as possible. Nor, of

course, should I give wine because a child had a fit, but only when the child was feeble to begin with, or was exhausted by the frequency of the attack, wearied too from imperfect sleep, and perhaps starving because the friends had not given enough nutriment before we were called. I should not give stimulants if the thermometer showed a great increase of temperature. Supposing nutrients and stimulants and the drugs mentioned were taken and retained, and failed to stop the Convulsions and to procure sleep, I should then venture to give narcotics. In no case should I prescribe narcotics except when I had ascertained that the child had previously taken nutrients and stimulants, or unless the child was fairly vigorous to start with. In no case, at any age, would I give opiates, when there was great excitement without vigor. It is as hurtful to give opium at this extreme, as in the condition of brain which occurs with general febrile states, and which condition is supposed to be due to congestion. Nor would I give the narcotic more than once in twelve hours, and then I should give a decided dose, *e. g.* a quarter of a grain of Dover's powder under the age of three months; half a grain to a year, and a grain to a year and a half. Chloroform has been used by Sir James Simpson, and, when the above ascending series of remedies—nutrients, stimulants, bromide of potassium, and opiates—have failed, this may be tried. Dr. West says: "In cases where depletion is inadmissible, where the Convulsions are not obviously due to organic disease of the brain, while they are both severe in their character and are returning with frequency, the inhalation of chloroform sometimes altogether arrests them." It is also, he says, of service in Convulsions of a more chronic kind. He tells us, however, that its effects are evanescent; he adds, that he has never seen mischief from its use. "It requires the constant presence in the house of some one competent to administer it." [It is a matter of universal testimony that ether is safer for inhalation than chloroform. Ether has been largely employed in America in the treatment of Convulsions. The remarks just made concerning the administration of chloroform apply equally well to it.—H.]

2. Now I come to speak of the cases where the severity of the individual fits threatens death by asphyxia, or when from the breathing we fear the blood is largely delayed in the lungs. When Convulsions occur in robust children, bleeding is sometimes advised for them. It is a remedy which has been urged by many writers, but does not seem to me—I speak very respectfully—to be likely to be of use; but I have never tried it in any form, either by leeches or otherwise. For I have no faith whatever that Convulsions depend on any increase of nutritive changes that we can arrest by taking blood. There is in children's Convulsions no certain evidence as to the pathological condition of nervous organs. It is, however, often evident enough in robust adults, as when epileptiform seizures follow on blows, and on tearing up of the brain by mechanical injury, or the irritation of tumor, that bleeding could do no good—no good by acting on the cerebral circulation. But it would, I think, be good practice to relieve the venous system when, after repeated fits, the circulation is becoming embarrassed by great congestion of the lungs. Death seems, in these cases, to result from pulmonary congestion. Indeed, I think we neglect to bleed as often as we ought to do, on the principle Markham has laid down. I should be entirely guided by evidence from the color of the lips and the state of respiration, and not by the heat of skin or by shivering. However, difficulty of the respiration so great as these signs imply would mark the advance of a fatal issue, and our treatment could only, I fear, be expected to delay it: when I did deplete, it would be by leeches only.

I have just spoken on the supposition that the fits are frequent and are running a rapid course, and are tending towards death by exhaustion or by asphyxia; but if the fits come on at intervals, as once every day, or once a week, and if from inability to discover what they really did mean, *i. e.* if unassociated with debility, irritation of teeth, &c., we were obliged to class them as epileptic or essential, I should adopt the same treatment as for epilepsy in the adult, supposing of course all general indications fulfilled. I should give bromide of potassium, a quarter of a grain under six weeks, half a grain under three months, a grain above, and a grain additional for every year. Indeed, the bromide is a most useful drug in chronic convulsions from any cause, and in most cases — excepting temporary and acute states like uræmia—I should prescribe it when other remedies failed, whatever was the state of the child. Since, however, there is manifestly an association of Convulsions with rickets in very many cases, it is well to treat the child for this condition in all chronic cases. I shall therefore conclude what I have to say on the treatment of Convulsions by the following quotation from a paper on rickets, by Gee (St. Bartholomew's Hospital Reports, vol. iv. p. 79):—"*Treatment.* This must be radical. And in cod-liver oil we possess a pharmaceutical agent worthy of a place beside iron, Peruvian bark, and mercury. We ought to lose no time over the symptoms of rickets; slight catarrh, diarrhœa, paleness, a tendency to fits, these will all

disappear under cod-liver oil. Give expectorants, purgatives, styptics, and the rickets will increase under our eyes; nay, occasionally it will even develop *de novo* while a child is being treated for coughs, deranged bowels, and other apparently simple disorders."

Convulsions in Adults.

It is a matter of exceeding great difficulty to write on the subject of Convulsion in the adult. We may arrange Convulsions as they are local or general, as they are acute or chronic, as they depend on acute states, such as uræmia, on organic disease, such as tumor, or on changes which we infer to be functional. I shall follow the course adopted in treating of Convulsions in Children. I shall speak first of the varieties of the paroxysm, and then of the causes of the seizures.

There are no doubt innumerable varieties of paroxysm, but for clinical purposes we may arrange most of them in one of two classes.

Class I. The Convulsion begins unilaterally, and begins deliberately. In this kind of seizures consciousness is not always lost, and when it is, it is lost *after* the spasm has begun.

Class II. The fit begins either without any warning or by a very vague one, such as a strange feeling in the head ("giddiness," "swimming," &c.), or by a sensation at or near to the epigastrium which is variously called "sinking," "faintness," "sickness," "fear," and sometimes by children "stomach-ache." In these cases the spasm is more contemporaneous, *i. e.*, it affects the two sides of the body more nearly at the same time, and it affects them more equally. In this class of cases loss of consciousness is the first symptom, which occurs very quickly after the first warning.

It is not pretended that there is an absolute distinction, for in the first class the spasm, in severe cases, spreads all over the body. In the second class it is not usually, probably never is, strictly equal on the two sides. Nor is it strictly contemporaneous; one side almost always, if not always, suffers not only *more* but *sooner* than the other. Moreover we often do not witness the fit, and we may be unable to learn how it began and how it affected the patient. Fits of the first class are almost always chronic. Those of the second also are mostly chronic (epileptic), but frequently acute; they may be symptoms of uræmia, cerebral hemorrhage, &c. In handling the subject clinically we must arbitrarily put in the second class cases in which we can only learn that the patient has had a severe convulsion. In strictness, Class II. is merely a grouping of cases which agree in that Convulsion is the most prominent symptom. We must make a provisional arrangement for clinical purposes, and the above distinction is at least convenient in practice.

The paroxysms of the first class only need be described at length. These fits are far simpler than those of the second class. The patient can tell us more about them; we can, when present at a fit, obtain a clearer idea of what takes place, because the progress of the Convulsion is more deliberate than that of the second class. It is for this reason that I speak at most length on the "causes" of Convulsion, after describing the peculiarities of the paroxysm and its sequelæ in this class.

Class I. The Convulsion begins Unilaterally.

Onset.—The patient or his friends tell us that he is subject to fits beginning by "working" in the hand, or in the face, or in the leg; sometimes there is a local sensation, often named an aura, before there is visible movement. The rule is, that in these cases the spasm starts in the very same place in each seizure. For instance, one patient's fits will always begin in his right index finger, another patient's always in his right great toe. The exceptions occur chiefly in cases of syphilitic disease of the brain. Taking a large number of cases, there is an order of frequency of onset to be stated. The spasm begins most frequently in the hand (usually in the index finger and the thumb), less often in the side of the face and tongue, and rarely in the foot.

Range (*Fits partial or general*).—In the severest fit the spasm first spreads over the side it begins in, then extends to the trunk, and then to the face, arm, and leg of the other side. It is important to observe that the spasm may stop at any stage. There are all degrees, from a slight twitching of one finger to general and severe convulsion. In other words, these fits may be partial or general. Moreover, the same patient may on one occasion have a fit limited to the hand or arm, during which he is quite conscious, and on another occasion the spasm may spread all over his body; he will then become insensible and may bite his tongue. The importance of studying the partial fits is that sometimes they occur for months before a severe fit. A few minutes' steady cramp—not "live blood," tremor, fidgets, or irregular jerks of a choreal kind—on one side of the face, in one hand, or in one foot, demand almost as serious consideration in prognosis as a severe fit does; for a patient who has such

local spasm will probably suffer from severe convulsions.

Ligature.—These are the cases of "epilepsy" in which the fit may often be stopped by tying something round the limb above the part in which the spasm begins (or in which some abnormal sensation (aura) appears), by unclenching the closing hand, or by briskly rubbing it. As Brown-Séquard has insisted, such procedures are as successful in cases of organic disease of the brain, syphiloma for instance, as in cases where there is no evidence of such kind of disease. The patient or his friends may often arrest the progress of fits of this kind. We hear patients remark to this effect, "If I can get the hand open, I have no fit." I am convinced from what I have seen in Brown-Séquard's practice that a garter of blister above the part in which the fit begins will keep off fits of this kind. Dr. Buzzard has recently written an important paper on this method of treatment in the "Practitioner" for October, 1868. In a few, but in very few cases, there is history of injury to the part in which the spasm begins.

Duration of the Attacks.—The duration of the attack varies much. It may last even ten minutes, but in such cases the spasm is long in spreading to the muscles of the thorax.

Consciousness.—These are the cases of Convulsion in which there is often no loss of consciousness whatever. The patient does not lose his consciousness when the fit is partial; the whole of one side may be involved in spasm without any insensibility, but the rule is that consciousness is lost at an earlier stage. For instance, when the fit begins in the hand, consciousness is usually lost when the spasm has largely involved the face, and when the head begins to turn. When it starts from the foot, consciousness is usually lost when the spasm or abnormal sensation reaches the body. There are in these seizures all degrees of impairment of consciousness, if such a phrase be permissible. The patient may assert that in some of his attacks he is conscious, but may qualify his statement by adding that he does not know people about him or where he is; at another time he may be profoundly comatose.

Affections of Speech (Epileptic Aphasia).[1]—In certain of these seizures the patient loses his speech without losing his consciousness. This is not to be confounded with *petit mal.* It most often occurs when the fit begins on the *right* side of the *face and tongue.* Occasionally there is only disorder of speech. Temporary loss or defect of speech is often observed even in cases where the spasm is limited to the right cheek and tongue. The patient may consult us because he occasionally suddenly loses his speech for a few minutes; for instance, he goes to a shop, and on trying to give an order finds that he cannot talk, or he talks so badly that he is supposed to be drunk. We mostly hear that there is local spasm of the right cheek at the same time. (We rarely get any facts about writing in this condition; this part of the subject is too complex for consideration here.) It is hard to describe the *defect* of speech which sometimes *follows* these seizures. The defect is not, I think, quite like the defects of speech which are the result of small destroying lesions such as small clots, limited softening from embolism, &c. There is very much hesitation; the patient slurs his words and mumbles. I use the word defect, because there is never, so far as I have observed, permanent *loss* of speech (complete inability to utter words) after a convulsion beginning by deliberate spasm in the face or hand. If an adult does not talk at all for several days after a "fit," especially if he expresses himself quite well in writing, we should suspect malingering. If there be loss of *voice* with or without loss of *speech* after a "fit," pretence or hysteria is almost certain.

Epileptic Hemiplegia.—These are the cases of Convulsion in which there is so often hemiplegia—epileptic hemiplegia of Dr. Todd. Sometimes hemiplegia follows such seizures, and sometimes it does not. The presumption is that when the spasm is very severe—severe in degree that is, not necessarily, although usually extensive in range also—there is palsy. Thus much, however, is certain, that a patient who has fits of this kind (at all events when the spasm begins in the hand) can never be considered safe from hemiplegia. In very many cases the palsy is trifling, such a "numbness," for instance, as prevents the patient picking up a pin, although he may strike the table pretty strongly; occasionally there is perfect[1] paralysis. The palsy always affects the side in which the fit begins; and when there is *complete* hemiplegia, the hemiplegia is quite like that produced by plugging of the middle cerebral artery. When, however, the spasm is limited in range, the consequent palsy is limited in range. If the arm only be affected by severe spasm, the arm only is palsied when the

[1] "There is a peculiar class of cases of epileptic hemiplegia, in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power and speech." (Todd, "Nervous Diseases," Lect. xv.)

[1] I use the words "perfect" and "imperfect" to express degrees of loss of power, and the words "complete" and "incomplete" to express differences in range of the paralysis.

fit is over; if the leg only, the patient drags it, but uses his arm well. Thus it happens that we may have complete palsy of the arm following a convulsion in which there had been no loss of consciousness whatever. Whether the palsy be complete in range or perfect in degree, it passes off in the vast majority of cases, and according to the degree of palsy, in hours, days, or weeks—perhaps, however, leaving a little numbness—the patient may afterwards speak of his "weak side." Unfortunately, since the patient will doubtless continue subject to convulsive seizures of a like kind, we shall be obliged to admit that he will probably have the palsy again. Indeed he may have another fit even before the palsy from the former one has had time to pass off. More rarely hemiplegia is the first symptom, and Convulsion of the kind I describe occurs after or during recovery. The Convulsion affects first and chiefly the side paralyzed. Since this order of events usually happens in cases where there is valvular disease of the heart, I suppose there is in these cases embolism of some part of the district of the middle cerebral artery.

Frequency.—In this class of cases the fits vary in frequency. They are, I think, more irregular in this respect than chronic convulsions of other kinds. The patient may have one a week, one a month, or he may have thirty or forty, nay literally hundreds, in a few days. When the fits are very frequent, the patient is usually, if not always, hemiplegic and deeply comatose. There is the "status epilepticus." It will be observed that in the paroxysms the spasm still affects first and more strongly the side already paralyzed. The signs of danger in the status epilepticus are not so much the number of fits or the degree of palsy—the frequency of the fits, of course, adds much to the gravity of the case—as increase of temperature, abnormal respiration, and irregular pulse.

CAUSATION.—A convulsion implies discharge of unstable gray matter. We have then three directions of investigation in these cases: (*a*) the seat of the changes in gray matter; (*b*) the pathological processes by which these changes are brought about; (*c*) the circumstances which favor the discharge (exciting causes).

(*a*) *Seat of Lesion.*—The probability is that in many cases we should discover no lesion *post mortem.* Although we must admit that gray matter is in an abnormal condition (because it discharges on slight provocation, and because it discharges abundantly), we must admit also that this abnormality does not involve any *great* alteration of structure. If it were much altered, even disorderly functions would not be possible. We are therefore not likely to *discover* the changes in the gray cells to which exaggeration of their normal function—to store up and expend force—is due. Niemeyer (op. cit.) says, "Experience teaches that the lesions from which abnormally active impulses proceed are insusceptible of anatomical demonstration."[1] Since increase of function, even in disease, implies increased nutrition, we infer that the gray cells affected in Convulsions store up force in large quantity, and reach a high degree of tension. Further, since they discharge on slight provocation—possibly even in periodical normal changes in the body, when by continuous nutrition a certain degree of tension is reached—we must suppose they are in a state of highly unstable equilibrium. Instability, in this article, is made to include two things—high tension and very unstable equilibrium. But admitting that we cannot, or have not, yet discovered the change which permits this duplex condition, we have ground for inference as to the position of the changes of instability.

In some cases we discover gross changes. (For convenience we shall suppose the gross change to be, as it often is, a syphilitic nodule.) The gross change affects the cerebral hemisphere[2] opposite the side of the body in which the spasm sets in. Moreover, in all the autopsies I have seen the disease has been in the region supplied by the Sylvian artery, and has affected convolutions—parts rich in gray matter. But the nodule we find is of course not the direct cause of the seizure—the seizure is the result of a discharge of gray matter, and the nodule is an overgrowth of connective tissue. In other words, the changes in gray matter on which the Convulsions depend are secondary to the nodule. Now, at least two views may be held as to the seat of these secondary changes. The view generally adopted is that they are in the medulla

[1] "The proximate cause of convulsions is an abnormal increase in the nutritive changes of the nervous centres." (Russell Reynolds "On Epilepsy.")

[2] Wilks says ("Pathology of Nervous Diseases," Guy's Hospital Reports, 1866) "that the morbid conditions which we find to give rise to epileptiform convulsions are remarkably uniform. They all point to the presence of local irritation of the surface [of the brain]." Speaking of a case of epileptic convulsions in a patient who had tumor in the pons Varolii—a case which had been supposed to confirm Schröder van der Kolk's "supposition that the cause of epilepsy is seated in this part"—he says, "I have no hesitation in saying that for one such case fifty might be found in which the marked changes producing these symptoms occupy the surface [of the brain]."

oblongata; and that they are the result of an "irritation" starting from the nodule, or rather from its neighborhood. Another view is, that the gray matter changed is near the tumor itself, or in its vascular territory at least. Arguments in favor of the latter view are, chiefly, first, that the muscles first and most affected in the seizures are those most and longest affected in hemiplegia due to the disease of the corpus striatum; secondly, that the epileptic hemiplegia, when complete, is quite like that which results from plugging of the middle cerebral artery. Other reasons could be given. The occurrence of temporary defect of speech in certain of the seizures (*vide* p. 753) is of some value in localizing. It may, however, be said that disease of certain convolutions often causes no symptoms at all, and therefore that when Convulsions occur along *with*, they cannot *depend on*, changes in the convolutions. The word disease is, however, here used vaguely. It is true, as every surgeon knows, that much of the convolutions may be *destroyed* without the supervention of symptoms. The patient can *do without* certain parts of his brain, but if much of its gray matter be very *unstable*, he must have symptoms, for it will discharge strongly, and of necessity put muscles in disorderly movement. It may be asked, How is it, if the patient can do without the part which discharges, that there is sometimes hemiplegia? The explanation is admittedly speculative. The hemiplegia comes on *after* the paroxysm. Suppose the fit to begin by discharge of unstable gray matter in a certain convolution, the violent impulse thus originated will probably discharge lower and yet related centres of healthy gray matter. At all events, the nerve fibres to the muscles, and the muscles themselves, are suddenly in excessive function. I suppose, then, that the hemiplegia remains until the normal conditions of these suddenly overworked parts have been slowly restored by nutrition. Wilks (op. cit.) says the paralysis follows by "an inhibitory action."

As my plan, however, is chiefly clinical, I have discussed the question of position very briefly. It suffices for the discussion of the next question to admit, as we must, that there are changes of gray matter in *some* part of the nervous system.

(*b*) *The Pathological Process.* — The changes of instability may no doubt be brought about in many ways; but we shall limit ourselves to these questions: "Are the changes the secondary results of gross organic disease—tumor, for instance—or are they minute changes, often inferable, rarely, if ever, discoverable?" If there are symptoms pointing to gross organic disease, we fear that the patient will die of that disease; if there are not, our fears are of a different kind, viz., that he will be subject to fits for years or for life, or, as it is often said, will be an epileptic. The first question then is, Is there gross organic disease?

If for no other reason than to economize space, it is well to consider first the symptoms which show that there is gross organic disease of *any* kind; and next, to consider the evidence by which we infer its particular nature—whether, for instance, it be syphilitic or not. Before beginning our task, it may be well to point out that certain symptoms which young practitioners sometimes rely on are not to be relied on, either for or against the diagnosis of gross organic disease.

Tongue-biting is of no value in the diagnosis of the cause of any kind of convulsion: it is only a sign of severe convulsion. It occurs in cases of the first class described, if the fit be general and severe, and does not occur if the fit be partial and slight. It occurs in severe fits from uræmia, cerebral hemorrhage, tumor, &c. Nor does the condition of the pupils during the fit furnish any evidence in diagnosis. Very great inequality of the pupils *after* the fit would point to organic disease. In these cases there may be, as has been remarked, no loss of consciousness when the fit is limited in range. It was said by the late Dr. Bright that absence of insensibility in convulsive seizures is some evidence that the lesion is organic. With very great deference, I must say I cannot accept this view. If we were to judge by post-mortem evidence alone, we might draw the conclusion; but we see patients who have convulsion without loss of consciousness, who not only show no signs of organic disease, but who, except for their seizures, seem to be in good health. Post-mortem examinations, in cases where there has been no evidence of organic disease, are very rarely had. I have not yet seen one. The fact that the fit is partial, let us say limited to the arm, the transitoriness of epileptic hemiplegia, the absence of fits for months, do not negative the existence of gross organic disease. It is to be especially insisted on that quick recovery from epileptic hemiplegia is of no value whatever in negativing organic disease, tumor for instance. The signs I have mentioned are of no value for or against the diagnosis of gross organic disease in this class of convulsions.

The evidence which warrants the diagnosis of gross organic disease in cases of Convulsion is of a different kind. We have carefully to distinguish betwixt the symptoms which are owing to local destruction of the nervous system and those owing to changes diffused in nervous masses about the destroying agent—the

results of the irritation it excites. For instance, a certain form of hemiplegia is owing to the destruction of fibres and cells of the corpus striatum by, let us instance, a clot. This symptom is special to the part injured; injury to no other part produces it. Again, it is primary, for it comes on at once, from destruction of nerve fibres there seated. But, next, the patient suffers headache; his temperature rises, his pulse and his respiration become irregular. Such symptoms may be called general, because they do not point to disease in any one part of the encephalon, and they may be called secondary because they do not come on at once, but are indirect results of the irritation of the clot as a foreign body—of a local encephalitis. Hence we see that whilst the *nature* of the lesion matters little or nothing so far as the production of such special and primary symptom as hemiplegia is concerned—it suffices that nerve-fibres in the corpus striatum are by *any* means *destroyed*—it matters very much with regard to the general or secondary symptoms. The encephalitis in cases of clot is often a rapid process, but the same distinction is to be made in cases of tumors, syphiloma, abscess, and other kinds of gross organic disease. But it frequently happens that tumors, syphilitic disease, &c., occur in regions of the brain—in the cerebrum and cerebellum—large parts of which may be destroyed without the production of any special symptoms—without hemiplegia or obvious mental defect. In other words, the patient does not suffer because the tumor has *destroyed* a certain part of his brain. He begins to suffer when a local encephalitis is excited by the destroying agent. And as this encephalitis does not always occur, he may have no symptom whatever from cerebral tumor.

What is the evidence which shows a patient the subject of a convulsion to have gross organic disease within the cranium? The symptoms which show there is organic disease within the cranium, not in these cases only, but in any cases—in cases of palsies of cranial nerves, hemiplegia, &c.—are such as severe headache, urgent vomiting, and *double* optic neuritis. The pain in the head has no value in diagnosis if it be the temporary sequel of a severe fit, or of a series of fits. We must be satisfied that the patient's "headache" is really pain, and not "confusion," "giddiness," "weight on the top of the head," &c. Nor can we lay stress on it as evidence unless it be intense, and unless it has lasted for some days or weeks. We can sometimes judge of its intensity by the patient's manner and by his expressions. He gives up work; he may remark, "it is not a common headache." He is said by his friends to "rave," and sometimes it is reported that he "knocks his head against the wall." The headache is of more value in diagnosis if it occurs in unusual places. If it be at the back of the head shooting forward, or on one side—I do not refer to nodes nor to neuralgic pain extending into the face—gross disease is likely. The vomiting is urgent, it is purposeless and capricious sometimes, for instance, occurring only at night or in the morning. The vomit is frothy "like phlegm," the patients sometimes say; and if there be very much retching, as there usually is, it is greenish or yellowish. The tongue may be quite clean, and the appetite may be good. Vomiting is not always present. When symptoms so well marked have lasted for several weeks, we suspect that the convulsion is the result of gross disease. We may, I think, be quite certain if there is also paralysis of the whole of any one motor cranial nerve. If there be double optic neuritis as well, or its usual sequel, double optic atrophy, we may be almost absolutely certain.

Let us suppose we have satisfied ourselves that there is a gross lesion of some kind, we have now to find out what is its particular nature. We may, I believe, exclude clot in nervous centres as a cause of chronic seizures of this kind. No doubt effusion of blood on the surface of the brain would produce fits of this kind, and in all cases we should inquire carefully for history of injury to the head, and seek for evidence of chronic renal disease, the two chief conditions under which meningeal hemorrhage occurs. I have only twice known albuminuria to occur with fits of this variety, and there is, I think, no warrant for the supposition that uræmia has anything to do with their causation. It is true that Convulsions called uræmic—after scarlet fever, for instance—are often unilateral, but as far as I know these seizures do not begin by a very deliberate spasm in one side. When they do, their real nature may be inferred by examining the patient's heart, urine, &c.

The gross organic disease may no doubt be of many kinds, but practically the point we wish to determine is, Is it syphilitic, or is it some other kind of new growth? It is needless to mention that when other symptoms of syphilis are present, such as nodes, the diagnosis of syphilis is almost certain, and needless to urge in all cases of this kind a very careful investigation for evidences of syphilis, such as scars on the skin, holes in the palate, white marks on the tongue. It is only necessary to speak of cases in which such decisive evidence is not to be had. In the first place the gross disease is frequently syphilitic. It is so with the very rarest exceptions when there is also double optic neuritis. It is next to certain that there is syphilitic disease if the patient

has Convulsions of this kind along with complete palsy of the whole of any motor cranial nerve; for one great diagnostic mark of syphilis is that it produces random associations of symptoms. The evidence is clearer if the motor cranial nerve paralyzed be on the same side as that on which the Convulsion begins, and on which there may be epileptic hemiplegia, because we are sure then that there are two lesions. (The facial paralysis, which is part of epileptic hemiplegia, is not of course included in the expression palsy of a motor cranial nerve, because it is not owing to disease of a nerve *trunk*.) If there is palsy of any nerve trunk, *e. g.* of the radial, palsy of one leg, or paraplegia, the great probability is that these symptoms and the Convulsions are owing to syphilis.

Still there may be some other kind of new growth, but this is very rare in cases of Convulsion of the class described, and rarer still when the Convulsion is attended by any of the other symptoms mentioned in the preceding paragraph. In some cases the recovery of the patient from local palsy, let us say of the third nerve, by iodide of potassium will make the diagnosis pretty certain. In other cases the length of time, *e. g.* many months or several years, the symptoms had lasted, would point to syphilis. There can be no doubt that we should treat for syphilis.

Fits of this kind occasionally follow blows on the head. In these cases we should carefully inquire for evidence of syphilis, as syphilitic disease of the brain is frequently "lighted up" by injuries. In some cases there is a depression of the skull on the side opposite the side of the body in which the fit begins.

Suppose now that there is no evidence of gross organic disease of any kind. In the vast majority of cases we can get no further, we can only infer that there are *not* gross changes in the brain, and as a corollary that the patient will not soon die, but will continue subject to fits. In a few cases there will be found evidence which will warrant the supposition that the plugging of small arteries of the Sylvian region is the cause of the pathological change.[1] It is certain that patients, the subjects of valvular disease, have seizures of this kind after or during recovery from hemiplegia. Yet although I have made post-mortem examinations of patients, the subjects of valvular disease, who have had Convulsions, I have had no post-mortem examination of one whom I knew to have had fits of the kind described. My supposition is that patients who have "epileptic fits" from intracranial aneurism suffer really from local embolism, and that when the aneurism is of the middle cerebral artery, or of some large branch of this vessel, the seizures will be of the first class. It is, however, held by some that the fit depends on irritation by the aneurism. Mr. Callender (St. Bartholomew's Hospital Reports, vol. iii. 1867) has made the very important observation that the "epileptic attacks belong to aneurism of the middle cerebral artery."

(c) *Exciting Causes of the Paroxysm.*—Some patients who are subject to fits of this kind are otherwise in very good health. Such cases are sometimes supposed to be owing to some very general cause. A patient who in the midst of good health has had a severe convulsion is naturally most anxious that his fit should be attributed to some very general and removable cause, and will dwell much on such facts as that he had taken something that had disagreed with him, or that he was in a close room, or will say that he was "bilious," or worried by anxiety the day it happened. Dyspepsia, overwork, fright, and the like, may be admitted to be factors in causation. I cannot, however, conceive that any such general conditions can alone produce fits which time after time begin in one hand and even in the very same finger even for months and years. In other words, I cannot conceive that they alone can determine the discharge of *healthy* nervous tissue in some particular locality. I class them as exciting causes, believing that there is some central change as well. Whatever view may be held, there is for therapeutical purposes a complete agreement that we should try to remove all such causes. We may find that the patient is dyspeptic. It is, I think, quite certain that in some cases the *paroxysm* frequently *comes on* when the patient is flatulent. So, although we may differ as

[1] The inference is not that each fit or each series of fits depends on separate pluggings. It is true that sudden plugging of the middle cerebral artery, or perhaps of some large branch, may lead to a *severe* convulsion in a patient whose nervous system was previously healthy, but in these cases there is persistent hemiplegia after a fit. If a smaller branch be plugged and perhaps slowly occluded, the hemiplegia passes off or diminishes greatly, and, as before said (p. 753), occasionally the patients become subject to convulsion beginning in some part of the region previously paralyzed. The hemiplegia depends on destruction of nerve fibres, the occasional spasm depends on instability of gray matter. It is evident enough that plugging will lead to destruction (softening even to diffluence) of fibres and cells, but it is not sufficiently borne in mind that plugging of small vessels may lead also to increased quantity of blood beyond the plug, and thus to altered nutrition and instability. At autopsies on patients who have died of or with plugging of the middle cerebral artery, whilst we find softening of part of the corpus striatum, we find also at the periphery "red softening."

to the way in which dyspepsia is connected with the seizure, there is no question that to treat the dyspepsia by careful dietary and by medicine is a matter of the very greatest importance. (See Dr. Paget, of Cambridge: Lectures on Gastric Epilepsy, *Lancet*, 1868.)

These seizures, like other convulsive attacks, and other nervous symptoms, sometimes follow fright. The first fit of a series evidently depending on organic disease may follow fright so closely that we are driven to believe there is some relation betwixt the two things. I imagine that the fright merely *determines* the paroxysm which some other cause would afterwards determine. There is nervous tissue in a state of highly unstable equilibrium, which will surely discharge soon from some provocation, and now and then fright is that provocation. As no special point of treatment is involved, and as the discussion of such causes belongs rather to epilepsy, nothing further need be said here.

As to local irritation by worms, teeth, &c., all that need be said here is that we should try to remove these sources of irritation. Then possibly the part of the nervous system diseased, ceasing to be worried by such eccentric irritations, may cease to discharge.

Class II.[1]

Let us now suppose the patient's fit to be one of the second class. These cases cannot be considered on the same plan as those of Class I.; the paroxysms are too sudden, and the conditions under which they occur are too numerous and complex. In chronic cases of this class the same reasoning as to position, nature of change, and exciting cause will to a very great extent apply. We shall include in this class those cases of which we obtain no history of the mode of onset of the fit. For instance, we find the patient comatose, and we only learn that he has had a convulsion. It is then that the question arises, Is the fit epileptic? This is the great question when we see a patient in or soon after his first convulsion. But since we may find him hemiplegic, it will be best to use the expression epileptiform, and modify the question thus, Does the fit depend on a state of the brain or system which is such that the patient will recover from the fit in all probability to suffer similarly again and again for months or years, or is it owing to such causes as cerebral hemorrhage, tumors, uræmia, &c., which will soon lead to a fatal result?

In the first place, the phenomena of the convulsion—it does not begin by deliberate aura in one limb—the nature of the paroxysm, stertor, coma, tongue-biting, furnish no reliable evidence. Cases of apoplexy from cerebral hemorrhage are now and then diagnosed as cases of epilepsy, because the apoplectic condition was ushered in by a fit of an "epileptic character." It is to be insisted on that neither the kind of convulsion nor its repetition are signs serving in the diagnosis of the *nature* of the lesion. Neither enables us to say whether the fit is epileptic or not. Nevertheless it is freely admitted that in most cases we are right in the prediction we make. Although it is difficult to make a diagnosis, it is easy to guess. If we are called to a young man who has had a severe fit and who is not paralyzed after it, and if we find that he has recovered or is recovering consciousness, we shall be right in the great majority of cases if we say, without any further medical examination, that the fit is one of epilepsy, and not one of uræmia, cerebral hemorrhage, &c. But it is not necessary even to see the patients to make diagnoses which shall be *generally* right. And when we hear that a patient has been long subject to fits, from each of which he quickly recovered, or if we hear that he has had attacks of *petit mal* only before the convulsion, we shall be right in nearly all the cases when we make the diagnosis of epilepsy. But even under these circumstances the practitioner will be wrong now and then. For instance, there may be chronic renal disease, notwithstanding the patient has had fits of an "epileptic character" months before. Again, the former fits may have been due to aneurism of one of the larger cerebral vessels, and the fit we are called to may be owing to rupture of that aneurism. The former fits may have been owing to tumor, and the one we are called to may be the result of fatal hemorrhage from that tumor. These are rare cases, but we are sure to meet with them now and then. If we do not consider these rare possibilities, we may make very painful blunders.

I repeat that a routine diagnosis of epilepsy in young people who have a convulsion will rarely be wrong, because such Convulsions are nearly always epileptic. And those who do not examine the urine unless the patient be dropsical, and who content themselves by saying in cases of death by Convulsion that the patient "died of an epileptic fit," and make no post-mortem examination, will not be aware that this diagnosis is sometimes grossly wrong.

In what follows, in order to encounter fully the difficulties of diagnosis which actually do occur, and because I have only to do with the most commonly occurring seizures (epileptic) in diagnosis, I will suppose that we are called to a person in

[1] See page 752.

his first fit, or first series of fits, and only incidentally notice what bearing on our diagnosis the fact of the previous occurrence of fits has. There is no position more embarrassing than that we are in when called to a patient in his first fit. As before said, it is easy to be *generally* right.

We will consider some of the recognized causes of Convulsion, or, it may be safer to say, the known conditions under which they arise. It will be well first to remark however, that when we are called to a patient who has "died in a fit," we must ask if the patient were eating when the fit came on. Dr. Lalor has written a valuable monograph on death by choking in epileptic attacks, and I could relate a case in which I feel convinced that death was thus caused, although the larynx was not examined *post mortem*.

Renal Disease (*Uræmia*).—In all cases of Convulsion we must examine the urine, however young the patient may be, and however healthy he may look, and notwithstanding that he has had fits described as epileptic on previous occasions. This examination is still necessary when the patient has recovered consciousness by the time we reach him. None of the above circumstances negative uræmia, nor is the quick repetition of fits of value in diagnosis. The fact that the patient has had no dropsy does not influence us. We must, I repeat, examine the urine. If it be smoky; if there be scarlet fever in other members of the patient's family; above all, if the patient be the subject of scarlet fever, we conclude almost with certainty that there is uræmia. I say almost, because now and then Convulsions in scarlet fever are followed by a liability to Convulsions for life. As we sometimes say, "some cases of epilepsy date from scarlet fever;" occasionally they leave persistent hemiplegia. It is hard to believe that there can have been uræmia only in these cases. Since endocarditis occurs now and then in scarlet fever, and since plugging of the middle cerebral artery will cause Convulsions, it is as likely that there is embolism as uræmia.

Cerebral Hemorrhage.—The mere presence of albumen, however, does not lead us to declare that there is uræmia. In a patient past middle age there may be cerebral hemorrhage. If there be hemiplegia with deep and continuing coma, we diagnose hemorrhage, and we do this notwithstanding that the patient is young—say twenty—and notwithstanding that he was "quite well before the fit." If there be no hemiplegia, and if the patient be young, the inference is very strong that there is uræmia, and not clot. But, as will be mentioned in the article on Apoplexy, a general convulsion followed by deep coma and universal powerlessness, in a patient whose urine is albuminous, may be owing either to very *large* hemorrhage into the cerebrum, into the lateral ventricles, or into the pons Varolii, or it may be owing to uræmia. We cannot rely on stertor, kind of coma, repetition of convulsion, or increase of temperature, although rapidly *increasing* stertor, rapidly *deepening coma*, are signs in favor of the diagnosis of hemorrhage. It is the *ingravescence* of these symptoms, after the convulsion, which favors hemorrhage. We will now suppose that there is no albuminuria.[1]

Cerebral Aneurism.—Dr. John W. Ogle and Dr. Murchison have pointed out that epilepsy (*i. e.* fits at intervals, like those usually called epileptic) occurs in patients the subjects of aneurism of large cerebral arteries. Such aneurisms will occur in young people, and therefore the question of age has no bearing on diagnosis. There is nothing special, so far as has yet been determined, in the kind of convulsion; there are, indeed, no symptoms which are characteristic of cerebral aneurism. There may be no symptoms at all, or none sufficient to send the patient to a doctor, until the fatal ones from rupture of the aneurism. We cannot therefore be certain whether a patient's fits are the results of cerebral aneurism or not. If, excluding albuminuria, syphilis, and other causes to be afterwards mentioned, we have reason to believe that there are vegetations on the heart's valves, we may surmise that the fits are owing to cerebral aneurism or (*vide supra*) to local embolism in connection therewith.[2]

We can occasionally diagnose that a fatal seizure is owing to rupture of a cerebral aneurism, and this is an important matter in a medico-legal point of view. If a young patient has had Convulsions now and then for months or years, and if after one severe fit he is more profoundly comatose than usual, with great stertor, and if he continues so for some hours, rupture of a cerebral aneurism is probable. It is all the more likely if the patient *has* been hemiplegic. If the patient dies in the fit in a few minutes or in half an hour, we are more certain, because we know that it requires a large and, what is more important, a sudden hemorrhage to kill quickly.[3] We are more sure still if

[1] Occasionally when there is chronic renal disease, we discover no albumen in the urine, and it is said that occasionally, after a severe convulsion, albumen appears in the urine in consequence of that convulsion. I do not see how we can avoid mistakes in these cases.

[2] See Dr. Gull on Cerebral Aneurisms, Guy's Hosp. Rep., vol. v. (3d series).

[3] It is not, of course, said that rupture of aneurism of the large cerebral vessels always kills suddenly or quickly, nor always by Convulsion. Rupture leads to death slowly,

the patient is known to be the subject of valvular disease of the heart, or if he has had rheumatic fever. Dr. J. W. Ogle and Dr. Church have shown, and my experience bears out their conclusions, that aneurism of the larger cerebral vessels frequently occurs along with vegetations on the heart's valves.

Embolism.—Embolism of the middle cerebral artery sometimes, although rarely, produces severe convulsions. It is followed by hemiplegia. The modes of onset of symptoms from plugging vary much. They sometimes come on suddenly and sometimes deliberately, according, the presumption is, as the vessel is slowly or suddenly plugged. Again, the degree of the symptoms varies. There may be no loss of consciousness, and the hemiplegia may be transitory. This is so when the branch occluded is small. If a patient the subject of valvular disease becomes hemiplegic after a severe convulsion, it is considered to be almost certain that there is sudden plugging of the main trunk or of a large branch of the middle cerebral artery: it is not quite certain. The convulsion and consequent hemiplegia may be owing to rupture of a large aneurism of this vessel. Aneurism of the middle cerebral artery usually ruptures so that the blood is poured out external to the brain, and the patient dies quickly because the blood gets out in large quantity, and, what is more important, with great rapidity. But occasionally it ruptures so as to *break up the motor tract*, corpus striatum, nor thalamus, and will then, so to speak, imitate common cerebral hemorrhage. If then a patient, especially a young patient whom we know to be the subject of valvular disease of the heart, becomes hemiplegic after a convulsion, we must take this rarer possibility into consideration.

If the coma be very deep, if it deepens, or, generally speaking, if the patient quickly gets worse, rupture of an aneurism is at least as likely as embolism. With all our care we shall be wrong now and then, as patients sometimes die in a few days in an apoplectic manner, from plugging of the middle cerebral artery. (See also Art. Softening.)

Tumors.—If the patient, especially if he be a young and healthy-looking man, have had for weeks or months severe pain in the head, vomiting, &c., there being no albuminuria,—above all, if there be also double optic neuritis,—a tumor of the brain is probable. Although headache is one of the symptoms of cerebral aneurism, cerebral aneurisms rarely if ever cause intense and persistent headache.

if the rupture of the aneurism is small, or if the blood, as when the aneurism is far in the Sylvian fissure, can only get out slowly.

Here I must refer to the evidence stated in more detail (p. 758). But in this connection one further fact is to be mentioned, viz., that a patient may have occasional convulsions for weeks or months from the "irritation of a tumor," and may die after one severe convulsion, or several quickly recurring convulsions, due to large hemorrhage from that tumor. If, then, we find a patient whom we infer to be the subject of cerebral tumor who has been seized with convulsions much more severe than usual, and if he becomes unusually deeply comatose, and if the coma deepens, especially if there be no further convulsion, we fear large hemorrhage from the tumor.[1]

Syphilis.—As before said, if the convulsion be of the first class, and if there be signs of organic disease, there is usually syphiloma of the brain: but if the convulsion be general, or if we know nothing of its mode of onset, we must infer from the evidence of present syphilis. If there be such demonstrative evidence as nodes, &c., or a clear history of recent syphilitic changes in any part of the body, our diagnosis is practically certain. If there is not such evidence, we may judge from the history of a random succession of nervous symptoms, such as palsy of a cranial nerve, followed by hemiplegia or paraplegia, or from the previous disorderly association of nervous symptoms, showing several lesions, *e. g.* palsy of the third or fifth, or portio dura with hemiplegia of the *same* side. When, however, such symptoms are of recent date, they may be still rarely owing to tumor. We must, however, always in these cases treat for syphilis.

Abscess.—Again, it is possible that there may be cerebral abscess. If we are to ignore this possibility altogether, we shall very rarely err, as cerebral abscess is very rare. I have, however, more than once been consulted for Convulsion which turned out to be owing to cerebral abscess. There are no certain points in diagnosis except the presence of bone disease in some part of the cranial wall, most often the bones of the ear, occasionally at the vertex. If the only evidence there is be that the patient a week or month ago received a severe blow on the head, the fit may be owing to *syphilitic* disease of the surface of the brain. It probably is, if the fit begins deliberately, and if there be epileptic hemiplegia. Syphilitic disease of the brain not infrequently follows blows on the head. If, however, there be no evidence of syphilis, no palsy of any cranial nerve—excepting amaurosis from

[1] Of course it is not to be implied that hemorrhage from cerebral tumor necessarily leads to Convulsions, any more than ordinary cerebral hemorrhage does.

double optic neuritis, which is scarcely to be called *palsy* of a cranial nerve,—if we find that there is a "puffy" tumor on the scalp, abscess is probable, and very probable if there be hemiplegia after the Convulsions. Occasionally, as is well known, a patient suffers from cerebral abscess without any symptoms at all, or any obvious symptoms. Occasionally after a period of latency it breaks into the lateral ventricle: then the symptoms are quite like those of hemorrhage into the lateral ventricles, and we can only make the diagnosis of what has occurred from evidence of blows, disease of the bones of the ear, of the nose, &c. If there be no history, and if we find no evidence of disease of bone, we cannot make a diagnosis.

Epilepsy.—Supposing now that we can negative the above causes, we conclude that the patient has had an epileptic fit. By this we mean that he has had a convulsion which does not depend on an organic lesion, or on an acute state like uræmia, or on a sudden quasi-accident like hemorrhage. We infer that he will quickly get into his usual health, but that he will in all probability have fits of a like kind again and again for years. We say probably, because now and then he does not suffer again, and it not infrequently happens that after the first fit or the first series of fits he has an interval of good health for many months.

Death in Convulsions.

Now and then, however, a patient dies in a convulsion, and we discover nothing *post mortem* which we can suppose to have been the cause of the fit. Of course there is something overlooked, and we should always search every organ of the body with great care in these cases. When the patient is known to have had fits of a like kind before, we may say that he died in an epileptic fit; but when it is his first fit, this nomenclature does not conceal the bald fact that a patient seemingly healthy has a convulsion the cause of which we cannot make out, even after post-mortem examination. He dies in it, we surmise, because it has been unusually severe, respiration, and probably the heart's action, having been suspended too long for recovery. The point that chiefly concerns us here is that such modes of death are well recognized, and do not indicate either violence or the administration of poison.

Treatment of Convulsions in Adults.

Obviously enough, treatment will vary so much in different cases that most of what has to be said will be found in the articles Uræmia, Cerebral Hemorrhage, Embolism, and Epilepsy. Indeed, I wish only to say a word on the treatment of Convulsions due to syphilis. In these cases we treat for syphilis, but in chronic cases, at least, this treatment is not of so great service, so far as removing the symptoms goes, as from superficial considerations we should expect. The bromide of potassium is of more service than the iodide in keeping off fits.

[*Puerperal* eclampsia is not alluded to in this article. Its full discussion belongs to works on Gynæcology. A few words, however, upon it may not be inappropriate here.

Convulsions *during pregnancy* may be either uræmic, reflex, or apoplectic. Pressure of the distended uterus upon the renal veins may interfere with the normal condition and action of the kidneys. Of this state of things albuminuria is an admonitory sign. When it amounts to such a degree as to produce marked uræmia, Convulsions may follow. If parturition be safely accomplished, the Convulsions disappear with the uræmia.

In women having excito-motor irritability greatly developed, there may be, in the absence of uræmia, Convulsions from irritation of the gravid uterus, acting reflexly. Here, also, delivery results in the cessation of the tendency to eclampsia.

A plethoric state exists during pregnancy in some women, predisposing to cerebral congestion.[1] Either *before*, *during*, or *after* delivery, one concomitant of this condition may be Convulsions; whose prognosis is always very serious. A fatal termination is least apt to occur in the utero-reflex cases; it is most frequent in the apoplectic. Signs of this form are, deep flushing of the face, distension of the vessels of the neck and head, full, sometimes slow, pulse, and stertorous respiration.

Treatment.—If, during pregnancy, a woman known to have good vigor of system, with a full pulse, warm skin generally, and hot head, is attacked with Convulsions, it is good practice to draw blood from the arm. About ten ounces will usually be enough; if the pulse be quickly reduced, less will do. An enema of castor oil, soap, and warm water may be administered. A large sinapism should be applied to the back, and cold water to the head. If the convulsion be prolonged, the

[1 The opinion of Cazeaux and Traube, that, instead of plethora, there is hydræmia in such cases, is not likely to be correct. That of Frank, Munk, and others, that the symptoms are due to high arterial tension, has more to sustain it. This may explain the symptomatology of many cases.—H.]

patient may be placed in a warm bath. After this, should further treatment be called for, inhalation of ether or chloroform may be suitable as a last resort.

When the convulsions are evidently utero-reflex, not uræmic, nor accompanied by apoplectiform congestion of the brain, bleeding is out of place. Sinapisms to the back and limbs, and the warm bath, will be proper, and the use of ether or chloroform by inhalation may be earlier resorted to. Nitrite of amyl (a few drops, by inhalation) has been employed in some such cases with benefit. When the convulsions show a tendency to repetition, bromide of potassium may be used, or hydrate of chloral may be given in twenty or thirty-grain doses; or morphia may be administered by hypodermic injection. Either of these remedies will require close watching of their effects upon the patient.—H.]

EPILEPSY.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—Epilepsy is a chronic disease of which the characteristic symptom is a sudden trouble or loss of consciousness, this change being occasional and temporary, sometimes unattended by any evident muscular contraction, sometimes accompanied by partial spasm, and sometimes by general convulsion.

The two elements probably present in every case of Epilepsy are diminution of intelligence and excess of muscular contraction; and these two elements may exist in almost every variety of combination, and be developed to any degree of intensity. The latter element is not always seen to exist; there may be no spasm of the facial muscles, not the slightest change in the expression of countenance; or the face may become dull in aspect, or pale in color, but consciousness is, for the moment, in absolute abeyance. There are reasons for thinking, as will be shown hereafter, that this loss of consciousness depends upon spasm affecting the vessels of the pia mater, but such spasm is hidden from our eyes. The former element, loss of consciousness, is that which is essential to our idea of Epilepsy; without its occurrence, no convulsion, however severe, should be regarded as epileptic; when it does occur, as a paroxysmal event, and with a chronic history, the case is one of Epilepsy, although no other symptom may be present.

There are two classes of errors into which authors have fallen with regard to the use of the word Epilepsy. The older mistake was to apply the term to every case in which there were convulsions appearing in a certain form, called "epileptic," "epileptoid," or "epileptiform;" the modern error is to use the word to denote a paroxysmal—*i. e.* occasional and sudden—loss or diminution not only of consciousness but of any function of any organ; or, indeed, sometimes to denote anything, or any condition, which occurs in a paroxysmal manner. The former led to the association, under one name, of diseases differing so widely from each other as tumor of the brain, Bright's disease of the kidney, intestinal entozoa, lead poisoning, and almost every form of malady: the latter might lead to the placing in one common group, and calling by one common name, such diseases as amaurosis from dyspepsia, stammering, deafness, paralysis, or asthma. The former tendency led to the production of such words as renal epilepsy, symptomatic and sympathetic epilepsy, toxæmic epilepsy, and the like: the latter has conduced to the coinage of such terms as epilepsy of the retina, acoustic epilepsy, and so forth.

There is, I think, a radical and very mischievous mistake in both of these modes of using words; the error is similar in the two, as far as regards its principle, but it differs in the detail of its development. The older authors exaggerated the importance of the form of a group of symptoms — convulsive — occurring in a number of organs, and common to many widely different diseases; the modern have exalted into undue prominence the pathological significance of one element out of this group of symptoms—viz., arrest of function—which single element may occur in many diverse organs of the body. By such a term as "renal epilepsy" was meant a disease resembling Epilepsy in its outward form, but dependent upon, not an unhealthy condition of the nervous centres, but on an irritation of the kidney, or an altered blood-state which kidney-

disease might have determined: by such words as "retinal epilepsy" something very different is intended, viz., a malady showing itself only in the retina, in which a change takes place, supposed to be analogous, in its intimate pathology, to that occurring in the brain in Epilepsy. In the one Epilepsy merely means convulsion; in the other it merely means arrest of function; and the objection I entertain to such use of terms is based upon the fact that, however widely different individual cases of Epilepsy may be, they do yet belong to and constitute a group which has a definite clinical history, and has had it for some hundreds of years. If good reason can be shown for getting rid of the word "epilepsy," I should rejoice to lose it from our nosology; but so long as the word is retained at all it should have a definite and intelligible meaning. Renal asthma would be a term as pathologically correct as "renal epilepsy;" dyspnœa of the fingers as justifiable as the expression "epilepsy of the retina."

Synonyms.—No useful end would be served by enumerating all the names by which this disease has been described, inasmuch as many of them have fallen into complete disuse. The most important are the following:—

Epilepsy (English); l'Epilepsie (French); Fallsucht (German); Mal Caduco (Italian); Epileptica passio, Morbus sacer, M. comitialis (Latin); Ἐπιληψία, Ἐπιλῆψις (Greek).

Natural History.—1. General Prevalence of the Disease.—Epilepsy is spoken of as a very common affection. Niemeyer states that in every thousand individuals there are to be found six epileptics.[1] Such statement cannot, I think, be true with regard to Epilepsy in this country; for among 1820 invalids, whose cases were recorded by myself as out-patients of the Westminster Hospital, there were only seven epileptics; and but thirty-four whose diseases could by any possibility be confounded with Epilepsy. It must be observed further, that Niemeyer is speaking of individuals generally, and that the results of my own examination at the Westminster Hospital are obtained from a small class of individuals, viz., those who are ill.

The proportion of true Epilepsy to other diseases of the nervous system has been found to be about 7 per cent.

2. Causes of Epilepsy.—(*a*) *Predisposing Causes.—Hereditary taint* has been found to exist in rather less than one-third of those cases which have fallen under my care, and have been carefully examined on this point.[1] It is not intended by this statement to affirm that true Epilepsy has existed in the parents of one-third of the cases; but that some disease of the nervous system, more or less closely allied to that under consideration, has been present in either the parents, the grandparents, the aunts, uncles, brothers, or sisters; that there has been a family proclivity to nervous disorder, in one case showing itself by idiotcy, in another by mania, in a third by convulsions, and so forth. I have found only 12 per cent. of epileptics giving a distinct history of Epilepsy in other members of their families; a number which is very near to that stated by Dr. Sieveking, and not far removed from that given by M. Delasiauve.

It has been said that the disease is more frequently transmitted on the fathers' than on the mothers' side,[2] but the reverse of this proposition has been found to obtain in cases examined by myself.

Of 130 epileptics, I found 80, or 61·06 per cent., who asserted the entire absence from their families of any predisposition to nervous disease; and 8 individuals, or 6·10 per cent., who were in some uncertainty as to the health of important relatives. These patients were derived from all classes of society; and I have no means of determining the question, on a scale sufficiently large to be satisfactory, whether Epilepsy is more commonly found to be hereditary in the upper, the middle, or the lower classes. Several elements of doubt enter into the solution of this question, the most important of which is the greater difficulty that is encountered in obtaining accurately the facts which belong to the latter. Hospital patients often know but little of their antecedent or even collateral relations. Among the upper classes there is not rarely a studious concealment of what are regarded as prejudicial family conditions. The middle classes are not only more accurate than the former, but more free than the latter; and, judging from what I have gathered

[1] Niemeyer, Handbuch der speciellen Pathologie, p. 637.

[1] In a careful paper by Messrs. Leech and Fox, in vol. i. of the Manchester Medical and Surgical Reports, p. 198, the proportion of those epileptics in whom hereditary taint was traceable was somewhat higher, viz., 36·8 per cent. These observers compare with each other epileptics and non-epileptics, and, having obtained particulars with regard to the health of a large number of the relatives of each group, show that the relatives of epileptic patients were found to suffer from "some form of nervous disease" in larger proportion than were those of non-epileptic individuals.

[2] Esquirol. Des Maladies Mentales, tom. i. p. 406.

from them, as they shade off on either side—above them and below—I should be of opinion that hereditary taint is more frequently discoverable in the better conditions of life than in the poorer. It is not intended that there are absolutely a larger number of hereditary epileptics among the former than the latter; but that, of an equal number of epileptics in the two extremes of society, a larger proportion will furnish evidence of hereditary taint among the rich than among those who are in want. This is probably due to the fact that the latter class are exposed more frequently and more severely than are the former to the most active determining causes of the disease, viz., anxiety, alarm, and want. With regard to the hereditary transmission of Epilepsy, as indeed with regard to the causation of all diseases by supposed hereditary taint, it must be remembered that, inasmuch as the large majority of cases owe their malady to other causes than inherited tendency, a certain number of those whose parents exhibit a like affection to their own may have become morbid independently of any hereditary taint. It is well known that many of the children of epileptic parentage are free from the disease, and it is quite clear that many epileptics, descended from epileptic stock, have been exposed to causes of the malady which would, of themselves, have been held sufficient to have induced the malady independently of any constitutional taint. It is, therefore, of practical importance not to assume too readily the operation of this cause, and hence to neglect an examination into other conditions. In the largest and most correct sense of the word, the etiology of Epilepsy is advanced but little by the discovery of hereditary taint; the causation may be thus thrown backwards, but it is not explained.

Sex.—Little that is of value can be shown with regard to the influence of sex as a predisposing cause of Epilepsy. Practically, the two sexes appear to be about equally affected; and the different statements that have been made by various authors—some of whom represent the male sex, others the female sex, as the more liable to the disease—may probably be accounted for by other circumstances than that supposed, viz. a special sexual predisposition. The relative number of female epileptics who are out-patients of hospitals may be determined by the hours at which the physicians make their visits, or by other conditions which have to do with the social position of the applicants, and which may render it easy, difficult, or almost impossible for either the one or the other sex to attend.

Similar degrees of fallacy, although different in kind, may influence the results obtained from private practice. The facts of a physician's age, and single or married condition, for example, might exert an influence upon the relative numbers of his male and female patients too great to be counterbalanced by proclivity to Epilepsy inherent in either sex.

Again, the statistics gathered from asylums are liable to disturbing causes so far as etiology is concerned. In proportion to the amount of disease a larger number of males than females find their way into public asylums. The reason for this is obvious, viz. that men are prevented from doing their special work in the world by an amount of disease which need not deter women from performing their domestic duties. Yet further, the statistics furnished on this point by some authors are complicated by limitations as to age, and by the fact of more or less clearly pronounced insanity of mind.

Little, then, that is definite can be stated on the influence of sex, as a predisponent to Epilepsy; and it seems to me to be the wisest course at present to leave the question open for further investigation.

Age.—The influence of age in the production of Epilepsy is strongly marked. This is shown in the following short table of cases collected by myself:—

Age at commencement.	Males.	Females.	Total.
Under 10 years . . .	10	9	19
Between 10 and 20 years	66	40	106
Between 20 and 45 years	25	20	45
Over 45 years	1	1	2
	102	70	172

The most important fact to be recognized in the above summary is the great frequency with which Epilepsy commences between 10 and 20 years of age—*i. e.* at a period of life embracing the processes of the second dentition and of the establishment of puberty; and, without going much further into detail, it may be stated in addition, that by far the larger number of the group showed their first symptoms of the disease between the ages of 13 and 17 years, inclusive. Further, that there is a comparative immunity from the commencement of the disease between 25 and 35, the greater proportion of cases forming the third group having been seized by the disease at or about the age of 40.[1]

[1] For further information on these points the reader is referred to Hasse, in Virchow's Handbuch, 1ster Abth. 4ter Bd. p. 264; Reynolds on Epilepsy, p. 126; Leuret, Archiv. Gén. de Méd. 4me Série, 1843, t. ii.; Sieveking, Med.-Chir. Trans., vol. xl. p. 158; Herpin, Du Pronostic, &c., p. 332, Leech and Fox, in Manchester Medical and Surgical Reports, p. 199. It is, however, to be remembered that in the case of some of the authors referred to care has not been taken to separate Epilepsy from other convulsive diseases.

When there is a marked hereditary taint as a predisposing cause of Epilepsy, the disease is found to develop itself somewhat earlier than under other circumstances. The difference, however, is not so great as that which is to be observed in regard of some other maladies which are held to be hereditary. The difference may be fairly represented in the following table :—

	Hereditary.	Non-hereditary.
Commencing under æt. 15	83·33	46·15
above "	16·66	53·82

It has appeared, further, that when Epilepsy is hereditary it shows itself at an earlier age among girls than among boys. The difference is not great, and Messrs. Leech and Fox have arrived at an opposite result.[1]

(*b*) *Accidental or Exciting Causes.*—Patients and their friends often exhibit a very great anxiety to refer the outbreak of Epilepsy to some external condition, which they may speak of as its cause ; and, in doing so, they occasionally attach undue importance to trivial circumstances. There is a natural reluctance to admit the presence of constitutional or hereditary taint, and an eagerness to find excuses for the poor sufferer, in the fact of his having been exposed to some extraordinary disturbance from without. In this way we may in some measure account for the wideness of the range of conditions to which the production of Epilepsy has been referred. It is so difficult to conceive that a disease having such strongly marked features as those of the epileptic paroxysm can lurk in an apparently healthy frame —that all the essential conditions of so terrible a malady may be present and yet give no sign—that many find an explanation of the outburst in some externally disturbing cause which they can appreciate, and ignore the operation of those internal conditions which had hitherto escaped their notice, or had been regarded from a different point of view.

It is important to classify the causes to which Epilepsy has been referred, and I have done so by distributing them into four groups : placing in the *first*, those which operated through the mind or the emotions, such as fright, grief, worry, and the like ; in the *second*, those which acted through the reflective centre, such as eccentric irritations ; in the *third*, those which produced their effect through changes in the general health, such as those which may be occasioned by pregnancy, by acute specific, or other diseases ; and in the *fourth*, those which may be regarded as acting physically, such as insolation, mechanical injury, and the like. It is difficult to determine into which category of causation some cases should be placed ; as, for example, those in which the fits have been referred to either falls or blows, inasmuch as it is possible that such accidents may have operated through the mind by alarm or fear, rather than through the body by the merely physical process of concussion or laceration. I have placed such cases in those groups to which they had been assigned by the patients or their friends at the time that the disease began.

The following table exhibits the relative frequency of the several kinds of causes to which I have referred :—

Nature of Cause.	Number of Cases.
I.—Psychical ; such as fright, grief, worry, overwork	29
II.—Eccentric irritation ; dentition, indigestion, venereal excesses, dysentery, &c.	16
III.—General organic changes; fatigue, pregnancy, miscarriages, rheumatic fever, scarlet fever, diphtheria, pneumonia . .	9
IV.—Physical influences; blows on head, falls, insolation, cuts	9[1]
	63

Besides these sixty-three cases, I have the records of sixty-one cases in which no cause could be assigned ; the patients or their friends either asserting their absolute inability to make any reasonable conjecture on the matter, or hazarding some explanation which was utterly nonsensical. It is important to know that of these sixty-one, there were forty-three individuals who, after examination and cross-examination, and suggestion, could give neither to themselves nor to me any clue to the solution of the mystery. Of 124 cases, therefore, sixty-three, rather more than the half, supposed that they could explain the causation of their malady ; forty-three, or 34 per cent., asserted their utter inability to do so ; while twenty-nine, or 23 per cent., referred their attacks to mental or emotional disturbance.

The frequency with which mental or emotional disturbance has been shown to be the cause of Epilepsy is such that it requires some further notice. The most common conditions that I have witnessed are those of continued anxiety and prolonged rather than intense alarm. I have in a very few instances found that an over-strain of the mental powers has been followed by Epilepsy, but in almost every one of these cases there has been considerable anxiety, as well, and it, I believe,

[1] Op. cit. p. 202.

[1] Messrs. Leech and Fox found a much larger proportion of cases falling under this category. Op. cit. p. 206.

has been the more efficient factor of the malady. Women and girls have much more frequently than either men or boys referred their attacks to emotional disturbances; the proportion being 36 per cent. of females, and 13 per cent. of males. The period at which the first attack has occurred after an individual has received some great mental shock varies widely; the fit may take place at the moment of alarm, or it may follow after an interval of hours, days, or weeks.

With regard to eccentric irritations, it must be remembered that in the list given above cases of "convulsions" are not enumerated. Both the first and the second dentition, and even the cutting of the "wisdom teeth," may be attended by convulsions, which in the large majority of cases disappear as soon as the source of annoyance has been removed. In a few rare cases, however, the processes referred to have appeared to cause genuine Epilepsy, and it is to these rare cases that reference is made. It is curious to know that in not more than half of the cases of Epilepsy can it be ascertained that "fits" have occurred during infancy; and it is a still more interesting fact that epileptic women appear to exhibit no high degree of proclivity to puerperal convulsions. Dr. Tyler Smith states that puerperal convulsions occurred only twice in fifty-three deliveries of fifteen epileptic women;[1] and so far as my own experience extends, it is exceedingly rare, and indeed almost unknown, for epileptic women to suffer from their attacks during or immediately after labor.

Among the second group of causes appears one to which, I believe, far too great an amount of importance has been attached, viz. excessive venery or masturbation. It is very common to hear suspicions expressed upon this point; much more common, I think, than to hear any such statement of facts as should prove that Epilepsy and masturbation have any special character or frequency of relation to one another. The one is a tolerably prevalent disease, the other a very widely distributed vice. There are multitudes of epileptics with regard to whom no such suspicion could ever be entertained; and there are, it is to be feared, much larger multitudes of masturbators who have never become epileptic. When, therefore, we find the two elements combined in the same individual, it is necessary to observe some caution in our attempt to interpret their relations. It is, I believe, sufficiently well proved to be regarded as a fact that the vice referred to is liable to induce various disturbances in the health, and that the major part of these is brought about by, and is exhibited in, the altered functions of the nervous system; but what it appears to me is yet wanting in proof is the special relationship of Epilepsy to this particular wickedness or weakness. Again and again it has occurred to me to see cases of vague and various nervous derangements which might be fairly inferred to be the result of masturbation; but it has in only an exceedingly small number of cases of Epilepsy been possible for me to establish the existence of such relation.

There can, I think, be no doubt whatever as to the existence of an intimate association between various forms of nervous malady and either various abnormal conditions of the sexual organs, or unnatural circumstances attending upon their exercise; but, as yet, the nature of that association is, I believe, and as undoubtedly, unexplained. Sometimes sexual excess, and sometimes the reverse; now great emotional involvement, and now the entire absence of all sympathy; at one time exuberant enjoyment, and at another disappointment or disgust, are conditions met with in epileptics, and in all forms of many sorts of disease; but, so far as I know, neither one of those conditions is more frequent than another in the history of epileptics. I have known cases in which morbid libidinousness occurred in epileptics, but only long after the development of the disease; and on the other hand I have met with cases where the sexual propensity had become diminished, or even extinct, after the occurrence of the attacks, and this without any previous excess in its gratification.

In endeavoring to determine this question, which is of considerable etiological interest, it would be undesirable to omit notice of the striking effects which have been observed to follow the administration of bromide of potassium in cases of Epilepsy. It cannot be doubted that this medicine is highly valuable in diminishing the number of attacks;[1] and the only point of interest to us now is to ascertain whether its *modus operandi* is such that it either countenances or discountenances the prevalent belief with regard to the etiological question under consideration. When this medicine was first introduced by Sir Charles Locock,[2] it was recommended as being of especial service in those cases of Epilepsy in women in which the attacks occurred only during the menstrual period; and since that time it has been very generally received that bromide of potassium possesses strong antaphrodisiac properties, and that its

[1] Lancet, 1849, vol. xxiv. p. 644.

[1] See p. 780 on Treatment, Dr. S. W. Duckworth Williams' Paper "On the Efficacy of the Bromide of Potassium in Epilepsy and certain Psychical Affections," also a paper by the editor in "The Practitioner."

[2] See Lancet, May 20, 1857, vol. i. p. 528.

utility in Epilepsy is to be accounted for by its special action upon the generative organs. From the very first I saw reason to doubt this mode of explanation,[1] and much enlarged experience has, from my own mind, removed all doubt whatever upon the point, and produced a settled conviction that bromide of potassium, when given in such doses as to be of service in Epilepsy—viz. from 10 to 30 grains either three or four times daily—exerts no recognizable influence upon either the sexual propensity or power. It is not asserted that doses might not be given so large as to exert such influence, but that where decidedly remedial effects have been produced in Epilepsy, their production has not been attended by any change in the generative functions. Dr. Duckworth Williams[2] says: "I have tried it (KBr) in every variety of uterine affection that has come within my reach, including nymphomania, satyriasis, menorrhagia, amenorrhœa, dysmenorrhœa, &c. &c., but without perceiving the least benefit accrue." Dr. Williams mentions cases in which the patients, in spite of their taking the medicine, "persisted in their bad habits, and their sensuality became if possible more confirmed" (p. 17); and his experience on this matter is in entire accordance with the results of my own observations. We cannot, therefore, support the prevalent creed in regard of one mode in which Epilepsy is produced by facts gathered from the treatment of that disease by bromide of potassium.

To what degree the view to which I refer is supported by the observations of Mr. Baker Brown,[3] must depend partly upon the therapeutic results of this mode of treatment, and partly upon the interpretation which must be given to the alleged facts. On the former point the evidence is unsatisfactory, being gathered from a small and too exclusive selection of cases;[4] on the latter point some misconception is possible. Considered *etiologically*, we want to know the proportion of cases in which the particular cause to which Mr. Brown refers had been in operation, but upon this point we are not furnished with any evidence whatever, inasmuch as in *all* the cases he records not only was irritation of the pudendal nerves believed to exist, but a certain kind of operation was performed. It would, I think, be pushing much too far the inference to be drawn from Mr. Brown's little book, to assert that his opinion is that *every* case of Epilepsy is produced in the manner described. What we want to know is the number of cases of Epilepsy in which Mr. Brown entertained no such suspicion, and, still further, the number of cases in which, having entertained it and acted upon it, the result was unsatisfactory. As to the interpretation of the facts that are stated, there is this to be borne in mind, that so far as I can understand Mr. Brown's theory, it is not that in such cases there have been of necessity immodest wishes, excessive sensuality, or irregular practices, but that there has been a morbid condition of irritability of a certain nerve, and that this has been taken away by the removal of the peripheral termination of the nerve. Referring for future consideration the question of the therapeutic propriety or desirability of the operation of clitoridectomy,[1] all that is necessary to say now is that—in the absence of any definite statement of Mr. Brown upon the question of proportion as described above—my own experience would lead me to believe that the cause he refers to is of very rare and very exceptional occurrence.

In another work[2] I took some pains to show how extremely rare it was to meet with a case of Epilepsy in which no causative conditions could be discovered. Although in one person we might find no predisposing cause, and in another no exciting cause, in only one-eighth of the cases was there an absence of both. In seven-eighths, either one, two, three, or more causative conditions of disturbance were present and were recognized.

The proportion, therefore, of cases of Epilepsy in which the causation of the disease is placed beyond explanation by our present knowledge of pathology is not greater than that which we meet with in many other chronic diseases, and is far less than that which is admitted to exist in several. There is some mystery in the causation of almost all diseases; and I do not think that it is greater in the case of Epilepsy than in that of many others with regard to which we think ourselves on easy terms with the science of pathology.

3. Symptoms.—It will be convenient to consider those which occur in, and constitute a paroxysm of Epilepsy, separately from such as may be observed during the intervals of attack. We have, therefore, to describe, first—

The *Paroxysmal Symptoms*, or features of the *attack*. In most characteristic cases of Epilepsy there is an entire loss of consciousness in conjunction with a peculiar series of involuntary muscular movements; but, on the one side of these typical cases, we see epileptics in whom the loss of consciousness is alone obvious,

[1] See Author, on Epilepsy, p. 332.
[2] Loc. ant. cit. p. 16.
[3] On Certain Forms of Insanity, Epilepsy, Catalepsy, and Hysteria in Females
[4] See Treatment, p. 781.

[1] See p. 781.
[2] Author, on Epilepsy, p. 261

and, on the other, individuals exhibiting certain highly marked spasmodic phenomena, and only very slight or even imperceptible obscuration of the mind. It is necessary, therefore, to classify cases, in order to render description possible, and it is proposed to do so by dividing them into four groups, which may be thus distinguished: First, those in which there is loss of consciousness without evident spasm; second, those in which such loss of consciousness is accompanied by local spasmodic movement; third, those in which it is attended by general tonic and clonic convulsion, following a particular order; and fourth, those in which general or partial convulsion occurs without complete loss of consciousness. The first and second forms may be termed "epilepsia mitior," or "le petit mal;" the third form "epilepsia gravior," or "le haut mal;" and the fourth "epilepsia abortiva," or irregular Epilepsy.

(*a*) *Epilepsia Mitior, or "Le Petit Mal," without evident Spasm.*—All that occurs and can be positively attested in cases of this description is a sudden, temporary, but absolute arrest of both perception and volition. The individual so attacked loses consciousness for two, three, or more seconds; and may after that or a longer period resume his sentence or employment perfectly unaware that anything abnormal has happened.

Sometimes there is slight loss of balance—the patient, if standing or walking, leans to one side, or staggers, but does not fall; sometimes there is pallor of the countenance followed by slight flushing; sometimes the latter without the former; sometimes there is slight dilatation of the pupil, and an absence of the expression of "looking at anything;" sometimes an irregularity and faltering of the pulse; but often, as I can testify from repeated observations, there is not any one of the physical changes I have mentioned; the patient's mind becomes a blank for a few seconds, and that is all that can be observed.

These seizures are often regarded as "faintings," and are described by patients under various terms, such as "blanks," "forgets," "faints," "sensations," "absences," "darknesses," &c. &c.

Occasionally these slight attacks are preceded by vertigo; the patient thinks he shall fall, and so lies down to avoid doing so: sometimes he staggers and grasps an object for support; but, much more commonly, he simply ceases to perform any act requiring volition—he stops speaking or writing; but the automatic movements of standing or sitting, and the secondarily automatic movements of riding, walking, or holding an object, are maintained.

Sometimes the attack is followed, for a few seconds or for a longer period, by an obscured or altered state of intelligence; the patient speaks in reply to what is asked of him, but in half an hour afterwards is found to have entirely forgotten what was said to him or by him. In more rare cases the mind is dull, or altered from its habitual condition for a period of some hours, the patient being low-spirited, or suspicious, and apparently laboring under some delusion which he afterwards forgets. In this condition he may be listless; or he may do some odd things which he cannot afterwards account for or even recollect.

(*b*) *Epilepsia Mitior with evident Spasm.*—This is more common than the preceding, which it resembles exactly so far as the mental condition is concerned. The extent and locality of the spasm differ widely in different cases, and also in the same individual at different times. There may be only slight strabismus, or drawing of the mouth, partial turning of the head to one side, or some movement as of swallowing or attempts at getting something from the mouth; or, on the other hand, there may be slight momentary rigidity of the whole body. Sometimes the patient fixes his chest walls, and appears to "hold his breath; sometimes he does some curious thing, such as stoop down to peep under a sofa, lie down and pull off his cravat, jump from his chair and walk quickly half-way across a room; but in any or all of these apparent attempts to do something he is suddenly arrested by the loss of consciousness, which is often absolute. Occasionally peculiar actions are performed after an attack of "le petit mal;" but it has never occurred to me to find an epileptic who could tell me why he did these things, or who could even remember that he had done them.

As to the locality of the muscles affected, it would appear that those of "expression" and of respiration are by far the most frequently involved. There is no evidence to show that either "trachelismus,"[1] or "laryngismus," or "phlebismus" occurs with anything like such frequency as to make them of any value in the interpretation of the epileptic paroxysm: although it is quite clear that the former may exist to such a degree as to occasion duskiness of the face.

The spasm in "le petit mal" is never violent; and it is only of short duration. It is tonic in its character, and painless to the patient, and the vascular changes which may be observed are of the same variable degree and kind as those enume-

[1] Marshall Hall, "Memoirs on the Neck as a Medical Region," 1849.

rated in the previous section. Patients sometimes have warning sensations of these attacks, and I have known more than one instance in which there was a highly marked and most painful "aura epileptica."[1]

The most common combination and degree of symptoms may be thus described,—a feeling of giddiness, faintness, or discomfort; slight twisting of the neck, with anxious, lachrymose expression of the face, dilatation of the pupils, and pallor; accompanied, or quickly followed, by entire loss of consciousness, which lasts for two or three seconds; the patient "becoming himself again" after making a few sighing sounds, but feeling faint and bewildered, and often perspiring freely.

(c) *Epilepsia Gravior, or "Le Haut Mal."*—This, the ordinary form of Epilepsy, is in the vast majority of cases characterized by complete loss of consciousness, and a peculiar combination and series of spasmodic movements. In very rare instances we have the latter element without the former; the more common and much larger group shall be described first, and it will be convenient to enumerate separately the premonitory symptoms, those of the attack, and the immediate sequelæ or after-symptoms.

Premonitory symptoms are sometimes absent altogether; in certain cases they are of regular occurrence, being in the same individual invariable in character, while in another set of cases they are sometimes absent and sometimes present, and are more or less variable in their features. Their duration may be almost momentary, it may extend to several minutes, or, but very rarely, to hours, or even days. When of long duration, the prodromata are diminished in specialty and in intensity; and consist, as far as I have seen, in some mental change, or in some alteration of the general appearance. Thus, there may be an exaggeration of any habitual condition of the mind or spirits; the patient becoming, to an unusual degree, depressed, morose, or taciturn; or, on the other hand, lively, irritable, and excited. I have known several instances in which an undue flow of spirits, and an emphatic frivolity and expression of "feeling remarkably well," have almost invariably preceded the epileptic paroxysms. Such sensations have occurred in those patients whose attacks were not of very frequent repetition. It is very difficult to describe those changes in general appearance, or in "the looks" of a man, which friends recognize as premonitory of an attack. Generally, I believe, they depend upon an alteration in the color of the skin, and some want of fineness in the outline of the features. The face becomes less red, more yellow, and somewhat dusky in tint, and there is a certain puffiness which, without altering in kind, diminishes in force its habitual expression. It is said, "He seems quite himself, but he does not look so; he is sharp enough, but looks stupid; and we know that an attack is coming on." There is no œdema, but there is a partial obliteration of the lines which make up "expression."

Those symptoms which *immediately* precede the seizures are widely different in character, variability, intensity, and duration. They may occur in the mind, the sensations, the muscular system, or the general bodily condition.

The mental prodromata are of many kinds: in some cases there is a distinct idea, never spontaneously presenting itself at any other period, and one which in its character and bearing is perfectly remembered afterwards; while in others there is a vague notion, recognized to be the warning of an attack, but of such indistinct character that only the fact of having entertained it is remembered. One gentleman told me that just as an attack was coming on he always thought, "This is what I had foreseen, I knew it would come on here, I ought to have avoided it by remaining away;" and this, although there had not been the remotest suspicion beforehand that an attack was imminent, or that the circumstances about to be entered upon would be likely to induce it. Much more common is a vague feeling of fear, which is horrible enough, but happily of only short duration.

Sensorial changes are by no means uncommon, and they are of every kind, description, and indescribability. By far the most common is a "painful feeling," sometimes said to be "most painful," or "horribly painful and distressing," but which, yet, the patient says—when minutely questioned—is not "pain," in the ordinary sense of the word. It would seem to be some condition of sensation which is intensely distressing, but which is unlike what we mean by smarting, burning, aching, &c. &c. Patients sometimes say that "it is in the head," and yet it is not "headache;" that it is in the epigastrium, and yet it is not "stomach-ache." In some cases the sensation is always in either the head, the epigastrium, or lower thoracic region, the lower abdominal region, or the limbs. These are stated in the order of frequency, as they have occurred to me. In a large number of individuals, however, the sensation—which is sufficiently distinct and consistent for them to know that an attack is coming—is so vague that they cannot assert whether it is in the head, chest, abdomen, or limbs. Sometimes there are

[1] *Infra.*

hallucinations of the special senses; one patient told me that he always heard "an infernal noise, something like that outside a booth at a country fair;" another that he had "a vision of a hideous donkey." It would be waste of space to enumerate further these prodromata.

Premonitory symptoms may occur in the form of tremor, twitching, tonic spasm, or co-ordinated movements, such as turning round, running some distance, &c.; or they may appear as partial paralysis of one or more limbs. The latter is stated to be more common in old people.[1]

The term epileptic "aura" has been sometimes used, very vaguely, to describe any premonitory symptom of which the patient could give an account; but, when more strictly limited in its meaning, it has been used to express a sensation of blowing, or of something analogous thereto, which, commencing in the periphery, passed upwards to the head, the patient becoming insensible when it had reached this point. Passing over for the present the pathological interest attaching to the interpretation of the so-called aura, it may be now stated broadly that anything characteristic of Epilepsy, in the second or limited sense of the word "aura," is rare, but that when such premonitory symptom does occur, it varies in character in different individuals; in one class there is a pain in the limbs, which "runs up them towards the head;" in another there are some twitching movements, and "the leg draws up," or "the arm becomes contracted;" and in a third there is some vague uneasiness about the hypogastric or epigastric regions, which "goes up through the chest." One peculiarity attaching to these symptoms is the facility with which they may sometimes be removed, and the attack averted. Pain may be stopped by rubbing, or by the pressure of the hand, or of a ligature; contractions may be undone by forcible extension of the limb; and the uneasiness in the abdomen may be removed by a cordial draught.[2] The duration of the aura is very variable, viz., from a few seconds to several minutes; sometimes the feelings "come, and go again," for hours, being arrested many times in the manners I have mentioned, but at last, as the patients say, "slipping by," and being followed by the fit.

There are, further, premonitory symptoms in the vascular system, and in the secreting organs, such as alterations in the color of the face, or of the fingers, a redness or duskiness of the lips, a blue color of the gums, an excessive salivation, a change in the nature of secretions;[1] but these are of rare occurrence, and of such variable character that the mere fact of their existence is all that need be stated here.

The relative frequency of the different classes of premonitory symptoms, so far as I have been able to ascertain, may be represented thus:—

Mental and emotional .	11·1 per cent.
Sensational	19·8 "
Motorial	8·6 "
Vascular and secretory .	3·7 "

Prodromata were declared, positively, to be absent in 40·7 per cent.,[2] whereas information was "doubtful" in regard of 16 per cent. The most common precursory sensation was vertigo; there was little difference to be observed between the relative liability of the two sexes to any one form of "warning."

Actual Symptoms of the Attack.—For the purposes of description it is desirable to divide the epileptic paroxysms into three stages.

In the *first* stage of the attack there are the following phenomena, which occur—not successively, as they are necessarily represented in writing, but simultaneously, or with only slightly varying order:—

Loss of consciousness, *i. e.* of perception and volition.

Tonic contraction of the muscles throughout the body, with some excess of power on one side, or in one direction.

Impeded or arrested respiration, with or without a crying noise.

Pallor, redness, or duskiness of face; either the one or the other, often the one succeeding the other in the order they are mentioned.

Dilatation of the pupils of the eyes.

Natural, weak, or imperceptible radial pulses, with throbbing carotids and distending veins.

This stage lasts from two or three to thirty or forty seconds.

The loss of consciousness is usually sudden and complete; the patient falls down, or is, as it were, "thrown down" in a moment, with or without warning; but even when the warning occurs, so that he may change his position, or call attention to his wants, habitually the passage from consciousness to unconsciousness is abrupt, and the loss absolute. Sensation is, at the same time, in abeyance; although some reflex acts may be excited.

[1] Tissot, Traité de l'Épilepsie; Œuvres, tome vii. p. 131.

[2] See cases recorded by Author, op. cit. p. 92.

[1] Romberg, Manual, Syd. Soc. Transl. vol. i. p. 198.

[2] Messrs. Leech and Fox give a smaller proportion. Op. cit. p. 218.

The tonic spasm of the muscles is peculiar, and it may precede the loss of consciousness. The patient usually appears to be straining round towards one side, as if striving to look over and behind one of his shoulders. The muscles of the face and front of the neck are those which most frequently mark the onset of the paroxysm.[1] The eyeballs, the head, the arms, and the trunk, turn and twist round, so as to give the impression I have mentioned. There is universal strain, but not actual equilibrium. Every limb is rigid, every muscle is at work; but some one set of muscles in each limb proves slightly stronger than its opposing set; and the limbs pass slowly, in a stiffened manner, and sometimes with slight jerking movements, from the positions that they occupied before the attack commenced. The head, neck, and trunk share in a similar movement, and its direction is usually uniform in the individual epileptic.

Respiration is arrested, the patient appearing just like a man forcibly "holding his breath;" and in nearly half of the cases which have fallen under my own observation, the stoppage of the breathing has been so complete that no sound whatever has escaped from the mouth. In a certain number of individuals the respiration proceeds without actual interruption, but its movements are diminished in force; whereas in a very small number there is no change whatever.

In an uncertain proportion of cases there is the "epileptic cry," a peculiar and hideous sound, of which there are two distinct varieties. Some individuals utter a yell at the very commencement of the attack, and just before there is the peculiar holding of the breath I have described. Others do not "cry," but emit a groaning sound, which is, as it were, squeezed out of them by the quasi-tonic contraction of the muscles of the chest. There is, in fact, in regard of respiratory movement, a condition analogous to that observed in the limbs and trunk, viz., that of strain, but of imperfect equilibrium. As in the limbs there is a stiffened movement, from the fact that one set of muscles overcomes its opponents, so in the chest, sometimes a slow expiration, sometimes an inspiration is performed, and with either of these there may be a groaning sound. Usually there is but one sound—either a yell or a smothered groan; there is no repetition of either the one or the other.[2]

[1] In twenty-four of forty-two cases. Leech and Fox, op. cit. p. 222.

[2] I state this as the result of special attention to this point, as in a singular case, occurring many years ago, the question of the possible number of "cries" an epileptic might make assumed some importance in a medico-legal inquiry.

Pallor of the face is observed immediately before, and at the very onset of the attack in many; it is not present in all; and it occurs more certainly and more notably in females than in males. In other instances the face remains absolutely unchanged in regard of color, whereas in a larger number there is suffusion of a florid dull red, or dusky hue. Messrs. Leech and Fox found pallor to exist in only 38 per cent. of their cases, whereas duskiness was "very marked and present all through the fit" in 53 per cent.[1]

Dilatation of the pupil occurs, and, so far as I have seen, invariably, at the onset of the attack. In one case, however, I witnessed a momentary contraction before dilatation commenced.

The pulse, as felt at the wrist, is usually small, and is sometimes quite imperceptible; but in several cases that I have observed there has been no change whatever in either the force or rapidity of its beats. When it has been imperceptible, there has been highly marked tonic spasm of the limbs; and often at the same time the heart may be seen, felt, and heard to be acting, and that even forcibly, and there is obvious throbbing of the carotid arteries.

In the *second* stage of the attack there are the following symptoms:—

Persistent unconsciousness.

Clonic convulsion.

Laborious breathing, with gurgling, foaming, and the like.

Darkness of face, and body generally, with cold and often profuse sweating.

Oscillation of the pupils.

Throbbing, labored pulse, and palpitation of heart.

This second stage may last from a few seconds to five or ten minutes, its features gradually passing into those of the third stage. The transition from the first to the second stage is abrupt, and is determined by what may be termed the "letting go" of the breath which had been "held" before.

Clonic spasms are, more or less, universal; often they begin in the extremities, and are more highly marked on one side of the body than on the other. The jaws are champed together, the tongue is bitten, the limbs are thrown about, the bladder, rectum, and vesiculæ seminales may be evacuated; there are rumbling noises in the intestines, hiccup, and vomiting. The eyeballs are rolled outwards, and in every direction but that which is natural, and the aspect is as hideous as can be conceived.

Respiration is violently and convulsively performed; the diaphragm may be felt through the abdominal walls; the chest heaves; the alæ nasi are forcibly dilated;

[1] Op. cit. p. 224.

and the patient is in the condition of one who has made a most violent effort, and is now "out of breath." Mucus is heard rattling in the trachea, and is often blown out of the mouth, bloody from the bitten tongue or cheek. There is obviously great excess of secretion, and much of the distress of the sufferer appears due to his want of power to get rid of it.

Duskiness or lividity of the surface appears to increase, and it reaches its maximum just as the clonic spasms begin to abate in their severity, and the second stage passes into the third. The sweating is often excessive, and in some cases has been observed to have a peculiarly fetid odor.

The pupils vary from contraction to dilatation, and back again, not, however, becoming so widely dilated as they were at the onset of the seizure; and they are, to some extent, influenced by exposure to light.

The veins are greatly distended, especially those of the throat; the heart beats tumultuously; friends of patients say, "It seems as if it would beat through the chest;" the carotids throb, and the arterial pulsation everywhere is violent, and the vessels are full.

In the *third* stage, there are many of the phenomena of the second, out of which it is gradually developed,—that which marks its arrival being the partial return of sensation, consciousness, and voluntary power.

The movements now witnessed are not wholly meaningless; the patient makes an attempt to change his position, or to do something, his efforts often, however, being frustrated by some violent spasm; he "looks" at those around him, with a bewildered, suspicious, or sad expression; still there is "expression;" and he may make some attempt to speak; the respiration becomes less unruly, he can clear his throat; the pupils are contracted, but he can see; the conjunctivæ are injected, and there are often petechiæ on the forehead, the temples, behind the ears, and in the eyelids; the pulse is variable; there is a jaded, exhausted state, and the patient seems tired and disposed to sleep.

This third stage may last from a few seconds to ten minutes, when the "after-stage" of stupor sets in. Often there is a confused mental condition, with occasional involuntary movements, lasting for several hours: often the patient recovers rapidly, and goes on with what he was doing before the attack occurred. There is, indeed, almost every degree of severity in the seizure; sometimes all the symptoms I have mentioned being passed through in a far shorter time than it takes to describe them; sometimes each stage being prolonged, and the patient passing gradually into a condition of stupor, from which he awakes, even after many hours, jaded and "beaten," and from which it takes several days for him to recover.

Vomiting often follows the attacks in many individuals; in some it is a constant sequence. Large quantities of pale urine are secreted in the majority of cases; both the urinary water and the amount of urea are increased; and deposits of uric acid and of urates may be discovered. I have, however, failed to find either sugar or albumen in the urine of those epileptics who were not affected by either diabetes or Bright's disease.

The *after-symptoms* of an epileptic paroxysm vary widely in character, severity, and duration. There is usually lassitude and stupor, with headache. It is difficult to rouse the patient, and, if awaked, he is often peevish and irritable, and sometimes suspicious. The sleep is usually tranquil, but occasionally disturbed, as if by dreams. There is commonly stertor, coming and going, guttural in tone, and unlike the noise made by mucus, rattling in the trachea, during the second stage. The muscular condition is that of relaxation, occasionally interrupted, for a moment, by clonic spasm or fibrillar contraction. This stupor may last, if the attack has occurred in the evening, throughout the night, passing insensibly into ordinary sleep. But when the seizure has taken place in the daytime, its average duration has been one hour. It has not appeared to me to bear any constant relation to the severity of the attack as measured by the violence of convulsion. It is often absent in lunatics who are subject to Epilepsy;[1] but Messrs. Leech and Fox show that there is some relation to be observed between the interparoxysmal and the post-paroxysmal mental state. When the post-paroxysmal symptoms are absent or slight, 38·7 per cent. are in the first mental class; whilst of those in whom these symptoms are slight, only 18·1 per cent. are free from interparoxysmal mental change.[2]

M. Voisin states that epileptic fits produce changes in the sphygmographic tracings of the pulse, which last for several hours after the attacks, viz. ascending lines of great height and well-marked dicrotism.[3]

(*d*) *Epilepsia Abortiva, or Epilepsia Gravior without complete loss of Consciousness.*—It is for the sake of practical convenience, rather than because it is strictly speaking pathologically correct, that the

[1] Dr. Bucknill, Asylum Journal, for October, 1855.

[2] Op. cit. p. 229.

[3] Ann. d'Hygiène, xxix. p. 358, quoted in Syd. Soc. Biennial Retrospect, 1867–8, p. 471.

class of cases now to be described are mentioned in this place. Names, as employed in the science of medicine, are useful modes of recognition, and not exhaustive descriptions of the maladies they denote. We must give names to the diseases we describe; we must define what we denote by the names we use; yet, in so doing, we draw, besides the necessary, some artificial lines; and it is occasionally the least of many evils to overstep them.

The attacks to be described are almost excluded by our definition of Epilepsy, yet they so closely resemble that disease in all their own features, that they find a more fitting place in this portion of a System of Medicine than they could find elsewhere. They are closely related pathologically, and we find in their position here an example of the general principle of terminology employed in this work, and no departure from its spirit.

Abortive attacks of Epilepsy have been described by Dr. Prichard (Treatise on Diseases of the Nervous System, p. 91); M. Doussin Dubreuil (De l'Epilepsie en général, p. 16); Schr. van der Kolk (Pathology of the Medulla Oblongata, Syd. Soc. Transl., p. 211); Maisonneuve (Recherches et Observations sur l'Epilepsie, p. 22); Dr. Radcliffe (Epilepsy and other Convulsive Affections, p. 164); Herpin (Du Pronostic et du Traitement de l'Epilepsie, p. 429); Messrs. Leech and Fox (op. cit. 226); and M. Brown-Séquard has detailed the occurrence of similar phenomena in animals (Journal de Physiologie, tome i. p. 474). Several cases of seizures of an abortive character have fallen under my own observation; and what is to be said about them will occupy but little space. There has been sudden tonic spasm of the face, neck, and chest, accompanied by arrest of respiration, and followed by clonic convulsion, having the general form of an ordinary epileptic paroxysm; and yet there has been either no interference with consciousness, or only such slight obscuration as to be at first completely denied by the patient. Such paroxysms may occur, at intervals, for many years; they may take place in those who are subject to ordinary epileptic attacks; or they may exist in connection with other signs of disease in the nervous centres.

The *interparoxysmal* symptoms of Epilepsy may be divided into those pertaining to the nervous system, and those not so related. The "nervous" phenomena exist in regard of mind, sensation, and motility, and they are of varying intensity, prevalence, and kind. The most important are those which belong to the mental history, and they will be considered first.

(*a*) *Mental Condition of Epileptics.*—A prevalent belief is that some form or degree of mental deterioration is necessarily associated with Epilepsy. The result of inquiry upon this point is to show that there is no such "necessary" relation. The general belief is, however, to be accounted for partly by the strong impression which some notable cases of mental failure have made upon the minds of those who witnessed and recorded them,—such strong impression being followed by an undue inference,—and partly by the fact that the words Epilepsy and Epileptic have been made to include every form of disease of brain, spinal cord, or other organs, and also every variety of that multiform derangement which we call "insanity of mind" which might be associated with fits. It is desirable, again, to assert that this article refers only to such cases as constitute Epilepsy proper; and that the statistics upon which my results are based, can only with a double injustice be compared with those derivable from lunatic asylums. A patient may be epileptic and a lunatic: he may be epileptic and asthmatic; but there are some epileptics whose minds are as healthy as their lungs: and, so far as the natural history of Epilepsy generally is concerned, it is a mistake to derive it from complicated cases.

The mode in which I have endeavored to answer the question,—what is the actual mental condition of epileptics during the intervals of their attacks?—has been the following. I have divided epileptics into four classes: in the first there are placed those in whom, neither by the patients themselves, by their friends, nor by myself, could there be detected any deviation from mental health; such individuals had "nothing the matter with them," but exhibited for their station in life and educational advantages the full average amount of intellectual vigor and cultivation. The second class consists of those who presented that slight defect of memory which is limited to the occurrence of recent and trifling events, the memory for those long since past being intact; and in those who formed this group, such impairment of memory was the only departure from mental health. In the third class are those cases which present, in addition to the loss of memory described, some diminution of the power of apprehension. Such patients are dull in acquiring new ideas, and often receive incorrect or imperfect and confused notions of what is brought before them. The fourth class includes those who, in addition to the failures exhibited by the preceding groups, are habitually confused, and unable to follow out any train of thought; people who seem to think little, but to be in a vacant, wandering state of

mind, often idle, stupid, and indifferent, and sometimes almost or completely demented.

Having determined upon this principle of arrangement, it is comparatively easy to answer many questions of interest with regard to epileptics, and to state the answers to such questions in numerical terms. This I have done in another work;[1] and all that it is thought desirable to do now is to give some of these results,—and with them others based upon a wider range of facts,—without burdening the reader with a number of statistical details.

In rather more than one-third of all the cases which I have examined there has been perfect (*i. e.* average) mental integrity; in a little less than two-thirds, there has been some intellectual deterioration, but this has existed to a high degree in only a very small proportion. Women have been found to suffer more frequently and more severely than men; and the commonest form of failure is that of defective memory; this faculty being diminished, especially in regard to recent and trifling events.

It is of much interest to know the conditions which determine mental failure in the epileptic, and thus to avoid certain errors which are prevalent upon the subject. The results of inquiry upon this point may be stated in the following propositions:—

Hereditary taint is without influence.

The age at which Epilepsy commences exerts a certain amount of influence, and to this effect—that the disease when appearing late in life is more commonly associated with mental failure than it is under the opposite condition; and that the chances of mental failure are less when the attacks commence before the arrival at puberty than they are when Epilepsy is developed after that epoch. This statement is supported by the further statistics of Messrs. Leech and Fox.[2] Late rather than early Epilepsy is a predisponent to intellectual failure, and this whether we divide the cases at the tenth, sixteenth, or twentieth years, and whether we consider the two sexes together, or each sex separately.

The duration of Epilepsy is, *per se*, without influence upon the mental condition of the epileptic.

The amount of mental deterioration is not in direct proportion—but in inverse ratio—to that of muscular disturbance, as shown by the presence of tremor, or spasm, either clonic or tonic.

The state of the "general health" does not account for that of the mind; the former may be good, and the latter bad, and *vice versâ;* and, contrary to what would be expected, such relation is more usual than the coexistence of marked failure or integrity in both directions.

The number of attacks does not determine either the degree or the existence of intellectual change.

Frequency of recurrence of the seizures is, however, associated with mental change; but in such manner as to show that it is not the sole condition of such result, and that it is not even a necessary condition.

The severity of the convulsive paroxysm is without apparent influence, when such severity is judged of by the duration of subsequent coma. The form of the attack appears, however, to exert a considerable influence. Neither seizures of "le haut mal," nor those of "le petit mal," necessarily induce the change of which we are speaking; but the mental deterioration of epileptics is much more clearly associated with the minor than with the severer seizures.

The nature of the exciting cause, viz., its existence in the psychical or the material elements of life, appears to be without influence in the determination of mental change.

(*b*) *Sensorial Condition of Epileptics.*—Headache and vertigo are the two forms of disturbance the most frequently complained of by epileptics. They exist, however, to a high degree in only a small number of the cases; and, when they do exist, have no special character which renders them of value in either diagnosis or prognosis. Headache is more frequent in females than males. The vertigo of epileptics is commonly of such kind that the patient rarely imagines that surrounding objects are in motion, but rather that he is, himself, moving or turning round; he feels as if he were doing so, and is unsteady in standing, or in his attempts to walk.

The pupils are more commonly beyond than below the average size; the special senses exhibit neither constant, prevalent, nor characteristic change.

(*c*) *Condition of the Motorial System in Epileptics.*—Some patients exhibit a tremulous state of the muscles; some, either with or without tremor, are affected by clonic spasm; others present tonic spasm, or cramp; whereas many are quite free from either of these forms of altered motility. In the majority of cases there is some kind of disturbance; but in the greater number of this majority the amount of such disturbance is slight.

The patient often says that he is "nervous," meaning by this that his hand is unsteady, or that the body is tremulous, or that he feels as if they were so.

It is often denied that any jerking of the muscles ever occurs; but the physician may frequently discover that such

[1] Auct. op. cit. [2] Op. cit. p. 212.

denial is incorrect. The amount of clonic spasm may be, therefore, very slight: it may, on the other hand, be very considerable, assuming one or both of two general forms. There may be, more or less constant and considerable, choreiform movement; and this may be observed not only when the patient is awake, but when he is asleep, and often with exaggerated force in the latter condition. There may be violent spasmodic shakings of the limbs or of the trunk; occurring at irregular intervals, but exhibiting an especial frequency of occurrence just as the patient falls asleep. Such jactitations have proved excessively annoying in several cases, and have been so troublesome as to entail much ulterior distress from the loss of sleep that they have occasioned. Sometimes the jerk of muscles is so sudden and so violent that the patient is thrown out of bed; or, if standing, is thrown down.

Cramp, or tonic contraction, is comparatively rare, and has apparently only an accidental relation to the disease. Messrs. Leech and Fox found it more frequent in occurrence than I have done (op. cit. p. 216).

(*d*) *Condition of the General Health.*—There are no changes in the "general health" of epileptics to be observed with such sufficient frequency or specialty that they deserve to be reckoned among the characteristic features of the disease. Epilepsy may exist in every condition of the general health; but among those who have been primarily poor, or who have become so owing to their disease, a low state of vitality is encountered. A similarly depraved condition may be found where the circumstances have been different; but such state is by no means necessarily present. Epileptics may be found in robust as well as in feeble health; but it is important to know the relative frequency of the one and of the other condition.

Patients have been examined by myself in regard to their nutrition, temperature, and strength, and the general results of such inquiry are those stated above. But, further, cases have been divided into four groups, viz.: into, 1st, those exhibiting, in every particular, good health; the limbs being well nourished, of normal temperature, and of natural strength;—individuals capable of enduring both exposure and fatigue, as well as any others of their age, sex, and social condition; 2d, those in whom some failure in one of the above particulars was noted; 3d, those in whom a double deterioration was observed; and 4th, those in whom there was deficiency in all three particulars. The result of such inquiry has been to show that more than one-half of the cases belonged to the first group; less than one-third to the second; less than one-tenth to the third; and little more than one-hundredth to the fourth. The most frequent change has been defective temperature; the least frequent, impaired nutrition. The pulse has exhibited no constant feature, either in frequency, force, or fulness.

There is, according to my experience, an entire absence of any specific change in epileptics, so far as regards their functions of digestion, respiration, circulation, and secretion.

If, as the result of this mode of inquiry, we regard epileptics as a whole, and put together all the results that have been obtained, we come to this important conclusion, that in a certain number (12 per cent.) there is nothing, absolutely nothing, abnormal to be discovered during the intervals of attack; that in nearly two-thirds of the cases some failure may be observed either in mind, motility, or general health; and that in less than one-third there is marked alteration.

It is then obvious that Epilepsy is a disease characterized only by its paroxysmal symptoms, and having, in the present state of science, no special features by which it may be recognized during the intervals of attack.

4. Relations between the Symptoms of Epilepsy.—(*a*) *Forms of Attack.*—The severer seizure, Epilepsia gravior, is nearly twice as common as the milder, Epilepsia mitior; and the former is much more frequently found by itself than is the latter. Hereditary taint seems to exert an influence in predisposing to the severer form of attack. The milder attacks, however, do not appear to take the place of the more severe, but to be found with especial frequency in those cases which exhibit a rapid recurrence of the latter, *i. e.* of Epilepsia gravior. The form of attack does not appear to be determined solely, or even notably, by the age at which the disease commences; but when Epilepsy is developed early in life, there is an increased proclivity to the attack in its milder form. Duration of the malady does not determine its form of seizure.

(*b*) *Frequency of Attacks.*—In about one-seventh of the cases that I have examined the seizures have exhibited a mode of recurrence which has been termed "serial;" that is to say, that the patients suffer from two, three, or more attacks in one day, and then pass through a period of freedom lasting from one to several weeks; and this mode of recurrence is more frequent in the female than in the male sex. The series, groups, or, as they are often termed, "bouts" of the fits, usually occupy one day only, and they are often limited to a period of twelve hours.

It is rare, very rare, to find an accurate periodicity in Epilepsy; but it is exceedingly common to observe that the recurrence of attacks has some kind of relation to time, as marked by its natural division into days, and periods of seven days, or multiples of seven days. Thus a large number of epileptics have their seizures every day, every two weeks, three weeks, and four weeks; while only a much smaller number suffer at such irregular intervals as cannot be thus expressed. An almost identical number of patients state that they have attacks at each of the periods mentioned; not meaning by that to say that there was always either perfect periodicity, or recurrence "to the day," but that, as a rule, every fortnight, three weeks, month, or day, there had been an attack.

There are four times as many epileptics who suffer from their seizures more frequently than once a month than there are of those whose attacks recur at longer intervals. The return of attacks at monthly periods is rather more common in the male sex than in the female; and it is very rare to find the seizures limited to the time of the menstrual discharge. It is frequently noticed that they are more common during menstruation; but, on the other hand, many women whose attacks recur at monthly intervals, exhibit no marked proclivity to their recurrence while the catamenia are present. A high rate of frequency is more common among women than among men.

The number of attacks in a given time ranges between very wide limits—from two to two thousand in a year; but half the cases are found to have a rate of recurrence ranging from one attack in fourteen to one in thirty days.

Great frequency of attack is not constantly associated with signs of motor disturbance, such as tremor, clonic spasm, and the like.

Again, a high rate of frequency is not determined by an enfeebled state of the bodily health; but, on the contrary, is observed in those whose general physical condition is up to the standard of health, whereas a low rate of frequency is found in those whose organic powers have undergone marked deterioration.

An early commencement of Epilepsy is commonly, but not necessarily, associated with a high rate of frequency in the attacks. As the disease continues it exhibits a tendency to increase in the frequency of its paroxysms; but duration is not the sole condition determining this result.

(c) *Morbid Motorial Phenomena* are not found exclusively in those who exhibit an impaired state of the general health, but the one kind of derangement—marked by tremor, or clonic spasm—is commonly found in combination with the other, viz. diminution of temperature, or nutrition, or strength.

The prolongation of Epilepsy is not necessarily associated with impairment of the physical condition; but a high degree of the latter is often found in conjunction with a protracted duration of the disease.

(d) *Consequences of Epilepsy.*—If Epilepsy were found to entail, of necessity, any definite changes in the health of its subject, in regard of either mind, motility, or general condition, we should expect to find that such changes bore a definite and direct relation to the time during which the disease had existed. On this point, however, the result of a careful examination leads to the conclusion that duration is, *per se*, without effect, and that the demonstrable "consequences" are *nil*.[1]

5. Complications of Epilepsy.—These may exist in any organ of the body, but they have no such definite character, except when they are presented by the nervous system, as to require any special comment in this place. The most important is—

Epileptic Mania.—This complication occurs in about one-tenth of the cases, if we reckon all those degrees of such disturbance as may warrant the application of such name. Having occurred once in a particular individual, it is likely to appear again, and this is especially the case when several attacks have followed in rapid succession.

The delirium is commonly but not universally furious and dangerous; it is sometimes ecstatic in form, sometimes dull and melancholic. It may appear in the form of preternatural gayety before the attacks, or in the intervals of their recurrence; it may break out as violent excitement just as the patient is emerging from the second stage of the paroxysm. Sometimes the mania has preceded the convulsions, but this order of events is, comparatively speaking, rare. Epileptics occasionally have some premonition of their maniacal state—an indescribable feeling which leads them to place themselves under restraint before the occurrence of the outbreak. More commonly, however, there is no such warning, and the physician fails to discover any special reason for the attack.

Meningitis in an acute, sthenic form, may follow epileptic paroxysms; but when it has done so, it has, in the majority of cases, been determined by some accidental injury inflicted by a blow or fall, which the patient has experienced in one of his attacks.

Apoplexy is so rare a sequence of Epilepsy that it is mentioned simply for the purpose of stating this fact, because it—

[1] Auct. op. cit. p. 199 *et seq.*

apoplexy—is one of the dangers often quite unnecessarily dreaded by both epileptics and their friends.

Idiocy may be complicated by Epilepsy; but when the two conditions are found together, or are stated to coexist, the truth appears to be this, that the idiotcy has been congenital, and that the idiot has been "subject to fits." Abundant facts and reasons have been already furnished for the purpose of proving that the mode of regarding Epilepsy proper which would show that idiotcy is one of its frequent complications is fallacious, inasmuch as it widens the meaning of the word Epilepsy beyond what is pathologically correct, or practically desirable.

Convulsions, such as those attending upon dentition or parturition, exhibit no special frequency of occurrence in epileptics.

Paralysis is so rare an event that it may be regarded as having—like meningitis—an accidental, rather than essential, relationship to the disease in question.

Cyanosis is often accompanied by fits, and these have often assumed an epileptic character; but cyanosis is a rare malady, and its mere mention as a complication is all that is necessary here.

PATHOLOGY. — Anatomical investigation has hitherto failed to give any explanation of Epilepsy; every kind of lesion has been discovered in every organ of the body; and, on the other hand, every organ and part of organ has been found in perfect health. The observations of Wenzel,[1] those of MM. Bouchet and Cazauvielh,[2] and the later researches of Dr. Schröder van der Kolk,[3] have shown the existence of disease in the pituitary body, in the white substance of the brain, and in the medulla oblongata; but the changes that each of these authors has described have been found to be inconstant, and some of them quite exceptional. We must, therefore, admit the disease to be what is termed "functional," using that word in the sense strictly defined in the first part of this volume.[4] It is believed that "nutrition" is changed, but that its alterations are too fine for detection by our present modes of examination.

Bearing in mind all the facts of Epilepsy, and proceeding to their interpretation by the aid of physiology, we arrive at the following conclusions:—[5]

1st. That the seat of primary derangement is the medulla oblongata, upper portion of the spinal cord, and vaso-motor system of nerves.

2d. That the derangement consists in an increased and perverted readiness of action in these organs; the result of such action being the induction of spasm in the contractile fibres of the vessels supplying the brain, and in those of the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs generally.

By contraction of the vessels, the brain is deprived of blood, and consciousness is arrested; the face is, or may be, deprived of blood, and there is pallor; by contraction of the muscles which have been mentioned, there is arrest of respiration, the chest walls are fixed, and the other phenomena of the first stage of the attack are brought about.

3d. That the arrest of breathing leads to the special convulsions of asphyxia, and that the amount of these is in direct proportion to the perfection and continuance of the asphyxia.

4th. That the subsequent phenomena are those of poisoned blood; *i. e.*, of blood poisoned by the retention of carbonic acid, and altered by the absence of a due amount of oxygen.

5th. That the primary nutrition-change which is the starting-point of Epilepsy may exist alone, and Epilepsy be an idiopathic disease, *i. e.*, a *morbus per se*.

6th. That this change may be transmitted hereditarily.

7th. That it may be induced by conditions acting upon the nervous centres directly, such as mechanical injuries, overwork, insolation, emotional disturbances, excessive venery, &c.

8th. That the nutrition-change of Epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ, rheumatism, gout, syphilis, scrofula, and the like; and further, that it may often be associated with change in the cortical substance of the hemispheres of the brain.[1]

9th. That it may be induced by some unknown circumstances determining a relative excess of change in the medulla, during the general excess and perversion of organic change occurring at the periods of puberty, of pregnancy, and of dentition.

10th. That it may be due to diseased action extending from contiguous portions of the nervous centres or their appendages.[3]

---

[1] Observations sur le Cervelet, &c., traduit par M. Breton.

[2] Archives Générales de Médecine.

[3] On the Minute Structure and Functions of the Spinal Cord, Syd. Soc. Transl.

[4] See p. 18 *et seq.*

[5] The reader is referred to the Author's Treatise on Epilepsy, chapter Pathology, for a full exposition of the views here stated propositionally, and also for complete reference to the different authorities quoted in support of each proposition.

[1] See Dr. Wilks's paper in Guy's Hospital Reports, 1866.

[2] Referring to the article preceding this, upon Convulsions (especially the part dealing

11th. That the so-called epileptic aura is a condition of sensation or of motion dependent upon some change in the central nervous system; and is, like the paroxysm, a peripheral expression of the disease, and not its cause.

Diagnosis.—Bearing in mind all the features of this disease as they have been described and limited in the foregoing pages, it will be comparatively easy to distinguish Epilepsy from every other malady.

The disease may be *simulated*, and when such is the case the fraud may be detected by the "over-acting" of the pretender, and longer duration of the paroxysm; by the choice of locality for the purpose of display; by the absence of those changes in color which have been described; and last, but most certainly, by the absence of dilatation of the pupil. After the attack the sphygmograph may be employed in the manner adopted by M. Voisin.

Syncopal Attacks often resemble those of "le petit mal;" and the latter may be mistaken for the former. There is, perhaps, a much closer analogy between them than is sometimes supposed. For practical purposes of prognosis and of treatment, the distinction will turn upon these points of difference; in Epilepsy loss of consciousness is sudden, absolute, and often without any sense of "faintness;" recovery is rapid, and there is no recollection of the attack.

Hysteria, when convulsive in form, differs in the presence of some volition, some sensation, some power of directing movements. The attack is "got up," or passed into, gradually; and is preceded by sobbing, crying, laughing, and gesticulations: it continues sometimes for an indefinite period, and passes off through a stage of hysterical excitement.[1] The history of the case before the attack, and after its occurrence, is that of hysteria; whereas in Epilepsy there is or may be nothing abnormal to be discovered. In the attack there is not the hideous distortion of the features, neither is there the meaningless eye, nor the dilated pupil, nor the bitten tongue; respiration may be and generally is disorderly, but there is no marked asphyxia. After the attacks the patient is exhausted, but does not pass into stupor; hysterical mania or paralysis may follow, but they have their own special features.

Convulsions—such as those of teething, of worms, and the like—differ as widely from Epilepsy as attacks of bronchial catarrh do from genuine spasmodic asthma. The presence of dyspnœa, cough, and expectoration does not constitute a case of asthma; the loss of consciousness and convulsion does not constitute a case of Epilepsy. The real nature of the disease must be determined by those facts of its history which lie behind these symptoms, and determine its position in nosology. Convulsions may occur many times, and may sometimes pass into the disease we are describing; but they do not necessarily do this, and mere periodicity of recurrence is not the only mark of distinction between them.

Convulsions are most frequently found during infancy, and especially so while the child is cutting its first set of teeth. It is rare for Epilepsy to date from so early a period. Usually febrile symptoms precede the attack, or there is some definite source of irritation in the mucous membrane or secreting organs—*e. g.* dentition, worms, indigestion, scybalæ, calculi. The first occurrence of the convulsion and its subsequent repetition may be traced to one or more of the irritations enumerated. The attacks cease on the removal of their "exciting cause:" and they differ from Epilepsy in the following features: Their invasion is less sudden, and the paroxysm is of shorter duration; there is not absolute loss of consciousness at the onset of attack; if perception, volition, and sensibility are entirely removed, such removal is during the clonic spasm, and not at the beginning; there is little or no subsequent stupor, and no paralysis.

The diagnosis of *diathetic* convulsions is based upon a recognition of the diathesis. At the onset of some of the exanthemata convulsions may occur, and assume an epileptic form, but they are to be distinguished by the fact of their appearance in early life, the patient being usually under six years of age; by the presence of febrile disturbance, and of some exanthem, or some acute inflammatory change such as pneumonia or bronchitis.

In "Bright's disease" of the kidney convulsions of epileptoid type may be the first symptoms which bring the patient under the notice of the physician. There will, however, be but little difficulty in establishing the diagnosis. There are marked and peculiar pallor, puffiness of eyelids or of ankles, and albuminuria. The attacks are followed, or have been

with Unilateral Convulsions) for an admirable, though incidental, discussion of the above subject, it may be added that its author, Hughlings Jackson, is regarded by recent writers as one of the pioneers in the study of the localization of brain-lesions and disorders, to which so much has been contributed since 1870, by Hitzig, Ferrier, Charcot, Vulpian, Duret, and others. Later in this volume, a further consideration will be given to this subject, in connection with the article upon Cerebral Hemorrhage and Apoplexy.—H.]

[1] See article on Hysteria; [also, Hystero-Epilepsy.—H.]

preceded, by drowsiness, listlessness, and a tendency to delirium; there are headache, vertigo, clonic spasm, alternating with marked rigidity of limb, great irritability of the muscles on percussion, and often a highly characteristic state of the mental functions. The latter has these features: The patient lies in apparently profound coma, with some limbs relaxed, and others rigid or in clonic contraction, breathing heavily with a stertorous sound, which may be found to exist in the mouth, and not in the throat; but, from this state of apparently profound stupor, he may be readily aroused to do that which he is told to do, or to answer questions; and immediately afterwards he falls again into the state of stupor. His condition resembles somewhat that of a person poisoned with opium.

It is sufficient to mention such diseases as *chronic alcoholism*, *lead-poisoning*, *syphilis*, and *rickets*, in order to indicate the means by which, when they are attended by convulsions, the diagnosis may be established.

Organic Diseases of the Nervous Centres may be distinguished from Epilepsy by the fact of their presenting symptoms over and above those proper to the latter. When conspicuous and persistent changes in the functions of the nervous system occur during the interparoxysmal period, we may infer the existence of structural disease. Again, there is more marked impairment of the general health; and the signs of disordered nerve-function have a more rapid development than have those which may occasionally be observed in Epilepsy. *Tumor* of the brain exhibits its most characteristic feature in persistent, or paroxysmally exaggerated pain, limited to a particular locality, and accompanied by local paralyses. *Chronic softening* may be diagnosticated by the gradual failure of mind, sensibility, and muscular power. *Chronic meningitis* may have a protracted history, but it is one of highly marked interparoxysmal change. There is irritability of temper, and, occasionally, delirium with loss of memory and impaired intellectual power: there is spasm alternating with local paralysis; and there are alterations of the special senses, with headache and general malaise.

The convulsions which occur in chronic cerebral diseases are not precisely like those of Epilepsy; there is less suddenness in their invasion, there is not the complete loss of consciousness, the convulsive movements do not pass through the several stages that have been described, but are irregular in their manner of development, protracted in their duration, and often limited to one side, or to one extremity. There are not the asphyxial phenomena of Epilepsy, neither is there the subsequent stupor.

Again, the ages at which intra-cranial diseases are developed differ from the prevailing age at which Epilepsy makes its appearance; neither aneurism nor carcinoma appears, as a rule, so early in life as does the disease under consideration; cerebral tubercle, when occurring in childhood, has a history widely different from that of Epilepsy; and, lastly, each of these is attended by its own special dyscrasia, which may afford all that is needed to complete a diagnosis.

Prognosis. — When the disease has been established for some time, and is recognized to be an idiopathic affection, the prognosis is unfortunately very unfavorable as regards perfect and permanent cure. When it is recent, much hope may be entertained. Cases of eccentric convulsions and of chronic meningitis, either syphilitic or simple, may be cured, and such are often spoken of as epileptic; but I do not include them in the present article: the remarks here made apply exclusively to Epilepsy proper.

The *general prognosis* is framed upon several different considerations. Hereditary taint is of unfavorable omen; whereas an early commencement of the disease is the reverse. The duration of the malady is of the highest importance; the longer that it has lasted the greater is the difficulty and improbability of cure. Those cases in which the intervals between the attacks are much prolonged are less amenable to treatment than are those which exhibit a more rapid recurrence. Mental failure is of evil augury, but not to so high a degree as has been supposed. Some of the most obstinate cases are those in which the general health is good; some of the most tractable are those in which there is a disturbance which may be corrected.

Next in importance to that of the prognosis of the disease as a whole, is the forecasting of *the mental state*, supposing that the disease itself cannot be cured. What conditions are there which would render mental failure probable? The section on "natural history" supplies the answer to this query, but its results may be recapitulated here. Hereditary taint is without influence: the female sex is of unfavorable omen; late commencement of the disease is a predisponent to intellectual failure; mere duration is without influence; an impaired state of the general health is of good rather than evil import; mere number of attacks is of no moment; rapid recurrence of seizures is indicative of danger; and attacks of "le petit mal" are more injurious than are the severer paroxysms.

The *danger to life* is somewhat remote, and need scarcely be entertained. It is excessively rare for an epileptic to be

killed by, or die in one of his attacks. Van der Kolk[1] has shown that the danger to life is greater in those cases in which the tongue is not bitten; but I have no observations to prove either the correctness or the incorrectness of this opinion, as I have never yet known a case in which the attack proved fatal.

Treatment.—There are two distinct elements to be regarded in the therapeutics of Epilepsy: the one is the diminution or removal of the condition which is the essential element in the disease; and the other is the mitigation of the paroxysmal symptoms when their removal cannot be effected. We have to direct the treatment of the disease and that of the attack.

It has been already stated that many epileptics, during the intervals of their paroxysms, present no abnormal condition; yet it is to be inferred that there must exist in them some departure from health, and the conclusion to which we have arrived is, that this departure consists in an undue readiness of action in certain portions of the nervous centres. Our object, therefore, is to control this over-readiness of action. For this purpose *sedatives* have been employed, and with success. It would be useless to attempt any estimate of the relative value of many of these agents, for there are no data sufficient for the purpose. Opium or morphia, conium, hyoscyamus, stramonium, belladonna, cannabis Indica, atropine, valerianate of atropine, selinum palustre, cotyledon umbilicus, chloroform, and other medicines have been employed with good effect in some cases, and without any appreciable effect in others, and hitherto no principle has been evolved from either their failure or success. When the attacks have been of very frequent recurrence, I have found preparations of the solanaceæ useful in diminishing the number of seizures, but I have never known them to effect a cure. Opium, or some preparation of morphia, has been of service when the patient was restless at night, and was obviously suffering from the effects of loss of rest. Chloroform has delayed attacks while the patient was actually under its influence, but has failed to prevent their subsequent recurrence. Dr. Murray has, however, been fortunate in the treatment of some cases by means of chloroform, and his observations are such as to warrant a further employment of this agent.[2] Indian hemp has relieved headache and restlessness, but has not cured or notably relieved Epilepsy.

The salts of zinc, and especially the oxide of zinc, have appeared to be of service in many cases; their action being obviously sedative. I have seen no good results from the sulphate of zinc given in heroic doses, and the good effects that have come under my own observation have been from oxide of zinc in doses of three or five grains given three times daily. The salts of copper and of silver have proved utterly useless in my own experience.

Bromide of potassium, or some other salt containing bromine, is the one medicine which has, so far as I know, proved of real service in the treatment of Epilepsy. Undoubtedly it is "sedative" in its action; it lessens spasmodic movements, especially those of paroxysmal character, and sometimes insures sleep when vegetable sedatives, and among them opium, have failed. Bromide of potassium in small doses has appeared to be of little or no service, but in large doses it rarely fails to give some relief. Sir Charles Locock has the merit of introducing this drug to the notice of the profession in this country,[1] and the testimony of all those who have had much experience in the matter concurs to a remarkable degree as to its utility. Given in doses ranging from ten to thirty grains, three times daily, it has had these effects: In some cases it has completely cured the patient, and the cure has been permanent for years, and is so now. In others it has arrested the attacks so that none have occurred for periods varying from a few months to two or three years; but, on the omission of the medicine, the seizures have returned. In such cases the attacks have again ceased on the re-administration of the medicine. In a third series of cases it has diminished the frequency and severity of the seizures, but has not removed them altogether; the patients while taking the bromide have had one-half or one-third of the number to which they were habituated. Such patients have gone back to the old frequency of recurrence when the drug has been omitted, and have again improved when it has been re-administered. In a fourth, but very much smaller number, the influence of the drug has been good for a time, and has then appeared to cease; and in a fifth, and yet smaller proportion, it has been apparently without any appreciable effect. Still further, there are a very few cases in which the number of seizures has been increased by bromides. Dr. Duckworth Williams has shown that it exerts much influence over those cases in which the attacks take place during the day, but that it is of little use in those patients whose seizures occur during the night.[2] I have found that this is true to a certain

[1] Op. cit. p. 252.
[2] Medical Times and Gazette, April 8, 1865.

[1] Lancet, May 20, 1857.
[2] Op. cit.

extent, but not to the degree described by Dr. Williams; for in several instances KBr has been very useful when the fits were limited to the hours of sleep. It is possible that Dr. Williams's cases may have been, from the fact of their complication with insanity, peculiar in this respect. Bromides appear less useful in growing girls and youths than in those who have reached adult age.

It often happens that the administration of five grains will diminish the frequency of attacks, or prevent their occurrence, for a period of weeks or months; but that then, the medicine being still taken, the seizures revert to their previous rate of frequency. An increase of the dose is followed by a similar succession of events; a further increase by a second succession of temporary improvement and subsequent deterioration; and so on, until a larger dose, of from thirty to forty grains, is administered three times daily, when the attacks cease altogether.

It is not the mere administration of the drug, but its presence in certain quantity, that is necessary for a cure; but the dose which shall prove curative is not determined by either one of the following conditions: sex, age, duration of disease, frequency of attack, severity of attack, or form of attack.

The number of cases in which it proves of no service, at any dose, is very small; and some of the cases which resist its action do not differ in any other obvious respect from those in which the bromide is highly efficacious.

In an earlier part of this article I have given reasons for thinking that the mode in which KBr proves useful in Epilepsy is not by its diminishing either the sexual propensity, or power. It is positively curative of Epilepsy when given in doses which exert no influence whatever upon the generative functions.

In some individuals the administration of KBr produces discomforts to which the term bromism has been applied. The most common of these is an acne-like eruption on the face, shoulders, and body generally; the most important is a state of stupidity and partial aphysia. Drowsiness, dulness of apprehension, muscular weakness, and general lethargy are often met with; and these symptoms may exist with varying degrees of severity, and be produced very easily in certain individuals. On the other hand, KBr may be given for many consecutive years, and in large doses, without producing any one of the discomforts that have been mentioned. It is easy to remove these symptoms by a discontinuance of the drug, and the temporary administration of a bitter infusion with a mineral acid; and it is equally easy, and much more desirable, to prevent their occurrence, by omitting the medicine for one or two or even three days in the week. All the good effects of Br are thus secured, and its evils are avoided.

Dr. Williams[1] states some facts which would appear to prove that KBr does diminish the force of the heart's action; but in my own experience this has not occurred to any such degree, or with such frequency, as to make me attach any importance to its occurrence. Bromide of potassium has arrested Epilepsy without producing any diminution of cardiac action; and in all cases where there has been the least suspicion of such effect, the addition of chloric ether, or of tincture of cinchona, or indeed of any diffusible stimulant, has at once removed the threatened inconvenience. The bromide of sodium was suggested to me, some time ago, by my friend Dr. Ransom, of Nottingham, and I have now employed it in a large number of cases with perfectly satisfactory results. The dose is the same as that of KBr, but the NaBr has this advantage, that it may be taken alone, as common salt, with food, and when mixed with an equal proportion of NaCl would be quite unnoticed in the salt-cellar.

Counter-irritation, and derivants, such as setons, issues, and the like, have appeared to me to be of signally little service in genuine Epilepsy, so that I have been led to the belief that those cases in which they have been said to be of great utility have been examples either of some other malady, or of some complication of the disease.

As to diet and regimen, these things seem to me important: first that the patient should eat digestible meals, with great regularity; and second that exercise, in the open air, should be taken as much as possible, short of fatigue. Many epileptics have been relieved from nocturnal attacks by being made to sleep with the head and shoulders well raised, not by pillows, but by a simple contrivance which is placed under the upper half of the bed or mattress on which they lie. Baths used for the purpose of cleanliness are useful in Epilepsy as in many other diseases; but I have seen more harm than good follow the employment of douche, shower, and sitz baths, when these have been administered in any manner or to any degree which exceeds that of producing comfort to the individual. Warmth to the extremities, especially at night, is of great value; the patient should never go to bed with cold feet, nor run the risk of their becoming cold during the night. Fires, hot water, hot-water baths, and woollen socks, may prevent much mischief. Sexual intercourse appears to me also to be one of those matters upon

[1] Op. cit.

which the dictates of common sense are sufficient without any special direction from the physician.

The mental state of the epileptic may be much injured by action upon one very common form of advice, viz. that the patient should "do nothing." It is desirable to avoid over-exertion, worry, and undue excitement; but moderate mental exercise is of great utility; and some definite employment, carried to a point short of fatigue, should be enjoined as part of the treatment of those cases which are not complicated with cerebral excitement.

It is impossible to pay too great an amount of attention to the "general health" of epileptics, but there is nothing *special* in regard to this matter. Cod-liver oil, quinine, iron, alteratives, and aperients must be given in circumstances which would render their exhibition desirable in other forms of disease.

Allusion has already been made to the operation of clitoridectomy, and reasons have been given for suspending judgment on the matter. Doubtless success has followed such treatment in some cases, but the results are, at present, too uncertain for the formation of a definite opinion, first as to the stability of the cure; secondly as to the class of case in which the operation is justifiable; and thirdly as to the therapeutic *modus operandi* of clitoridectomy when it has appeared to be useful. It is not only possible, but highly probable, that an operation of severity equal to that of clitoridectomy might prove servicable in some cases of Epilepsy if it were performed on the back of the neck, the mouth, or the toes. A strong impression upon the mind, or a violent change in the body, such as the opening of an issue, the performance of tracheotomy, or the occurrence of an accidental burn, has often arrested the attacks. It is probable that clitoridectomy and circumcision may, in some cases, act beneficially in a similar manner; but it is obvious that, if they do, the form and locality of operation might be changed with advantage. So far as my own observation extends, the cases are almost infinitely rare in which such an operation would appear to me to be allowable.

The application of ice to the spine has, of late, been advocated with great ability by Dr. John Chapman; and there appear to be many theoretical considerations warranting the employment of this mode of treatment. It is not my purpose to detail the theory upon which Dr. Chapman has acted, as it is fully explained in his own writings. I regret to say that such application has utterly failed to do any good in a very large number of epileptics for whom I have prescribed it. Ice has been applied in the manner recommended by Dr. Chapman, and has been persevered in for many months, without producing the smallest effect upon the frequency or severity of the paroxysms. In one case, at University College Hospital, it was applied both night and morning, without influencing the disease, and on more than one occasion the fits took place while the ice-bag was on the spine. In one instance it was followed by relief, but in this case the patient was taking at the same time bromide of potassium. For the purpose of testing its utility I have employed it in a number of cases without giving any medicine whatever, and the result has been absolutely negative; it has done no harm, but it has done no good. It has appeared in several individuals to be of service in the first instance, but soon, in spite of its persevering application, the attacks have recurred with their usual frequency and severity. Patients have not complained of its application, but I have failed to find that it exerted any influence upon their temperature, when this was tested by their own sensations, or by the thermometer applied to either the axillæ or the extremities.

The *treatment of the attack* is mainly of value when directed towards its prevention; and there are several means by which some good may be accomplished. When an "aura" is present, the paroxysms may sometimes be arrested by cauterizing the surface from which the aura comes, or by applying pressure between the starting-point of the aura and the trunk. Sometimes the attack begins by a special form of contraction in particular muscles, and its progress may be arrested by forcible extension of these muscles. Chloroform, or ammonia, if inhaled, will often prevent the seizures just at the moment of their onset; and in like manner a draught of wine, of salvolatile and water, or of some other diffusible stimulant, will put off the attack. When patients have warning sensations, of sufficient duration for them to do anything, it is desirable that they should carry with them some little draught of this kind, which they may take at the moment of threatening. By such means a large number of fits may be averted.

[Nitrite of amyl has been found to exercise great control in this way in some cases. It is a powerful remedy; to be employed with much circumspection. A few drops only should be inhaled at a time; the dose to be, after frequent repetition, very cautiously increased.—H.]

Tracheotomy has been shown to be of no such real service in Epilepsy as to warrant its recommendation.

When the attack is once established, there is little that can be done beyond that of preventing the patient from injuring himself. Compression of the carotids may arrest or shorten the attack, but it

does not cure the disease. It is possible that the pressure upon the nerve trunks is an important element in this mode of treatment. A piece of India-rubber may save the tongue from being bitten; a loose cravat may diminish the petechial discoloration of the face; and a strong arm may hinder the bruising of the extremities.

When the paroxysm is over the patient should be allowed to sleep, and should be placed with the head and shoulders raised.

In some epileptics the mental symptoms are the most highly marked features of the interparoxysmal period, and to these attention must be mainly directed. In others the general health is greatly at fault, and in them the treatment must be turned towards its improvement. In a third class there is excessive motility of involuntary kind, and in such cases the vegetable sedatives are of marked utility; but in all, the medicine which has proved most useful, in my own experience, is the bromide of potassium.

MUSCULAR ANÆSTHESIA.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—A loss of the feeling of muscular action, attended by irregularity, sluggishness, and diminished force of voluntary movement; but unattended by any necessary loss of cutaneous sensibility or by distinct paralysis.

Nomenclature.—The property which is diminished or lost, in the affection above defined, has been described under different names, of which the following are the more important:—"the muscular sense;" "le sentiment d'activité musculaire;" "le sens d'activité musculaire;" "la conscience musculaire:" "le sens musculaire;" "le sens de la force;" "le sentiment du mouvement;" "der Muskelsinn."

Symptoms.—The essential features of this condition are the following:—awkwardness and clumsiness in performing certain voluntary movements, sometimes of the hand and arm, sometimes of the leg, sometimes of the face. The patient tries to do what he wishes, or is told to do, and succeeds in the attempt by looking carefully at his limb, and helping it with one of the others which is unaffected; but if not paying great attention, or making any great effort, he fails to effect the movement, lets objects fall out of his hands, knocks his legs one against the other, or in some other manner exhibits clumsiness and want of co-ordinating power. If placed in absolute darkness, or if the eyes are bandaged, he may be unable to execute any movement. The negative features are, that there may be no loss of cutaneous sensibility; the special senses may be intact; and there is no distinct paralysis.

Movements instituted in the affected parts are less vigorous than is natural; the limbs are somewhat inert, and often hang idly by the side or are carried by some mechanical contrivance; but they can be, by a strong effort, rendered almost as vigorous as in health, and the individual, after two or three awkward failures, may succeed in performing some complex act, provided that he thinks much about it and looks fixedly at what he is attempting to do.

If the muscles are pinched forcibly between the fingers, or if they are submitted to the electric current, they exhibit a diminution of sensibility. This has been well shown in a case lately under my care in University College Hospital; the patient did not know when the magneto-electric current was applied to the muscles of the right leg, although they could be seen to act quite vigorously. There was loss of cutaneous sensibility and of muscular power in the same limb, but electric irritability, although diminished, was preserved. As the voluntary power and the sensibility of the skin returned, some electric sensibility returned also, but it was notably deficient long after the electric contractility was almost normal; the patient being scarcely conscious of an amount of actual contraction of the muscles induced by electricity, which amount could not be tolerated for a moment in the muscles of the unaffected limb.

Without looking to see, the patient does not know the position of his limbs; and even when he has voluntarily assumed any attitude or position, he swerves from it if his attention be directed to some other object than his own limbs.

Minor degrees of this disturbance may often be observed in conjunction with definite but partial paralysis, in either a paraplegic or hemiplegic form. Such patients can only move their toes or fingers when they are looking at them; and they do not know, if their eyes are closed, whether they are moving their extremities or not, but, in perfect innocence, may ask the physician to inform them.

Commonly, Muscular Anæsthesia is seen in combination with other evidences of profound change in the nervous centres; but sometimes it exists, and that for a considerable time, alone. It may be, and often is, the precursor of paraplegia, and under such circumstances may be confounded with ataxy, spinal congestion, commencing myelitis, or softening of the cord.

The following case affords a fair illustration of the malady:—

A. B., female, at the age of 18 or 19 years, "caught cold" during menstruation, and soon afterwards felt "loss of power" in the legs and hands; she stumbled in walking, and found it very difficult to dress herself. The symptoms became slowly better, but occasionally returned; and three years after their commencement she married, and at the time of my seeing her, eighteen months after marriage, had a baby three months old.

She walked into my room leaning upon the arm of a friend, but she stumbled, and nearly fell down in doing so; her position in standing, when without support from another person or a table, was that of inclination forwards, and she rocked about from side to side, and antero-posteriorly: when she attempted to walk she occasionally reeled, and did this especially when engaged in conversation. If told to make an effort to walk in a straight line, she looked carefully at her feet and managed to do so without much deviation. When standing with her heels together she maintained steadiness of position as long as her hand was on the table, or she was paying attention to her drill; but, in a moment, if her mind was distracted with conversation, she staggered, and caught at some object for support. She told me that her hands were much better than they had been previously; but that still they were "very odd." It was, as I observed, difficult, and indeed impossible, for her to do sundry little things, such as putting a pin into her dress or taking it out, fastening or unfastening a button, without seeing either her fingers or the reflection of them in the glass. She found it impossible, or very difficult, to play on the pianoforte, and, as she expressed it, she "could not fasten anything she could not see." Objects fell out of her hands when she did not look at them; when standing with support on both sides, but with the eyes closed, she could not raise either foot from the ground,—the sole of the foot seemed glued to the carpet. The cutaneous sensibility was perfect; the electric contractility and sensibility were natural; there was no failure of general health, no tenderness of spine, no alteration in the special senses, no pain; and there were no symptoms of hysteria. All that was lost in this case was the sense of muscular condition and action.

CAUSES.—Nothing definite is known with regard to these, beyond the frequent association of Anæsthesia Muscularis with hysteria.

I once saw a marked case of Muscular and Cutaneous Anæsthesia which had been induced by exposure to cold. The symptoms in this instance were developed suddenly; but in other cases their commencement has been insidious, and their progress slow; and it has been impossible to assign any rational cause for their production. In many they have followed a series of convulsions or other symptoms of hysterical character.

DIAGNOSIS. — From *paraplegia* generally, whatever may be its cause, Muscular Anæsthesia may be distinguished by the facts that power is not lost, and that forcible movements may be determined, although not directed with exactness. In ordinary paraplegia the awkwardness of movement is due to and proportioned to the want of power; in Muscular Anæsthesia there is no such relation. Moreover, the patient exhibits none of the signs of interference with those functions of the spinal centre which are speedily involved in all cases of paraplegia depending upon changes in the nutrition of the cord. The limbs do not waste, the skin undergoes no special alteration, the urine is not altered, and sensation in other directions is unchanged.

From *hemiplegia*, indicative of those cerebral diseases which are usually accompanied by paralysis of one side of the body, Muscular Anæsthesia is separated by considerations similar to some of those which have just been mentioned; but mainly by the absence of conformity of the case to the known types of cerebral lesion, by freedom from intellectual change, and by the limited distribution of the symptoms.

Locomotor ataxy resembles Muscular Anæsthesia in its most striking symptom, viz. want of co-ordinating power; and in many cases of the former there are symptoms of the latter. It was present, for example, in 28 of 50 cases analyzed by Topinard;[1] but it was absent in 22 cases,

[1] De l'Ataxie locomotrice, p. 203; Paris, 1864.

and was but slightly marked in 8. The clinical history of locomotor ataxy is different.[1] There is not necessarily the special want which is the essential condition of Muscular Anæsthesia; and in the latter there is an absence of pain in the limbs, of implication of the genital organs, and of affections of the eyesight.

Hysterical patients often exhibit the phenomena of Muscular Anæsthesia; indeed it is one of the expressions of their malady; and the only point of interest to ascertain is the degree to which this condition, the hysteric, may account for all the symptoms. The general course of the case usually affords the information that is required; it would be unsafe to refer Anæsthesia Muscularis to hysteria, unless other symptoms of the latter disease were present; it would be unwise to suspect the existence of grave central lesion unless hysteria could be excluded.

PATHOLOGY.—The present state of physiology with regard to the existence and nature of the muscular sense is so unsatisfactory that it would be quite idle to occupy much space in the discussion of its pathology.

With regard to the existence of such sense there appears to be evidence similar to that which we possess in respect of other senses, viz. our consciousness of its existence. It is a matter of fact that we do know when, in what direction, and to what degree we contract our muscles. We guide our movements without looking at our limbs, we know where our extremities are placed, we determine movements when we like, and apportion the amount of effort to the task set before us; we guess at the weight of a body by the effort we make to raise it, and do not break an empty egg-shell if we hold it between our fingers in the dark. The patient with Muscular Anæsthesia has lost the power or faculty which renders these adjustments of movement possible. The fact of the existence of a muscular sense may be regarded as established, and also that of its distinctness from all other modes of sensation. It is tolerably certain that the peripheral expansion of the muscular-sense nerves exists in the muscular tissue itself, and not in either the skin or the structures around the joints; but beyond this point there is grave doubt even as to whether the fibres pass in the anterior or posterior roots of the spinal nerves. M. Trousseau[2] admits the existence of muscular sensibility, but denies that of the sense of muscular activity; and the most important fact upon which he bases his opinion is contained in the following words: "Lorsque, fermant les yeux, nous exécutons sans efforts un mouvement assez étendu, il nous est impossible, avec la plus sévère attention, de sentir nos muscles se contracter; mais nous sentons le mouvement imprimé aux leviers que la contraction des muscles met en jeu. Le fait est si vrai, que si nous interrogeons une personne fort intelligente, mais complètement étrangère aux notions anatomiques et physiologiques, et si nous lui demandons quel est le siège du mouvement d'extension et de flexion des doigts, elle le place exclusivement dans la main et jamais dans l'avant-bras." This observation is quite correct, but M. Trousseau's conclusion from it is, I think, erroneous. We do not see objects nor hear sounds *in* either our eyes or ears; but involuntarily project these sensations, not into a distant part of our own body, but into space outside ourselves. The senses of taste, smell, and of tact, we refer to something or somewhere just beyond the extreme peripheral expansion of the nerves which minister to those senses. We do not feel —or mentally recognize as such—the condition of our own nerves, but instinctively and of necessity feel and believe in something outside ourselves, or objective, that presses on the skin; something not ourselves that we taste in our mouths; something not ourselves that we smell in our noses. It is well known that a patient who has lost his leg imagines that he feels pain in his amputated toes, and in this we have another illustration of the principle that the mind does not refer sensation to the spot which receives the impression which may occasion it. Because, therefore, in the act of muscular movement our consciousness refers the sense of such movement to the extremity moved, and not to the moving organ, it is not proved that there is no sense of muscular activity; on the contrary, it is shown by this fact that the muscular sense obeys a law similar to that which we recognize in regard of other senses. For the existence of the sense we have the evidence of consciousness; and for the absence of the sense, there is the testimony of disease. It matters, comparatively speaking, little for our present purpose to determine the exact nature or metaphysical relations of the property in question; it is enough that in health there is a faculty which has been called "muscular sense," and that in disease this function is destroyed; that such disease may exist alone, and that the name by which it is denoted is "Muscular Anæsthesia."

PROGNOSIS.—The future of such cases cannot be predicted with certainty. It may be guessed at by regard to conditions

[1] See article on Locomotor Ataxy.

[2] Article "Ataxie locomotrice progressive," Nouveau Dictionnaire de Médecine et de Chirurgie pratiques, tome 3me, p. 777.

other than those of the affection itself. If it be but one of many symptoms of that manifold disease called hysteria, the prognosis is that of the latter malady; if it be associated with grave changes in other portions or functions of the nervous system, the nature of such ulterior symptoms must determine the prognosis. There is nothing special in the character of the symptoms of Muscular Anæsthesia, *per se*, which can form a satisfactory guide.

TREATMENT.—Faradization of the affected muscles has proved of service, as has friction of the skin, and its electric irritation; but there are no medicines that have been shown to exert any special influence upon this variety of nervous disorder.

WASTING PALSY.

BY WILLIAM ROBERTS, M.D., F.R.C.P.

DEFINITION.—A chronic disease, consisting in a progressive atrophy of the voluntary muscles, independent of any antecedent motor or sensory paralysis. The disease attacks the muscles in groups: in some cases it is *partial*, and limited to the extremities; in other cases it is *general*, and implicates the muscles of the head, neck, and trunk.

SYNONYMS.—Paralysis Atrophica; Progressive Muscular Atrophy; Cruveilhier's Atrophy; Atrophie Musculaire Progressive (Fr.); Progressive Muskelatrophie, Progressive Muskel-lähmung (Ger.).

HISTORY.—Cases of extreme wasting of the muscles of the upper and lower limbs, without loss of voluntary power, were published in this country, in the earlier decades of the present century, by Cooke, Bell, and Darwall; but the establishment of the affection as a distinct type of disease is due to the labors of Cruveilhier, Aran, and Duchenne, in France, in the years 1851–53. The present writer collected all the information existing on the subject up to 1858, in an Essay published in that year.[1] To this Essay the reader is referred for the earlier notices of the disease. Since 1858 the pathology of Wasting Palsy has been elucidated by the investigations of Gull, Lockhart Clarke, Luys, and others.

ETIOLOGY.—The subjects of Wasting Palsy are mostly found among young adults and middle-aged individuals; but children are not unfrequently attacked. The mean age of eighty-eight cases collected by me was thirty years—the youngest was only two years of age, and the eldest sixty-nine. The male sex is considerably more liable to the disease than the female (about six males to every one female). This disproportion probably depends, mainly, on the greater and more sustained muscular exertion which men's occupations demand; also on the greater exposure to cold and external violence of individuals of the male sex. Women of the working-class—washerwomen, domestic servants, sempstresses, &c.—are seemingly not much less liable to Wasting Palsy than men employed in kindred occupations; but females belonging to the easy classes enjoy a remarkable immunity from this disease. It is, however, somewhat difficult to explain why cases arising from hereditary influence should occur more frequently among males than females.

Partial or *local* muscular atrophy prevails mostly among handicraftsmen—mechanics, masons, smiths, miners, needlewomen, scriveners, laborers, and domestic servants. The subjects of *general* Wasting Palsy are found equally in every grade of life.

The influence of *consanguinity* in the production of this disease has been marked in a number of instances. The present writer collected the history of ten families in which a tendency to Wasting Palsy prevailed. In four of these families the disease was confined to two brothers in each. Dr. Meryon's first described cases were four boys who had six healthy sisters. In another family mentioned by him, all the boys—namely, two—were affected, while the two sisters were healthy. A sea captain, whose history is related by Aran, had lost two maternal uncles and a sister by the same disease. In another instance, recorded by the same ob-

[1] An Essay on Wasting Palsy, by William Roberts, M.D. London, 1858.

server, the patient's two aunts had died from general muscular atrophy; and, in a family known to Oppenheimer, two uncles and a cousin were already deceased, while another cousin and two brothers still suffered from the same disease. Altogether these ten families included twenty-nine individuals affected with Wasting Palsy, and of these only four were females. Cases arising from hereditary influence present another well-marked feature—in nearly all of them the disease became generalized, and consequently tended to a fatal termination.

As a rule, the subjects of Wasting Palsy have been persons of good physical development: in several cases the patients are reported to have been men of remarkable muscular power and activity; in a few instances—nearly all of which were associated with a hereditary proclivity to the disease—a certain weakness existed from early youth.

The exciting causes of Wasting Palsy (excluding hereditary predisposition) may be ranged under three heads: namely, excessive muscular action, cold, and disease or violence affecting the spine. In a considerable number of cases, however (36 per cent.), no reasonable cause could be assigned for the breaking out of the disease.

Aran directs attention to the fact that the particular muscles which are necessarily in long-continued contraction in persons following certain mechanical trades (masons, milliners, shoemakers, smiths, &c.), are those which are first invaded and most deeply involved. In persons of this class the muscles of the shoulders, arms, and hands are first affected, and very frequently the atrophy is permanently limited to these parts. There are numerous exceptions, however, to this rule.

Cases arising from cold (wearing of damp apparel, immersion of the limbs in cold water, rapid cooling of the perspiring surface, exposure to inclement weather) are marked by a train of neuralgic or so-called rheumatic pains in the affected parts, either at the onset of the atrophy, and ceasing when this has fairly set in, or continuing throughout its progress, and imparting a special character to the symptoms. The invasion of the disease in this class of cases is often somewhat sudden, and accompanied by cramps and twitches of the muscles.

In cases traceable to cold, the wasting is more apt to extend to the muscles of the trunk than in cases due to overwork. Of twenty-five cases attributed to overwork, eighteen were partial and only seven general; whereas, of the sixteen cases charged to the agency of cold, six were local and ten general. These two causes are often in operation together: the miners in my neighborhood, who work in damp or wet excavations, are frequent victims of Wasting Palsy.

The connection of Wasting Palsy with injury or disease directly or indirectly implicating the spinal cord, has of late years attracted increasing attention; and the interpretation of these cases has an important bearing on the pathology of the disease, as will be more particularly noticed hereafter. The history of some antecedent violence occurs too frequently in the reports of cases of Wasting Palsy to allow of its being set aside as a merely fortuitous circumstance, though the precise connection between the injury and the subsequent atrophy is often obscure. In a youth under my care at the Manchester Infirmary, who ultimately died from implication of the respiratory muscles, the first symptoms of atrophy in the ball of the right thumb occurred six months after the fall of a bale of cotton cloth on the nape of the neck. The immediate effects of the injury were confined to slight stiffness of the neck, and occasional pains extending down the arms. Valentiner records a case in which the first failure of health followed a fall on the back from a height of eight or ten feet: yet the atrophy did not appear until six years after. Bergmann's patient[1] fell on his back from a horse, and lay for a while unconscious. From this time he suffered pain and stiffness in moving the head; afterwards, and very slowly, a weakness in the shoulders came on, which ended in complete atrophy of the muscles around the shoulder joints. In a remarkable case recently reported by Dr. Thudichum and Mr. Lockhart Clarke, a gentleman, æt. 54, suffered what he considered a slight injury. In jumping across a flower-bed for a wager, he came down heavily on his heels, and then fell backwards upon his head. He was stunned for a time, but gradually recovered, and, after some days' confinement to his bed, appeared to be quite well again. It was, however, soon perceived that a great change took place in his habits. Having been extremely fond of manly sports and exercises—rowing, cricketing, riding on horseback, dancing, and the like—he discontinued to take part in any of these, although he continued to go every autumn to the Scotch moors for the purpose of shooting grouse. Five years after the above-mentioned accident, while engaged in this last-named sport, he perceived that his right leg had lost a part of its usual strength. From this time gradual atrophy and loss of power in the muscles crept over the patient, until at length death took place from failure of the respiratory muscles. Wide-spread degeneration of the spinal cord was found after

[1] St. Petersburger Med. Zeitsch., p. 116. 1864.

death. (Beale's Archives of Medicine, 1863.)

In other cases, disease, manifestly primary, of the spinal cord is followed by complete atrophy of certain groups of muscles. In a case published a few years ago in the *London Medical Review* by the present writer, a young man suffered from acute general paralysis of all the muscles of the extremities, and of most of those of the trunk. The intellect was not affected. Gradually, in the course of months, the patient recovered the power of the muscles; but after complete restoration of the remainder of the body, the intrinsic muscles of both the hands and feet passed into a state of total atrophy, and still continue in the same condition. In the so-called essential paralysis of infancy and childhood—which is evidently of spinal origin—certain limited groups of muscles not unfrequently pass into a state of permanent atrophy, while the remaining portions of the paralyzed members recover their mobility.

Certain other exciting causes of Wasting Palsy are sometimes doubtfully mentioned—namely, constitutional syphilis, venereal excesses, onanism, and antecedent zymotic fevers.

Symptoms.—The invasion of Wasting Palsy is always gradual, and the disease has usually been in progress some weeks or months before the patient discovers its existence. The first symptom perceived is a certain weakness in the affected member: the tailor finds he cannot hold his needle; the shoemaker cannot thrust his awl; the mason fails to wield his hammer; the gentleman experiences an awkwardness in handling his pen, in pulling out his pocket handkerchief, or in putting on his hat. Some such incident calls attention to the affected limb, which is then usually discovered to be more or less wasted and shrunken.

The disease begins, in the great majority of cases, in the upper extremities, either in the ball of the thumb and hand, or in the shoulder—much more commonly in the former than in the latter. Sometimes, however, it begins in the muscles of the neck, of the face, the tongue, in the thigh, the leg, or the foot. The extension or spread of the disease follows an erratic course. In the immense majority of cases the disease is permanently limited to one or a few groups of muscles in the upper or lower extremities; in other cases, and these are by far more formidable, the atrophy invades successively the voluntary muscles of the entire body, trunk and extremities. The only muscles which, as yet, have not been known to be attacked, are those of mastication, and those which move the eyeball. When the atrophy is confined to certain regions of the extremities, the life of the sufferer is not imperilled; but when the trunk is invaded, and the muscles of respiration participate in the disease, death by suffocation is the ultimate result.

The wasting and disappearance of the muscles produce notable changes in the configuration of the body. The natural rounded contour of the limbs is replaced by an unsightly flattening; the bones stand out in unaccustomed distinctness, giving to the member the appearance of a skeleton clothed in skin; but the skin itself, and the subcutaneous cellular tissue, undergo no change, and cannot be distinguished from the integuments of healthy parts. Certain distortions of the head, trunk, and extremities are also occasioned by the unequal wasting of opposed groups of muscles—those less atrophied overcoming the resistance of those more diseased. These changes of configuration are a marked feature of Wasting Palsy. The hand is frequently the seat of a very singular deformity—namely, the "claw-shaped" hand, or "main en griffe" of French writers. The palm is robbed of its muscular cushions; flat planes or hollows occupy the sites of the thenar and hypothenar eminences; the hollow of the hand is traversed by the visibly prominent diverging flexor tendons, which are stretched between the wrist and the bulging bases of the fingers; the proximal phalanges are bent backwards, away from the hollow of the hand, while the middle and distal ones, inclined in an opposite direction, are in a state of continued semiflexion. The back of the hand is hollowed out in long furrows, corresponding to the interosseous spaces, and the first joints of the fingers are pulled backwards, giving the hand a broken-backed appearance.

Passing up the limb, the forearm is found flattened, or even hollowed, on its anterior and posterior aspects. When the shoulders are affected, the whole arm dangles powerlessly at the side; the roundness of the shoulder has given place to a flattening, and the head of the humerus, the acromion, and the coracoid processes are plainly discerned through the thin covering of skin. If the serratus magnus be destroyed, the angle of the scapula is tilted upwards and inwards, and stands prominently out from the trunk. Corresponding deformities are witnessed when the lower limbs are invaded: the foot is distorted by the unequal involvement of its extrinsic and intrinsic muscles, and contractions of the toes on the sole, deflections of the foot inwards, or of the heel upwards, are produced—interfering very seriously with the steadiness of progression.

But perhaps the most remarkable of all the anatomical changes are seen in the face, when the muscles of expression are

destroyed. The face is veiled, as it were, by an impenetrable mask; no emotion changes its unvarying aspect—the expression is always solemn, stolid, and unmoved. The muscles of the eyeball are, however, spared, and by their movements alone, in the later periods, the mind holds an imperfect communion with the external world. The oral and buccal muscles are usually invaded early, and the saliva dribbles over the lips. When the muscles of the neck are involved, the head falls forwards—the chin resting on the sternum—or, laterally, the head falling over on the shoulder.

When the abdominal muscles are implicated, the lumbar curve is enormously exaggerated by the unopposed action of the erector spinæ, and the belly projects in front, while the chest is thrown back as a counterpoise. The invasion of the lingual muscles leads to a falter in the speech, and to imperfect comminution of food in the mouth. The involvement of the laryngeal muscles produces a change in the voice, which loses its register, and is finally reduced to a monotone. When the diaphragm and intercostals are reached, violent suffocative fits of coughing are occasioned; the play of the chest is at length so reduced that a slight additional difficulty to respiration proves fatal. Dissolution is usually brought about by a bronchitic seizure; the air-tubes are speedily clogged with mucus, which no efforts of the patient can dislodge, and rapid asphyxia closes the scene.

When the disease is partial in its extension, it is observed that certain parts of the body, and certain groups of muscles, are much more obnoxious to its inroads than others. The muscles of the trunk are less liable than those of the extremities, and those of the lower extremities are far less frequently affected than those of the upper. Of sixty-two cases of partial Wasting Palsy collected by me, the upper extremities were alone affected fifty-one times, the lower extremities alone five times, and the upper and lower together seven times. The right arm was much more frequently attacked than the left, and the hands oftener than the shoulders. As a general rule, it was found that when one limb was attacked, its fellow of the opposite side shared its fate; that when the disease was unilateral, the right side was more likely to be its seat than the left.

One of the most striking characteristics of Wasting Palsy is the capriciousness of its line of attack. Scarcely two instances are exactly alike in the combination of muscles implicated—hence an almost infinite variety of feature; yet there are certain more common combinations.

Among the most common cases are those in which the disease is confined to the hands, or to the hands and forearms. Not uncommon, likewise, are the cases in which the shoulder and upper arm of one or both sides are atrophied, while the forearms and hands remain healthy.

Coincidently with the loss of substance in the muscular masses, there is necessarily a corresponding loss of power. Certain less constant symptoms also sometimes make their appearance—namely, fibrillary tremors, cramps, twitches, and diminution of electric contractility in the muscles.

The loss of power corresponds, in the typical cases, very exactly to the grade of muscular atrophy, and gradually proceeds as the muscles diminish in bulk. In extreme cases absolute immobility of the limb, or part, is at length produced; more commonly the various movements are still capable of being performed, but with greatly diminished force. Not unfrequently, however, this correspondence is not exact; and the loss of power exceeds, more or less considerably, what is due to mere atrophy of the muscular fibres.

During the active stage of the disease the affected muscles sometimes exhibit curious vibratile tremors—fugitive wavy oscillations of the muscular fibres—which are visible under the skin, but do not produce any movement of the limb, nor are they sensible to the patient. When absent, they may occasionally be evoked by stripping the part or filliping the skin. These vibrations are sometimes the earliest symptom of a new advance of the disease into parts not yet affected. They disappear altogether when the atrophy has reached an extreme degree, or when its progress has been arrested.

In uncomplicated cases the muscles of the wasting members respond to the electric stimulus readily, and with a force corresponding to their bulk. As a rule, there is no alteration in the tactile sensibility of the affected limbs; but in rare cases there is a slight numbness of the skin, and not unfrequently the parts are highly sensitive to impressions of cold. In about half the cases there is more or less pain of a neuralgic character in the course of the nerves leading to the diseased muscles, or in the neighborhood of the muscles themselves. In some cases pain of an agonizing character is a marked feature of the complaint.

The general health is usually quite unaffected, the intelligence is clear, and the functions of organic life are performed with their usual regularity, so long as the muscles of deglutition and respiration are spared.

Course and Duration.—The course of Wasting Palsy is essentially chronic, and its duration uncertain. After destroying a certain group of muscles it may

be permanently arrested, or it may proceed step by step until nearly all the voluntary muscles are disabled. The atrophied muscles may be again restored by therapeutical means to their original bulk: this is unfortunately not a very common termination—more commonly the wasted parts are crippled for the remainder of life. When the disease is progressive, its advance is seldom continuous, but is rather marked by repeated pauses and recommencements. The pauses may extend over a few weeks or months, or even several years. In a case now under my care in the Manchester Infirmary, the disease has started afresh in great intensity, after complete arrest for five years. In twenty-eight cases in which I was able to ascertain the continuance of the active process, the mean duration was thirty-eight months. Of these, four ended in recovery, thirteen in permanent arrest, and eleven in death. The cases which ended in recovery had a mean duration of fourteen months, those ending in arrest a mean duration of twenty-seven months, and those ending in death averaged a duration of more than five years.

Cases which could be traced to the effects of over-exercise of the muscles, were nearly always found to terminate in permanent arrest after the destruction of one or more groups of muscles; whereas cases which appeared to have arisen from exposure to cold, or from hereditary predisposition, showed a more decided tendency to a progressive course and a fatal termination.

Diagnosis.—The partial form is liable to be confounded with paralysis from injury to a motor nerve, lead palsy, and malarious palsy. In all these there is a marked atrophy of the muscles; and the affection may be confined to a narrow region, around which are healthy muscles, offering a strong contrast to the decayed ones.

Atrophy, resulting from injury to a nerve, is distinguished by the exact limitation of the wasting to the parts supplied by that nerve; also, if the nerve be a mixed one, there is, or was, an accompanying loss of sensation.

In lead palsy there is a comparatively sudden invasion: in a day or two—a week, or a fortnight, at most—the paralysis is at its height; whereas in Wasting Palsy the loss of power is excessively gradual. The precursory or concomitant phenomena distinctive of lead poisoning, seldom or never altogether fail—namely, colic, blue line on the gums, tremblings, pallor, and other symptoms of saturnine cachexia. Duchenne states, that the electric contractility of the muscles is markedly diminished or altogether lost in lead palsy; whereas, in Wasting Palsy, the muscles respond to the electric stimulus in a degree proportionate to their bulk. It will also be remembered that in saturnine poisoning the atrophy is distinctly essential to the paralysis.

From ordinary general paralysis of central origin, Wasting Palsy is distinguished by the *dissecting* character of its march. It attacks the muscles in separate groups—in detail, as it were—and does not diffuse its ravages uniformly over extensive regions or the entire body. It is very rare also that in general paralysis the wasting of the muscular masses bears any proportion to the loss of power.

Extreme muscular atrophy sometimes follows infantile paralysis, and the distribution of the disease may resemble that of Wasting Palsy and produce ultimate results indistinguishable therefrom. The cases are, however, totally different in their history. Infantile paralysis has always a sudden invasion, and the wasting is subsequent to the loss of power.

Morbid Anatomy. — The essential changes found in the bodies of persons who have died from Wasting Palsy are confined to the muscles, the spinal cord, and the nerves.

The muscles of the affected regions are found wasted in various degrees. Some are only slightly atrophied, others more profoundly, while others again are reduced to pale, thin, membranous strata, or are altogether destroyed, and can only be identified by comparing the origins and insertions of certain fibro-cellular bands, which are the vestigial representatives of the previously existing muscular masses. The color of the wasted muscles is changed to a pale red or rose, sometimes with a buff or ochreous tinge, and not unfrequently streaks of adipose tissue run, in lines, between the fibres. Where there is much fatty change, the wasting, which is so conspicuous a characteristic of the disease, is less marked; sometimes even the muscles are almost undiminished in bulk, but are transformed into masses of fat. This peculiarity has been observed only in the lower extremities.

The difference in the degree of atrophy undergone by adjoining muscles, and sometimes even by different parts of the same muscle, is very remarkable. Scarcely any two muscles are affected in an equal degree. Side by side with a pale, almost filamentous remnant, may be found a muscle of full red color and undiminished bulk. One or two fasciculi of an affected muscle may survive in vigor after the destruction of the remainder.

The decayed muscles have been examined microscopically by Meryon, Galliet, Oppenheimer, Virchow, and others. Meryon describes the primitive fibres as completely destroyed, the sarcous elements

being diffused, and, in many places, converted into oil-globules and granular matter; while the sarcolemma was broken down and destroyed. Gailiet, who examined the muscles in one of Cruveilhier's cases, states that in those parts of the muscle which had retained a rosy hue, the primitive fibres had preserved their striæ tolerably distinct, and between the striæ were seen fine gray or brilliant molecules, resembling fat. In the completely decolorized parts—those which to the naked eye appeared of a straw tint—there could still be recognized long cylinders, representing the primitive fibres. The sarcolemma was preserved, but the contained substance had lost its striated character, and was replaced by a uniform granular mass, presenting numerous minute gray molecules mixed with fatty granules. In parts where the disease was still further advanced, the granular matter and its enveloping sarcolemma had entirely disappeared, and there remained only the fibro-cellular framework of the muscle, destitute of any true sarcous tissue.

The condition of the spinal cord and of the spinal nerves has been examined in some thirty-five cases, of which thirty-four have been tabulated by Bergmann.[1] The results of the investigations have not been by any means uniform. In sixteen cases the cord and the nerves were pronounced healthy, and in six of these the parts were examined microscopically. In six cases the cord itself was found healthy, but there was marked atrophy of the anterior roots of a certain number of spinal nerves. In one case both the spinal cord and nerves were healthy, but there existed disease of the medulla oblongata. In six cases the cord was found diseased when examined microscopically, though it appeared sound, or nearly so, to the naked eye. Lastly, in seven cases the cord appeared to the unaided senses palpably softened and disorganized.

Atrophy of the anterior roots was first noticed by Cruveilhier, and was supposed by him to supply the key to the pathology of this disease. He thus describes the condition of these structures in the body of the showman Lecompte, who died from general Wasting Palsy of five years' duration: "The anterior roots of the spinal nerves are remarkably small compared with the posterior, and this inferiority of size is particularly great in the cervical region. The proportion between the two roots had become greatly changed. According to my observations, in the normal state, the posterior roots compare with the anterior, in the cervical region, as three to one; in the dorsal region, as one and a half to one; and in the lumbar region, as two to one. But here the proportion was as ten to one in the cervical, five to one in the dorsal and lumbar regions. Further, by plunging the cord into dilute nitric acid, I was able to observe that a very large number of the anterior cervical rootlets had been completely reduced to their neurilemma, and appeared as gray filaments, which, searched with a strong lens, presented no trace of nervous tissue; while, on the other hand, the anterior roots in the dorsal and lumbar regions had only suffered atrophy by emaciation. I was unable to trace the gray nervous filaments, or those simply atrophied, beyond the point where the anterior root joins the posterior; but I was able to establish the existence of atrophy of the nerves as they were about to penetrate the muscles."[1] A similar atrophy of the anterior roots was found in ten other cases, either with or without discoverable disease of the corresponding regions of the cord. In the great majority of the cases, however, the anterior roots were not perceptibly atrophied, and this leads directly to the inference that such atrophy is not an *essential* feature of the morbid anatomy of Wasting Palsy.

The morbid anatomy of the spinal cord is confessedly a subject of great difficulty. Until recently only the coarser changes of consistence—softening or induration—were appreciated by pathologists; and even after the microscope had been brought in aid of the examination, it soon became apparent that very important changes in the structure of the cord might be overlooked, unless the observer possessed special skill and practice in this branch of inquiry. The positive results of Gull, Lockhart Clarke, and Luys, who may be regarded as experts in the examination of the spinal cord, throw considerable doubt on the trustworthiness of the negative results obtained by Meryon, Savory, Oppenheimer, Friedburg, and others, who failed to detect in the spinal cords of patients who had died from Wasting Palsy any appreciable changes of structure.

Luys describes as follows the microscopical changes in the *apparently* sound cord of a man, aged fifty-seven years, who died of pneumonia, and who had been the subject of advanced atrophy of the muscles of the left hand and forearm. There was also slight atrophy of the muscles of the right hand. The loss of power had corresponded accurately with the degree of wasting. Five of the anterior roots coming off from the cervical enlargement of the cord were atrophied. The microscopic examination of the cord showed increase of the capillary vessels in the gray substance at the level of the atrophied roots.

[1] St. Petersburger Medicinische Zeitschrift, Bd. vii., 1864.

[1] Archives Générales, 1853.

The walls of the vessels were thickened and surrounded with a granular deposit, which extended into the gray substance. In the anterior gray cornua, at the point of exit of the anterior roots, there was an absence of nerve-cells, which were replaced by granular deposit. Some of the nerve-cells of the anterior horns were in process of degeneration—brownish, and filled with dark granulations. These changes were found especially on the left side, and very slightly on the right side. The rest of the cord was healthy.[1]

Dr. Gull gives an account of a man, aged forty-nine years, who became the subject of Wasting Palsy after striking his head against a beam, whilst driving under an archway. Some months after this accident he began to suffer pain from the occiput down over the shoulders, and in about a year the muscles of the upper extremities began to waste. Three years after the accident he was admitted into Guy's Hospital. He then presented a remarkable example of muscular atrophy, without actual paralysis. The upper extremities were principally affected. The extensors of the right hand, the muscles of the thumb, and the interossei were extremely wasted. The wrist dropped. The muscles of the shoulder and arm, including the pectoralis major and minor, were much wasted; but in a marked degree less so than those of the forearm and hand. Very slight diminution of sensation. He could still lift the arm over the head. The left arm was similarly affected, but less than the right, so far as muscular atrophy was concerned—but there was numbness through the whole arm down to the fingers, accompanied with severe neuralgic pains. The trapezii, serrati postici superiores, rhomboidei, and all the long muscles of the neck and back were remarkably atrophied. The legs were wasted and weak, but the patient was able to walk. There was constipation and dribbling of the urine. He died with febrile symptoms and dyspnœa.

Autopsy.—Sections of the cord examined with the naked eye gave no distinct evidence of disease. There was a slight yellowishness of the posterior columns, and increased vascularity and thickening of the pia mater covering them. In these columns, especially in the right one, abundance of granule-cells were discovered with the microscope. The exudation was greatest in the middle and lower third of the cervical enlargement. The gray matter was hyperæmic. There was no exudation into its tissue, nor into the anterior columns. The ventricle of the cord was enlarged and distended with delicate granular nuclei.[2]

[1] Gaz. Méd. de Paris, 1860. No. 32.

[2] Guy's Hospital Reports, 3d Series, vol. iv. p. 194.

The limitation of structural changes in the cord to narrow tracts and spaces, with a healthy state of the intervening parts, and the absence of any alterations visible to the naked eye, are also strikingly illustrated in the case of Dr. P., whose spinal cord was subjected to an exhaustive examination by Mr. Lockhart Clarke.

Dr. P., æt. 65, engaged in literary pursuits, began to complain some five years before his death of neuralgic pains in the ball of the thumbs of both hands, which before long extended to the forearms and arms. After some months there was marked weakness and wasting of the muscles of the thumbs and index fingers, which also became bent inwards towards the palms. The loss of power and volume in the muscles progressed steadily, accompanied with the most excruciating pains, until his death. The right hand and arm were more profoundly affected than the left. In the later periods of the disease the pains extended to the lower limbs. The right pupil was constantly larger than the left, but the movements of the two were normal.

The cerebellum, pons Varolii, medulla oblongata, and spinal cord, were hardened in dilute chromic acid, and sent to Mr. Lockhart Clarke. He found nothing unusual in the external aspect of the cord, neither were the anterior roots of its nerves, in any of the regions, smaller than usual to any appreciable extent. The interior of the cord, from the *filum terminale* through the whole of the lumbar and dorsal region, to the lower end of the cervical enlargement, presented no actual change of structure, either in the white or gray substance; but there was a considerable deposit of corpora amylacea round the central canal. In the cervical region, however, the case was different; for here there were decided evidences of morbid changes of structure in the posterior gray substance. These structural changes extended in a variable degree from the lower end of the cervical enlargement upwards to the third cervical nerves: they were more conspicuous at its upper than its lower part. Thin transverse sections of this part of the cord presented to the naked eye no appearance that would excite suspicion of any lesion whatever; for the morbid portions, although numerous, were small and isolated. Under a low magnifying power the posterior gray substance was seen to be interspersed with a number of unnaturally transparent streaks, patches, or spots, of different shapes and sizes. Some of these spots were seen to interrupt the course of certain nerve-fibres which extended from both the anterior and posterior cornua to the opposite side. In all the sections examined, it was around or at the side of the bloodvessels that the

morbid appearances were most frequently found. The morbid spots were more numerous and extensive on the right side than on the left. The morbid spaces varied in shape, size, and relative position in the different sections. In some they appeared as mere fissures or cracks, which, under a low power, might have been considered as the result of accident, if they had not been so uniformly found in only one portion of the gray substance, and more on the one side than on the other. But when a sufficiently high power was employed, it became at once evident that they were not merely vacant spaces, but composed of a substance which differed entirely in its nature from that of the surrounding tissue. This substance had a delicate, transparent, and very finely-granular aspect. The granules were more closely aggregated towards the centre of the mass, but were generally so fine that they could not be distinctly seen under a magnifying power much less than 400 diameters. Sometimes at the edges of these morbid spaces there seemed to be a kind of transition or degeneration of the surrounding nerve-tissue into the granular substance of which they were composed. In some instances, the broken ends of nerve-fibres proceeding from the posterior roots were seen to project into the opposite sides of these spaces, across which there was strong reason to believe that they had once been continuous.

The morbid appearances generally disappeared about the level of the third pair of cervical nerves; in the middle third of the cervical enlargement they appeared to be more extensive than elsewhere, and they disappeared on approaching the dorsal region. The sympathetic in the neck was also examined, and found normal.[1]

The peripheral distribution of the nerves to the wasted muscles was in some cases found unaltered; in other cases the nerves were found atrophied; and in one instance, examined by Frommann,[2] the nerves leading to the atrophied muscles contained fat-molecules and granular pigment.

The sympathetic in the neck was found diseased in a case examined by Schneevoogt.[3] The ganglionic cord was found extensively affected with fatty degeneration. Two similar cases have more recently been communicated by Jacoud to the Société Médicale des Hôpitaux.[4] On the other hand, the sympathetic, in two other cases, examined by Landry and Bayldon,[5] was found perfectly healthy.

[1] Beale's Archives of Medicine. 1861.
[2] Deutsche Klinik. 1857.
[3] Schmidt's Jahrb. 1857.
[4] Nouveau Dict. de Méd. et de Chir. Paris, 1866. P. 48.
[5] See Author's Essay, p. 163, and Beale's Archives, 1861, p. 11.

PATHOLOGY.—Although defective nutrition of the muscles, ending in degeneration and atrophy, is an invariable feature of Wasting Palsy, it is evident that something more is necessary to the conception of the disease as a nosological entity. Muscles may be atrophied under a variety of pathological conditions, which are essentially distinct. Muscles may waste from want of use, as is witnessed in limbs which are temporarily kept immovable by surgical appliances, or more permanently by anchylosis of the joints. A similar result follows severance of the connection between a muscle and its nervous centres, especially its spinal centres; and, lastly, atrophy of muscle may follow metallic poisoning. In Wasting Palsy there is also muscular atrophy, and, so far as is known, the local changes are not essentially different from those occurring in the afore-mentioned cases; and yet how widely different is the clinical significance of the fact! In order, therefore, to obtain any clear idea of the pathology of Wasting Palsy, it is absolutely necessary to consider circumstances which are antecedent to the mere atrophy.

It must be borne in mind that the several vital endowments of a muscle may be struck with paralysis in their entirety, or singly, or in certain combinations. A muscle paralyzed by a cerebral lesion loses its voluntary power, but it retains its reflex functions and its power of self-nutrition, and does not become atrophied. Other cases are known in which the peculiar "muscular sense" is lost, with preservation of all the contractile and nutritive endowments. In Wasting Palsy, the muscle preserves its voluntary and reflex contractility, its muscular sense, and its sensitiveness to the electric stimulus; but it loses its power of healthy nutrition, and becomes degenerated and atrophied.

Pathologically, Wasting Palsy may be defined as an atrophic degeneration of certain groups of muscles, independent of any antecedent loss of mobility, or of any metallic poisoning. But the question immediately arises, whether the morbid process is primarily in the muscle itself, or in some part of the nervous system which controls its functions.

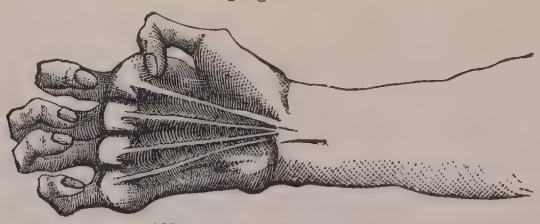
The former opinion has been adopted by Aran, Duchenne, Friedberg, Dr. Meryon, and others; it was also advocated by the present writer in his Essay on the subject, published in 1858. It must, however, be admitted that the additional facts observed since that epoch have tended materially to weaken this opinion, and to give support to the view that the primary lesion in Wasting Palsy exists in the spinal cord, or, at least, in some part of the nervous system.

The principal arguments against a nervous origin of the complaint consisted in

the failure to discover, in several of the earlier post-mortem examinations, any palpable alteration in the spinal cord; and, secondly, in the want of correspondence between the range of muscles affected and the distribution of the nervous trunks. With regard to the former point, the multiplication of post-mortem examinations has very greatly increased the proportion of cases in which a lesion was discovered in the nervous system, and very much strengthened the suspicion that the earlier observations, in which the spinal cord was pronounced to be healthy, were not altogether trustworthy. The researches of Luys and Lockhart Clarke have demonstrated that profound changes in the substance of the cord may exist in detached and very limited areas, which might very easily be overlooked, seeing that it is exceedingly difficult to examine every individual section of the cord with the requisite care. Mr. Lockhart Clarke, speaking on this point, very significantly observes: "There may be very obscure structural changes in the gray substance of the cord, or perhaps only in the ganglia on the posterior roots of the nerves, that may affect the nutrition of the parts to which they are subservient, without interfering with the functions either of sensation or motion; and in cases where the lesions occur in small isolated spots, the limitation of disease to particular muscles, or even to particular fasciculi of any one muscle, could be explained, I think, by the particular nerve-fibrils within the gray substance." (Beale's Archives, 1861, p. 21.)

The opinion, also, seems to be steadily gaining ground, that the nutrition of the muscles is placed under the control of a

[Fig. 41.



"Main en Griffe." (Roberts.)]

special set of organic nerves, having upward connections with the sympathetic ganglia and the cerebro-spinal axis, which are by no means identical with the central connections of the motor nerve-fibres of the same muscles.

Assuming the existence of such nutritive centres, all the clinical phenomena of Wasting Palsy, and the various findings of the post-mortem examinations, admit of easy explanation on the supposition that these centres, or some of their ganglionic connections, are the primary seat of the disease. And the numerous associations and complications of the disease can scarcely be accounted for on any other hypothesis.

In considerably more than one-half of the cases now collected, and examined after death, actual disease was found in some part of the nervous system. This is a proportion which does not permit the assumption of a coincidence of two independent morbid processes. Some relation between the atrophy of the muscle and the disease of the nervous system must, I think, be admitted. Either it must be assumed that the disease of the muscle is capable of evoking disease of the corresponding nervous centre, or the converse. And although the former supposition is by no means a difficult one *à priori*, it stands on a very slender basis of fact. So far as I know, the only authenticated instance of the centripetal transmission of a morbid process along a nervous trunk is atrophy of the optic trunks after destruction of the eye. With regard to the muscles, evidence of any such transmission has yet to be given; the observations hitherto made, indeed, tend the other way. Schiff,[1] who made resections both of mixed and of purely motor and sensory nerves, found no alteration in the central portions of the cut nerves even after the lapse of a year and three-quarters. Türck[2] also examined the central origins of the nerves and their vicinities, in withered and amputated limbs, without finding appreciable alteration therein. Nor are suppurative and cancerous affections of the muscles known to be capable of transmission along the nervous trunks to the nervous centres.

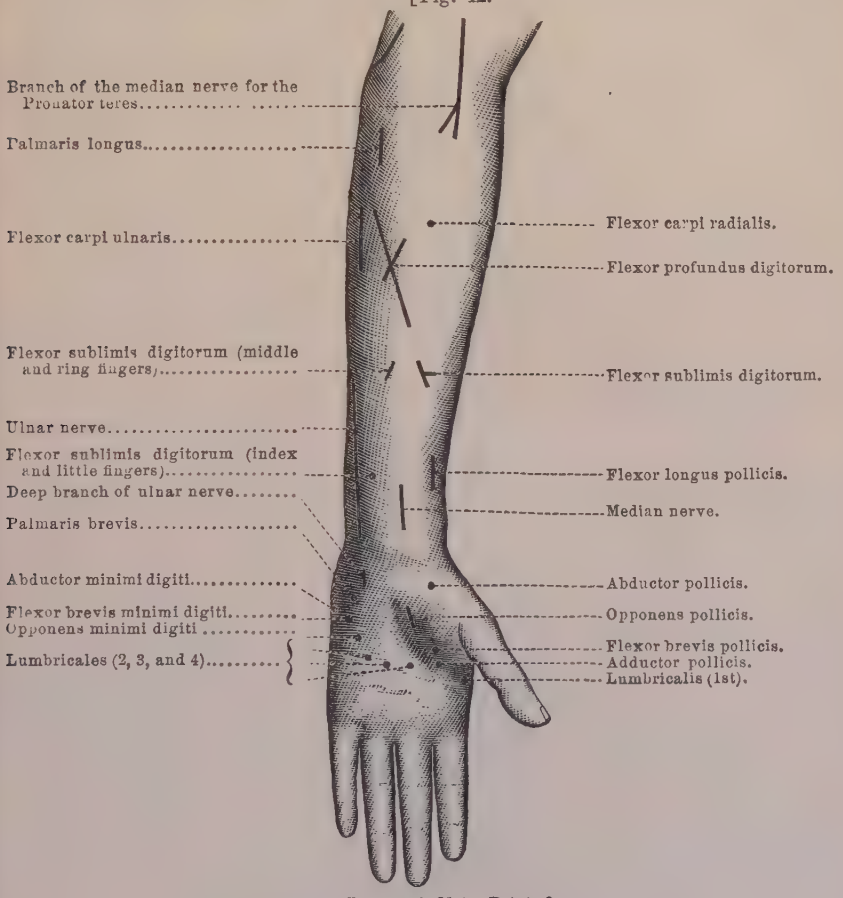
The etiological conditions of some cases of Wasting Palsy, and the collateral phenomena in others, point also very strongly to a nervous origin. Several of the cases were sequential to falls or blows on the neck, or were associated with morbid growths in the spinal canal. In several

[1] Muskel-und Nerven-physiologie. Jahr. 1859. P. 122.

[2] Zeitsch. der K. K. Gesellschaft der Aerzte in Wien. 1853.

well-marked cases of Wasting Palsy, also, motor paralysis, of undoubted central origin, affecting either the atrophied muscles or some other parts, preceded the atrophy. A strong impression was made on my mind by a case of this kind which fell under my notice four years ago. A young man was affected with acute paralysis of the voluntary muscles of the upper and lower extremities, unaccompanied by any wasting beyond what was due to general emaciation. After an almost total loss of motion for a period of three months, recovery set in, which, in the course of a few months, ended in complete restoration of the muscular power in all parts except the hands and feet. The muscles of these latter parts passed on to a state of characteristic atrophy, from which only partial recovery took place. Dumenil, Duchenne, and Trousseau have also published cases in which there existed motor paralysis of the tongue without atrophy, combined with atrophy

[Fig. 42.



Ziemssen's Motor Points.]

without paralysis (Wasting Palsy) of the upper extremities.[1] It is easy to conceive that a morbid process in the motor centres may extend by continuity of tissue to contiguous nutritive centres (supposing such to exist), or, conversely, that disease of the nutritive centres may implicate motor or sensory centres in their vicinity, and so produce complicated clinical phenomena, analogous to those above mentioned, and which on any other supposition are very difficult of rational explanation.

The case-history of Wasting Palsy is rich in combinations of this sort. In the pure typical, uncomplicated cases—where atrophy of the muscles is unmixed with any degree of motor paralysis, or convulsive movements, or with numbness, or neuralgic pains—it may be assumed that the morbid process is strictly limited to the nutritive centres in the cord, or to their connections in the sympathetic ganglia. In the complicated cases it may

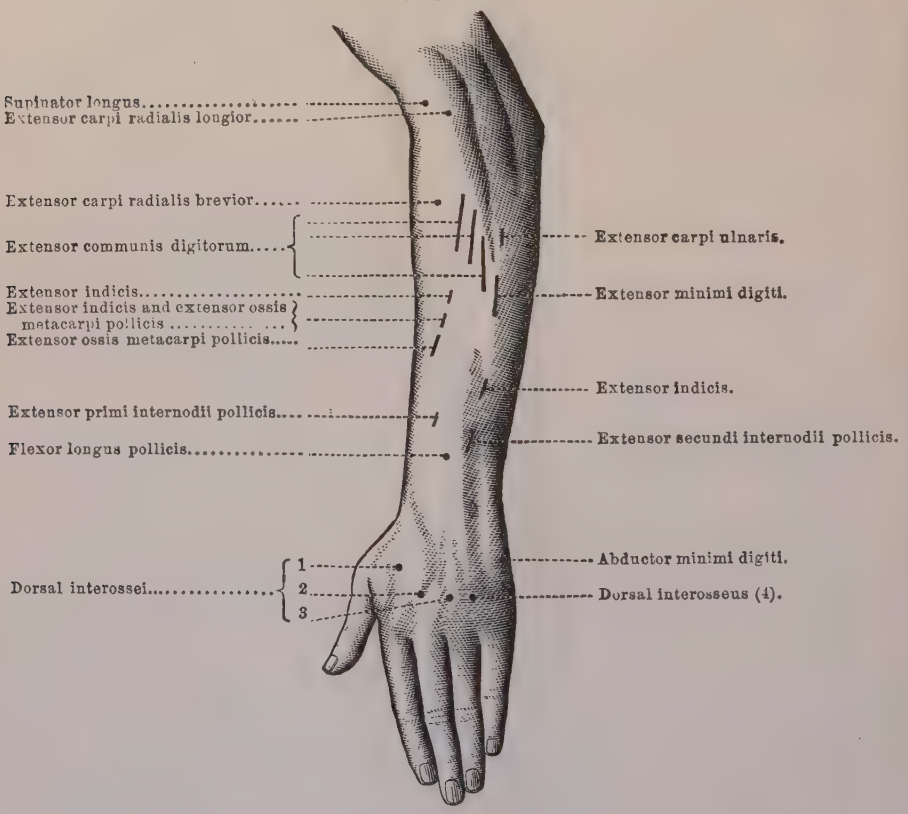
[1] Bergmann, loc. cit. p. 88.

be assumed that the morbid process radiates into those contiguous parts of the cord which control motor and sensory functions. The question can only be finally elucidated by repeated accurate examinations of the spinal cord in complicated cases.

[Of those who have more recently investigated this subject, Joffroy, Hayem, and Charcot, assert the coincidence of Progressive Muscular Atrophy with degeneration of the anterior gray cornua of the spinal cord. Friedreich, Lichteim, and Cohnheim, however, have reported cases in which no lesion either of the cord or nerves was found.—H.]

Prognosis.—Wasting Palsy must be counted among the most intractable diseases; and when it invades the muscles of the trunk, it almost always goes on—sometimes very slowly, sometimes more rapidly—to a fatal termination. In the partial forms—when permanent limitation of the disease to one or two members is established—life may be regarded as no longer menaced, but the usefulness of the limb, if the atrophy be complete, is hopelessly impaired. If remedial measures

[Fig. 43.



Ziemssen's Motor Points.]

can be applied early, and persevered in, while the atrophy is still in progress, there is some prospect either that the advance of the disease may be permanently checked, or even that partial or perfect restoration of the injured muscles may be effected.

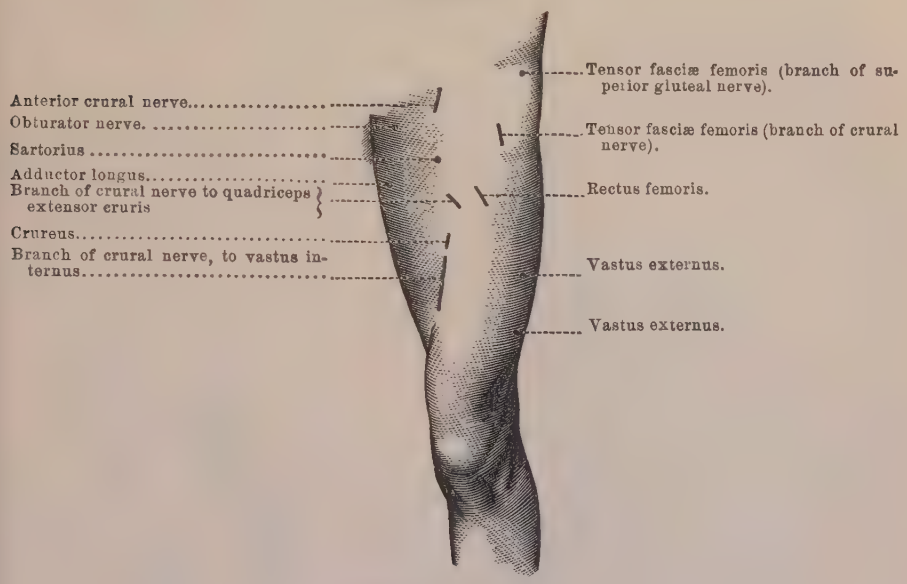
The gravity of the prognosis, in so far as the preservation of life is concerned, depends on the disease confining itself to the extremities, or extending its ravages to the muscles of the trunk. When the respiratory muscles are invaded, the fatal termination is not far distant.

The probability of the disease becoming generalized is greatest when the origin of it can be traced back to hereditary predisposition. The same danger, though in a greatly inferior degree, is to be apprehended when the disease has arisen from cold, and when the lower limbs are the first attacked; also when the upper and lower limbs are *both* implicated. On the other hand, the prognosis is much more favorable when the disease is occasioned by overwork, and when it is confined to the hands and forearms.

The longer the atrophy has existed, the

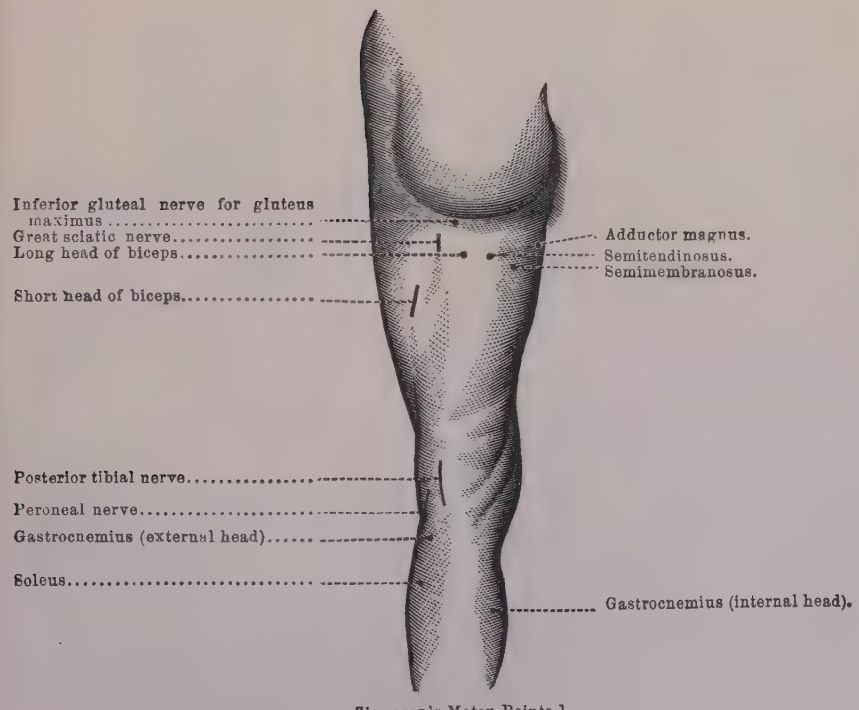
less is the prospect of recovery: if the disease has become stationary for a year or two, there is no chance of any considerable improvement in the condition of the muscles, but the danger to life has become comparatively small.

[Fig. 44.



Ziemssen's Motor Points.

Fig. 45.



Ziemssen's Motor Points.]

THERAPEUTICS. — In projecting the treatment of a case of Wasting Palsy, the first necessity is to ascertain, as accurately as possible, the etiological circumstances under which the disease has originated. The removal of these—supposing them to be still in operation—follows as a matter of course. Mercury and iodide of potassium have been employed with success in cases where the disease depended on a syphilitic taint. If the disease has arisen from overtasking any set of muscles, these must be allowed to remain at rest.

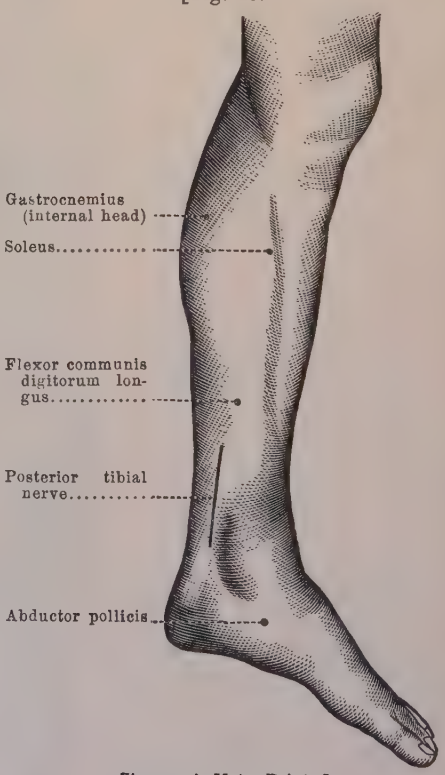
The direct treatment embraces the employment of hygienic means—baths, methodical exercise, change of air, &c.—and the employment of galvanism and frictions to the affected muscles. Remak strongly advocates the use of the constant galvanic current applied to the spinal cord—especially the cervical portion.

Thermal and sulphur baths have been highly recommended by a number of writers. Wetzlar has especially called attention to the beneficial effects of the waters of Aix-la-Chapelle. Cold baths are objectionable.

The most effective remedy in Wasting Palsy is, undoubtedly, galvanism. Numerous observations attest its value when applied locally to the affected muscles. After a very considerable experience of its employment, I am convinced that it very rarely fails of some good effect when perseveringly applied. This effect is too often temporary: too often also it is found difficult to keep up the treatment with the requisite regularity for a sufficient length of time. In some cases marked improvement in the power and bulk of the muscular masses was witnessed; in others, the disease, previously progressive, was brought to a standstill. In the case of a man, still under observation, suffering from atrophy of the muscles of the thighs and upper arms, and of the erectores spinæ, which had been steadily progressive for twelve months, the daily application of the secondary current arrested the disease completely. The arrest has now continued for more than six years.

Duchenne gives the following directions for the employment of galvanism: "Every muscle ought to be faradized in a special manner, according as it has suffered more or less in its electric contractility and nutrition. Thus the more a muscle is atrophied and its contractility diminished, the longer it should be subjected to the stimulation, the more intense should be the current, and the more rapid its intermissions. And this strong current and quick intermissions are the more necessary, according as the sensibility of the muscle is more benumbed. But when the sensibility is seen to return, it is prudent to diminish the intermissions and abate the intensity of the current, and even to abridge the number of sittings, lest there be provoked unmanageable neuralgia, and, which sometimes has arisen, inflammatory accidents. During the faradic treatment, I have excited the muscular sensibility, as much as possible, by rapid intermissions, inasmuch as I have found this the most effective means of reacting on the nutrition of the atrophied muscles. Sittings of too long duration fatigue and even exhaust the muscles, just as forced exercise induces atrophy, instead, like moderate exercise, of favoring nutrition. I believe that no sitting should be protracted beyond fifteen minutes, at the most. I rarely give more than one minute to each muscle. To prevent weariness, and a bruised feeling, that sometimes follows the application of electricity, I pass rapidly over the muscles, taking care to return to each of them several times during the same sitting, so as to leave a short interval of repose between each excitation."[1]

[Fig. 46.



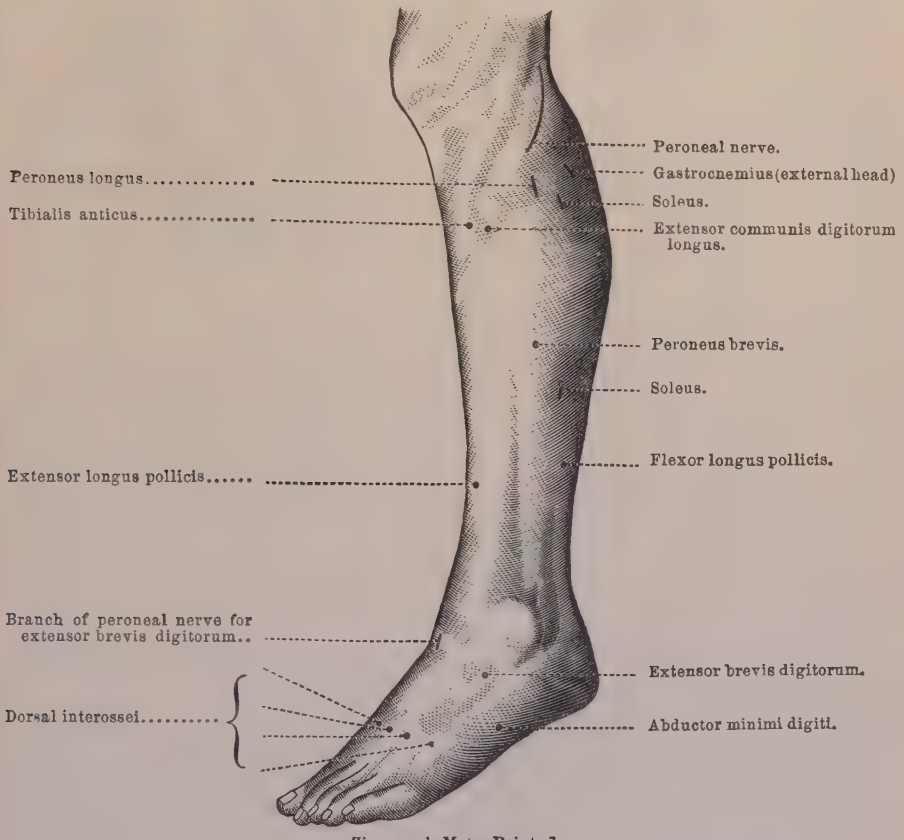
Ziemssen's Motor Points.]

The secondary symptoms—cramps and neuralgic pains—are most effectually subdued by warm baths, temporary rest in

[1] De l'Electrisation localisée, p. 702.

bed, and anodynes. The hypodermic injection of morphia has, in my hands, been followed by the happiest effects in relieving the excruciating neuralgia which is not unfrequently associated with this disease. One of my patients, thus afflicted, is in the habit of having half a grain of morphia injected early in the morning, when the pains are severe. Such an injection enables him to pursue his employment through the day in comfort—a result which he fails to attain by any dose of the same remedy internally administered.

[Fig. 47.



Ziemssen's Motor Points.]

[*Pseudo-hypertrophic Muscular Paralysis*, after having been described by Costa and Gioga in Italy (1838), and Meryon in England (1852), was, in 1868, more definitely studied and classified by Duchenne. Many pathologists, in America as well as in Europe, have, since that time, reported cases.

It occurs in children, up to the age of 13 or 14, mostly in boys. No causes have as yet been made out; but its occurrence in several children of the same family, or nearly related to each other, has been established, especially by Poore.[1] In thirty-seven instances, there were two or more of the same family thus affected. Hereditary transmission of the tendency to it appears to have been observed only on the mother's side; that is to say, when the parents of children having it were not subject to it, examples have been found in the mother's family; her brothers, &c.

The first well-marked symptom is muscular weakness of the lower limbs and back. This gradually increases. The child stands with the legs far apart, the shoulders thrown backward, and the belly forward. The toes point downwards, sometimes approximating *talipes equinus*. In the course of a few months some of the muscles of the lower limbs will be found to be enlarged; and this gradually extends to those of the trunk and upper extremities. Sometimes those of the face also are involved.

The enlargement of the muscles of the calves of the legs, in many instances, gives an appearance like that of extraordinary

[1 N. Y. Medical Journal, June, 1875.]

strength; and yet the limbs are almost powerless for locomotion. In from one to three years, paralysis extends to nearly all the muscles of the body. An impairment of the mental faculties has been

Fig. 48.

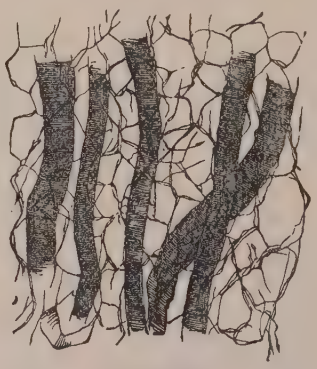
Early stage of Pseudo-hypertrophic Paralysis. (Bristowe.)

noticed in a number of cases. In this condition, unable to rise from the position in which he is placed, the child may live for several years more; seldom to adult age.

Sensation is not involved in this disease; neither are the functions of digestion, respiration, or circulation. At first, the electro-contractility of the muscles is unchanged; at a late stage, it is gradually lost.

Griesinger and Billroth, in 1865, first ascertained the presence of deposits of fat in the enlarged muscles of this affection. Duchenne and others have observed also the great increase of connective tissue, along with atrophy of the muscular fibres. It is altogether a degenerative, not a hypertrophic alteration that the muscles undergo.

Fig. 49.



Pseudo-hypertrophic Muscle. (Charcot.)

Several facts in the history of this disorder point, in its pathology, to a nervous origin. 1. It is symmetrical, and progressive upon both sides of the body alike. 2. The intelligence is not unfrequently impaired. 3. It presents an analogy (notwithstanding that shrinking occurs in the one, and increase of bulk in the other) to progressive muscular atrophy; in which, as above seen, lesions of the spinal cord, and sometimes of the sympathetic ganglia, have been demonstrated. Yet no satisfactory demonstration has been obtained of the association of Pseudo-hypertrophic Muscular Paralysis with morbid changes of the nervous system.

The only treatment so far suggested as affording much prospect of advantage, is the use of electricity. Duchenne advised local faradization; under which he reports two recoveries. McLane Hamilton[1] recommends massage; of which Bristowe[2] also expresses approval.—H.]

[1 Nervous Diseases, &c., Philada., 1878; p. 275.]

[2 Manual of the Practice of Medicine, American Edition, p. 1052.]

METALLIC TREMOR. TREMBLEMENT MÉTALLIQUE.

By William Rutherford Sanders, M.D., F.R.C.P.

Synonyms.—Tremor Metallurgorum; Paralysis Agitans Metallica; Rheumatismus Metallicus (Schönlein); Metallic Shaking Palsy; The Trembles.

1st. Mercurial Palsy or Tremor; Mercurial Shaking Palsy; Mercurial Trade Disease; Tremor ab Hydrargyro; Paralysis Agitans Mercurialis; Tremblement Mercuriel; Tremblement des Doreurs; Mercurial-Zittern.

2d. Lead Tremor or Shaking Palsy; Tremor Saturninus; Paralysis Agitans Saturnina; Saturnines Zittern.

Definition.—Metallic Tremor is a species of paralysis agitans, caused by the slow poisonous action of certain metals, particularly mercury and lead. It consists of spasmodic tremors with diminished muscular power, occurring in various parts of the body.

1. *The Mercurial Tremor or Shaking Palsy*, being the form best known and most important, will be first described.

Causes.—*Exciting.*—The chief source of this disease is the inhalation of mercury in a state of vapor, this metal being volatile at nearly ordinary temperatures (68° to 70° Fahr.). By some authors this has been regarded as the only mode of origin; but it is certain that the introduction of mercury by the skin, either in consequence of manipulating the metal, or of prolonged friction with mercurial ointment, has sometimes brought on the peculiar tremors; and the same effect has also resulted, in a few instances, by absorption, from the intestinal canal, of mercurial preparations administered medicinally. The principal sufferers from the disease are accordingly:—1st. The workmen employed in the quicksilver mines, especially when fire is used in the reduction of the ores. 2d. Water-gilders (who plate with gold dissolved in mercury), looking-glass silverers, barometer makers, workmen in chemical manufactories, where mercurial preparations are made, button and toy gilders, furriers, and others whose business exposes them to contact with mercury.[1] 3d. Persons using mercury medicinally. In former times, the Iatraliptæ, an inferior class of surgeons, who practised as mercurial anointers or rubbers, without protecting their hands, were frequently subject to tremors which sometimes proved incurable. A similar instance is recorded recently. Dixon, the anatomy porter of the Irish College of Surgeons, "who at one time rubbed in immense quantities of mercury for the cure of venereal among the *Mohawks*, or swells of the day," was subject for thirty years to mercurial stammering (psellismus mercurialis).[1] Syphilitic patients, after long courses of mercurial treatment, especially by friction, often suffered severely from the trembles.[2] On the other hand, the internal use of mercurial medicines alone very rarely gives rise to the tremors; nevertheless undoubted examples of this kind have been observed even in recent times, both in venereal and in other cases.[3] In the present day, there is little risk of tremors originating from excess in either the external or internal medicinal use of the mineral, but the possibility must not be overlooked. 4th. Persons are sometimes accidentally exposed. In 1810, the *Triumph*, man-of-war, took on board a cargo of mercury, saved from a wreck. In consequence of the bladders bursting, in which it was held, the mercury spread through the ship, and in the space of three weeks "two hundred men were affected with ptyalism, ulceration of the mouth, partial paralysis in many instances, and bowel complaints."[4] In 1803, a fire broke out in the quicksilver mine at Idria, near Trieste, and about

[1] Ramazzini, De Morb. Artif. caps. i.—iii. 1717; Patissier, 1822; Thacrah, 1832; Darwall in Forbes' Cyc. Pract. Med. i. 151, 1833; Tardieu, Dict. d'Hygiène, 1852; Whitley in Sixth Report of Med. Officer of Privy Council, 1863, p. 358.

[1] Mapother, Mercurial Trade Disease, Med. Press and Circular, i. 531, May 23, 1866.

[2] Hutten, De Morb. Gall.; Fernelius de Luis, Ven. Cur. c. vii. p. 234, 1656; Ramazzini, l. c.

[3] Colson, Arch. Gén. de Méd. xv. 338, 1827; Lancet, ii. 1838–9, p. 767.

[4] Burnett, Phil. Trans. 1823, Pt. ii. 402. The *Phipps*, schooner, which assisted, was similarly affected: Ed. Med. and Surg. Jour. vi. p. 513, 1813.

nine hundred persons in the neighborhood were attacked with nervous tremblings.[1] Medico-legal questions have also risen as to the alleged deleterious effects of emanations from workshops where mercury was used.[2]

In a few instances a single strong exposure has been known to cause the tremors;[3] but usually a prolonged and habitual contact, for months or years, is required, under conditions which favor the development of the disease.

Predisposing Causes. — The circumstances which dispose to the disease or aggravate it, are:—1st. Bad ventilation; 2d. Cold and damp weather (hence the tremors are worse in winter, in consequence of the low temperature and close confinement); 3d. Defective cleanliness; 4th. Intemperance; 5th. Violent emotions (a fit of passion has sometimes originated an attack of tremors suddenly); 6th. Idiosyncrasies must be taken into account. Certain constitutions are more susceptible than others to the mercurial poison. The same exposure which in some individuals affects the mouth, producing salivation and ulcerations of the gums, without tremors, will, in others, cause tremors without salivation. 7th. The mode of application has considerable influence. As a rule, inhalation of mercurial vapor is followed by tremors; inunction or internal medicinal use, by salivation.

Description.—Previously noticed by several writers, and especially by De Haen,[4] the tremulous mercurial disease has been most fully described by Mérat,[5] as observed among the water-gilders of Paris. Less complete accounts have been given by various authors of the disease among workmen in other countries, and as it affects the quicksilver miners at Almaden and Idria.[6]

[1] Murray's Handbook to S. Germany, 9th edit. 1863, p. 400.

[2] Chevallier, Annal. d'Hygiène, xxv. 388, 1848; Orfila, Toxicologie, 4th edit. 1843, i. p. 593.

[3] Christison on Poisons, Merc. Tremor, 4th edit. p. 418, 1845.

[4] De Haen, Ratio Medendi, Pt. iii. c. 28, 1761.

[5] Mérat, Mém. sur le Tremblement des Doreurs, &c.; Appendix to the Traité de la Colique Métallique, Paris, 1812; also in Dict. des Sc. Méd. xxx. 232, 1818, and lv. 521, 1821; Bateman, Ed. Med. and Surg. Jour. viii. 376, 1812; Mitchell, Lond. Med. and Phys. Jour. 1831, p. 394; Bright, Med. Rep. ii. 495, 1831; Stokes, Ryan's Lond. Med. and Phys. Jour. v. 519, 1834; Lancet, ii. 1853, pp. 231 and 317; Med. Times, ii. p. 578, 1853; Marshall Hall, Watson, Romberg, Valleix, Falck in Virch. Handb. der Spec. Path. u. Ther. I. iii. 136, &c.

[6] Jussieu, Mém. de l'Acad. Roy. des Sciences de l'Année 1719, p. 357, &c.

Symptoms. — *1st Stage. Simple Tremors.*—The commencement is sometimes sudden, but most frequently the disease comes on gradually. The upper extremities are nearly always first affected. The patient finds his hands and arms getting weak, unsteady, and less under control; they vacillate and tremble whenever they are used. He can do coarse work, but nothing requiring precision. The attempt to seize or hold anything increases the trembling. At the same time numbness or formication is sometimes felt in the hands or feet, and occasionally pains in the joints, particularly the thumbs, elbows, knees, or feet. These simple tremors are very common among quicksilver miners and water-gilders. They are not so severe as to prevent work altogether, and by judicious means they may be kept from increasing.

2d Stage. Convulsive Tremors.—If the patient continues or increases the exposure, or becomes more susceptible to it, the trembling augments in intensity till it becomes convulsive or spasmodic in character. Muscular subsultus occasions vibration and jerking of the hands and arms. The tremor is easily excited either by exertion or emotion, and once begun cannot be stopped for some time. The voluntary acts also become spasmodic as well as tremulous, and are accomplished by interrupted violent starts, like the movements in chorea. In bending the arm, for example, the flexion cannot be done by a single continuous contraction, but takes place by two or three jerks. The tremulous hand cannot be directed with precision, but is projected beyond or beside or away from the object; it soon becomes unfitted for work, and can scarcely convey food or liquids to the mouth. As Dr. Pope tells of a miner at Friuli, "he could not with both his hands carry a glass half full of wine to his mouth without spilling it, though he loved it too well to throw it away."[1] The convulsive nature of the movements depends greatly on the predominance of the flexors over the extensors; so that when a patient has seized an object, he often cannot let go his grasp. At this period, the patient is usually obliged to discontinue work, and after an interval of rest, steadiness may still be completely restored. But if he persist, or resume his employment too soon, the tremors become greatly aggravated, and extend by degrees over the whole body. The legs begin to shake, especially at the knees, and in walking they tremble and dance as if hung upon wires. The lips, tongue, and jaws are in tremulous vibration, and speech is hurried, staccato, and stammering, becoming at last unintelligible (psellismus mercurialis). The head oscillates, shaking, or

[1] Phil. Trans. i. p. 21, 1665.

nodding; and sometimes the features are distorted by spasmodic grimaces; the eyeballs alone are unaffected in their movements. Mastication is impeded. Finally, the tremulous subsultus appears in the muscles of the trunk, and the respiratory movements are convulsive and attended with dyspnœa. Tonic spasms also occur in the affected parts, and are frequently attended by pains, to which the Spanish miners of Almaden give the name of "calambres," *i. e.*, cramps. These pains are sharp and lancinating, and sometimes of intolerable intensity; they are not always in proportion to the muscular contraction.[1]

When the tremors attain their greatest intensity, they amount to a kind of convulsion, and the patient presents a most pitiable aspect. In constant tremulous commotion, tottering, trembling, shaking, and stuttering, he is powerless to execute any combined movement; he cannot walk, or speak, or chew; he dares not touch any object for fear of breaking it or letting it fall; on raising his agitated hand, with food to his mouth, he misses his aim and inflicts involuntary blows on his face. He must be fed and clothed like a child.[2] Some unfortunates deprived of assistance, have been known to creep on all-fours, and seize food with the lips, like the lower animals. Unless in the very worst cases, however, whenever the body is supported, sitting or reclining, the tremors gradually subside, and soon cease altogether, and they do not return until excited in consequence of some voluntary movement or mental emotion. During sleep, they remain in entire abeyance. The patient is thus allowed time for repose and recovery. But in the most advanced cases, the subsultus takes place even when the body is reposing, so that the involuntary shaking of the head on the pillow has prevented sleep.[3] In the tremulous parts, the muscular strength is diminished (paresis), but there is no interruption to the conduction of the stimulus of volition (paralysis). The sensibility is not impaired.

Concomitant Symptoms.—The condition of the other functions, accompanying the disorders of the nervous system just described, indicates the presence of the mercurial cachexia. At the beginning this is slight and unimportant. The skin exhibits a sallow, brown, or earthy tint; it is dry and sometimes rather warm; the expression is sometimes animated, at other times languid; there is little or no emaciation, which indeed does not appear till the disease is of long standing. The digestive functions are unimpaired; there is no colic, the abdomen is soft and of ordinary volume, and the urinary and alvine excretions are natural. But as the tremors become more severe, the appetite diminishes, and it ultimately ceases altogether; the tongue becomes white and pasty, but without bad taste, and gas accumulates in the intestines. The respiratory organs are natural, till dyspnœa and asthma arise, from the respiratory nerves being involved in the tremors. The pulse is usually at first strong and slow, as in metallic colic, but it may afterwards become small and weak; sometimes it is accelerated.

But sooner or later signs of general mercurialism usually make their appearance, especially salivation, loose teeth, inflamed and ulcerated gums, aphthæ, fetid breath and sweat, swelling of the parotids, and a pustular eruption over the body. These symptoms occur early, and are particularly obstinate in workmen who take their food in the workshops or mines, and who are not careful to use ablutions, and change their clothes and shoes. Attacks of excited circulation (erethism) are frequent in the early stages; and in the later, anæmia, emaciation, and great debility. If we except the cachectic symptoms just described, complications are rare in the course of mercurial tremors. The colic, which is sometimes observed, depends upon lead which is mixed with the mercury, or has been used along with it.

3d Stage. Mercurial Tremors, with Affection of the Brain.—The tremors are not of themselves dangerous to life, but in the advanced stage they are often accompanied by serious cerebral disorders, as headache, loss of memory, loss of consciousness, sleeplessness, delirium, epilepsy. These symptoms would soon end fatally were it not that generally their gravity compels the sufferer to desist from his employment; and by this fortunate interruption, recovery usually takes place even from this dangerous condition. Indeed, the disease when subjected to treatment is rarely fatal.[1] Some inveterate cases prove incurable, or are succeeded by

[1] Tardieu, Dict. d'Hygiene, ii. 481, 1854, who quotes Roussel, Lettres Méd. sur l'Espagne, Union Méd. for 1848–9; Ed. Monthly Med. Jour. Retrospect for 1848, p. 254.

[2] De Haen's description is graphic. Case 2. "Deaurator, 25 annorum, horrendo artuum omnium, maximè superiorum . . . vexatus . . . ita ut nihil laboris ultra perficere, ut nec comedere, bibereve solus, nec loquens amplius intelligi potuerit. Nutriendus, vestiendus et infantis instar, alvum urinamque posituras, adjuvandus erat; dolorum cæterum immunis." It is satisfactory to add, "Virtute electreâ trium septimanarum spatio adhibitâ, perfectissimè convaluit, ita ut ipsi, sive in motu, sive in loquelâ, ne vel minimum quidem desit." (Loc. cit.)

[3] De Haen, loc. cit. case 7.

[1] See fatal case from "general failing of the vital powers." Lancet, 1839–40, ii. p. 588, and Guy's Hosp. Rep., 1864, p. 175.

motor paralysis, but it is only in those instances where the noxious exposure is obstinately persisted in, notwithstanding repeated attacks of increasing severity, that death finally takes place, accompanied by symptoms of profound mercurial cachexia, and especially extreme marasmus and exhaustion.

But although mercurial tremor by itself is not directly fatal, and is a curable disease when submitted to proper treatment, yet, under the circumstances in which workmen were placed till within a recent date, the effects of the mercurial poison, taken as a whole, were most disastrous, and the mortality in certain employments was excessive. In Paris, in 1821, it is stated that the looking-glass manufacturers could not remain at the trade above eight or twelve years. When necessity compelled them to persevere too long, their faces became pale, with an expression of intoxication, their intelligence and memory gradually failed, they fell into a kind of idiotcy, and after lingering in this state for some years, they died of consumption, or were struck with apoplexy.[1] In 1847, Dr. Sanderet reports that the trade of water-gilding at Besançon, where it was extensively carried on, was most injurious to health,[2] the mortality among the workmen being enormous, and due chiefly to phthisis. Fortunately these trades, conducted under better hygienic conditions, or by means of new processes, are either entirely innocuous or are much less injurious at the present day.

The condition of the quicksilver mines was, in ancient times, most dangerous.[3] At a comparatively recent date, when Jussieu visited Almaden, in 1719, he found that the free miners, who adopted proper precautions, preserved their health and lived like other men, but the convicts and slaves who took no care suffered severely, and fell victims to disease. In 1848–9, there were no slaves nor convicts in those mines; but it was observed that the native miners, who knew the risks and avoided them, were little affected, while the poor laborers from a distance, careless and dissipated, experienced the most disastrous effects. The average number of workmen was 3911; of these forty-eight were "calambristes" (in the second stage of mercurial tremors), half of which number died within the year, and the other half remained unfit for work in the mines. Besides this, there were two deaths from accidents, three mutilations, and thirty-nine injuries more or less serious. And although many workmen do not fall victims to the mercurial poison, none of them entirely escape its action.[1] At Idria, although the hygienic conditions of the place are in other respects highly favorable, it is stated that the whole population is subjected to the influence of mercury, not the workmen at the mines only. The annual mortality is 120 out of 4500 inhabitants. The workmen exposed directly to the action of the metal suffer severely. In 1856, 122 out of 516 were seriously affected.[2] Information is wanting in regard to the quicksilver miners in California and Australia.

Course and Prognosis.—Mercurial Tremor is essentially a chronic and protracted disease. It runs a uniform course. Once begun, if the exposure is persevered in, the symptoms gradually get worse; the tremors become more intense, are accompanied by spasms clonic and tonic, and spread over the whole body. But if the patient be removed from the exciting cause, exposed to fresh air, and placed under suitable treatment, amelioration soon begins, and, after a few weeks or months, perfect steadiness may be restored. The prognosis, therefore, is generally favorable, provided the patient can avoid the contact with mercury. The prospect of cure, and the time required for it, will depend on the severity of the symptoms, and especially on their duration previous to treatment, on the age of the patient, the presence of serious cerebral symptoms, and the degree of mercurial cachexia which may accompany the tremors. The affection is most frequent probably in middle life (thirty to forty); it is more severe in old people. If taken at an early stage, twenty days may effect a cure, but in a confirmed case usually from two to seven months, sometimes a year or more, are required. When the tremors are spasmodic and generalized, the cure is tedious and imperfect, some tremor of the hands nearly always remaining permanently. The upper extremities, which are the parts earliest and most severely affected, resist cure the longest. It is seldom that the tremors are persistent and irremediable, and, as already stated, fatal results only ensue in consequence of general cachexia or phthi-

[1] Burdin, Art. Tain, Dict. des Sc. Méd. 1821, liv. 276.

[2] "Une des industries des plus fatales à la santé." (Annal. d'Hygiène, 1847, xxxviii. 457.)

[3] At Idria, in 1665, Dr. Pope says, "All of the miners in time (some later, some sooner) become *paralytick* and dye *hectick*." (Phil. Trans.) Also Dr. Edward Brown, in Phil. Trans. Dec. 13, 1669.

[1] Tardieu, loc. cit. and Roussel. In the inclosure called Brutrones, where the furnaces are situated, the animals which are allowed to graze there are liable to Mercurial Tremors. (Edin. Month. Med. Jour., Retrospect for 1848, p. 255.)

[2] Med. Times and Gazette, xxxix. p. 616, 1859, and Gaz. Hebd.

sis or apoplexy, the effects of an unhealthy constitution, or of unpardonable neglect, or of obstinate persistence in exposure to the poison.

After a first seizure relapses are frequent, and usually of increasing severity. If the patient, in spite of due care, is still subject to attacks, he ought to change his employment. Some constitutions, peculiarly sensitive to the poison, are unfit for any trade requiring the use of mercury.

DIAGNOSIS.—The symptoms and the cause distinguish Mercurial Tremor readily from other diseases. It could only be confounded, 1st, with chorea, or St. Vitus's dance, which it resembles in the jerking nature of the movements, but it differs by the presence of tremors; 2d, with idiopathic paralysis agitans, with which it is identical as regards the character of the irregular movements (viz. tremors and jerking), but it is distinguished from it by the exciting cause (mercury), and by the concomitant symptoms of mercurial poisoning. In addition, the speech (tongue and jaws) is much sooner, more invariably, and more characteristically affected in the mercurial disease than in idiopathic shaking palsy. The loss of memory and consciousness, and other cerebral symptoms also, are peculiar to the mercurial disease. On the other hand, the irresistible tendency to walk or run forwards, which marks the paralysis agitans festinans, is not met with in the Mercurial Tremors; the only disturbance of equilibrium in the latter is that which results from debility, tremors, and spasmodic jactitation.[1] 3d. It is not likely, with ordinary care, to be mistaken for delirium tremens, or alcoholism.

PATHOLOGY AND MORBID ANATOMY.—The disease being rarely fatal, the information in regard to the morbid anatomy is scanty. In a recent case,[2] Dr. Alfred Taylor found the brain and spinal cord, the muscles, lungs, heart, liver, and kidneys, in appearance quite healthy. On chemical examination of the brain, liver, and kidney, minute globules of metallic mercury were obtained, in largest proportion from the kidney. The spinal cord and medulla oblongata are doubtless the seat of the principal morbid action, just as in idiopathic paralysis agitans. The molecules of mercury entering probably into combination with the nervous substance, seem both to irritate and partially to paralyze the nervous centres of motion, while they leave the apparatus of sensation intact.[1] The opinion which some authors entertain, that the morbid lesion has its seat in the muscles and not in the nerve-centres, is insufficient to account for the spasmodic and variable nature of the phenomena, and is inconsistent with the cerebral symptoms which ultimately become developed.

The *treatment* is twofold, preventive and curative.

Prevention is accomplished, 1st, by limiting the exposure to a short period at considerable intervals, or by adopting various contrivances which remove the mercury from contact with the operator. In the time of Pliny[2] the workmen protected their faces with masks of loose bladder skin, sufficiently transparent to admit of being seen through. Masks of glass were afterwards substituted. Sponges over the mouth, and various kinds of respirators, have also been proposed. But the chief improvement has taken place in recent times by the better construction and ventilation of the workshops, and by the introduction of flues and chimneys, which carry off the mineral vapors by a powerful draught out of the apartment, while the workman is further protected by a glass sash interposed between his face and the stove where the mercurial vapors are disengaged. To D'Arcet's[3] draught chimney for this purpose the French Academy of Sciences, in 1816, awarded the prize founded by M. Ravrio, who had made his fortune as a manufacturer of gilt bronzes, and was anxious to obtain some means for protecting workmen from the risks of the employment. Mérat bears testimony to the efficacy of D'Arcet's chimney in warding off the tremors, to some extent. Similar flues and stoves have been used in this country;[4] but none of these inventions have proved successful in entirely preventing the disease. 2d. On the part of the workmen, regular habits, personal cleanliness, change of clothes, frequent ablutions, and the practice of never eating in the mine or workshop, or with unwashed hands, are essential. Intemperance invariably predisposes to or aggravates the disease. Melsens observed that workmen

[1] De Haen's fourth case presented considerable disturbance of the balance in locomotion. "Adeo difficulter ingreditur, ut sæpius humi concidat, dumque corpus pronando sustinere se nititur, vi quasi supinatur." (Loc. cit. p. 230.)

[2] Guy's Hosp. Rep. 1864, x. 176; Lancet, 1839–40, ii. p. 589.

[1] Jussieu's idea of the pathology is curious. The tremors, he says, are "les tristes effets du séjour du sang dans les vaisseaux du cerveau, dévenus variqueux par le poids de quelques particules mercurielles, qui y ont séjourné." (Loc. cit. p. 360.)

[2] Hist. Nat. xxxiii. 40.

[3] Mémoire sur l'Art de dorer le Bronze; Paris, 1816. Dict. des Sc. Méd. 1818, xxvii. p. 299.

[4] Darwall in Forbes' Cyc. Pract. Med. 1833, i. 157.

who used much salt with their food are less liable to suffer from Mercurial Tremors, and he also recommended the iodide of potassium as a powerful preservative. Of late years in this country Mercurial Tremor has greatly diminished in frequency, and under proper hygienic rules would probably cease altogether, at least in its more aggravated forms. Water-gilding, the most dangerous kind of mercurial trade, has been now almost altogether superseded by electro-plating, which is completely innocuous. Looking-glass silvering, when conducted in large well-ventilated apartments, with means for preventing the diffusion of the metallic dust, is also quite safe, if the workmen are employed only at intervals, and are careful and temperate. But when these conditions are not attended to, and especially if the men are kept too continuously at work, slight tremors soon make their appearance, and severe cases occasionally happen.[1] According to Dr. Whitley's report,[2] the number of persons affected in England and Wales appears to be small, and the cases, for the most part, slight. The same statement may be applied to Scotland and Ireland. The condition of the quicksilver mines is probably still one of considerable danger to health and life. But full and accurate information is wanting in regard to the amount of sickness and mortality among the miners, and the means used for their protection.

The *Curative treatment* consists, first, in complete removal of the patient from his noxious employment; next, in change of dress, ablution, exposure to a free atmosphere of moderate temperature, and the administration of a nutritious tonic diet. The objects of treatment then are, 1st, to eliminate the mercury from the system by the secreting organs. *Sudorifics*[3] have been much used for this purpose: acetate of ammonia, Dover's powder, guaiac, sarsaparilla, sassafras, &c. Sulphur has been regarded by some as specific; warm and vapor baths, or sulphurous baths, &c., are always employed. Neufchâtel had a reputation for the successful treatment of water-gilders' palsy, chiefly by means of vapor baths.[1] *Diuretics* have become favorite remedies, and especially the iodide of potassium, since Melsens[2] brought evidence to show that this salt has the power of redissolving the mercury contained in the tissues and eliminating it by the urine, in which its presence may be detected chemically.[3] The caution must be observed not to give the iodide so largely as to disengage an excess of mercury at once within the body. Various other diuretics, common salt, bitartrate of potash, &c., may be employed. Purgatives are also useful. 2d. Another indication is to soothe and strengthen the nervous system, by means of *antispasmodics*, *narcotics*, *tonics*, and *stimulants*. Steel and quinine, singly or in combination, are especially serviceable. Opium is useful. Nitrate of silver has also been recommended. Stimulants, particularly alcohol, wine, &c., exert a powerful immediate effect in arresting the tremors: hence workmen are apt to resort to them to steady their hands; but when the immediate effect is over, they aggravate the tremors. The most beneficial stimulants are *electricity* and *galvanism*, which have afforded very satisfactory results.[4] Dr. Haen's cases were cured, some of them rapidly, with electricity as the only remedy applied.

2. *Lead Tremors; Tremor Saturninus; Paralysis Agitans Saturnina.*

Mérat denied that any other metal than mercury could give rise to tremors, and, with few exceptions,[5] other writers appear to be of the same opinion. In regard to artisans using lead, this view is probably correct; but the case is different with miners exposed to the vapors of the metal. Brockmann,[6] in particular, from his experience in the Harz Mountains, has described a species of lead tremors, which

[1] See recent cases, Scott Orr in Glasg. Med. Jour. i. 37, May, 1866, and Mapother, loc. cit. Also Taylor, loc. cit.

[2] Sixth Report of Med. Officer of Privy Council for 1863, p. 22; and 1864, p. 358.

[3] Jussieu remarks (1719) that at the mines of Almaden the medical treatment differed from the usual practice then in vogue of purging and bleeding, and consisted simply in exposing the patients to the free air, and administering absorbents, as hartshorn, ivory, or crab's-eyes; and what is singular (he adds) the treatment succeeds almost always in temperate subjects and those who abstain from wine, whilst those who indulge perish without resource. (Loc. cit.)

[1] Sandaret, Ann. d'Hygiène, 1847.

[2] Annal. de Chimie et de Physique, 1845, xxvi. 215, and transl. in Brit. and For. Med.-Chir. Rev. for Jan. 1853, p. 217.

[3] Schneider of Vienna controverts the assertions of Melsens, Ed. Med. and Sur. Jour. 1861–2, p. 394.

[4] De Haen, loc. cit. Gull, Guy's Hosp. Rep. 1853, viii. p. 136.

[5] Percival, Ed. Med. and Surg. Jour. 1813, ix. 62, ascribed tremors rather to lead than to mercury!

[6] Die Metallurgischen Krankheiten der Oberharzes, 1851, p. 282; Schönlein, Allg. und Spec. Path. und Ther. 2 Thele, p. 191 (St. Gallen, 1841); Falck, in Virch. Handb. d. Spec. Path. und Ther. ii. 1 Abth. 517–8, 1855. Wilson, in Edin. Essays Physical and Liter. 2d edit. 1771, p. 517, in describing the disease called *Mile-Reek* among the miners at the Lead hills, mentions that the "extremities tremble and are convulsed." Sauvages, Nosol. Meth. 1768, p. 558, Tremor Metallurgorum.

affects the miners there, and which is almost identical with the mercurial shaking palsy, consisting, like it, of oscillating spasmodic contractions of the muscles, and consequent tremulous motion in various parts of the body. His account includes two forms, the local (partialis) and the general (universalis), both the result of an affection of the nervous centres by lead.

SYMPTOMS.—1*st. Tremor Saturninus partialis.*—As a rule, the upper extremities are alone affected. The arms and hands are in continual vibration, more or less, greatest when any powerful effort is made, or during emotion. With this there is often associated a peculiar nervous tremor of the lips (musc. orbic. oris) and angle of the mouth (levator anguli oris), like that observed when a shy sensitive person opens his lips. The local tremor usually follows violent and persistent attacks of lead colic, especially in highly nervous subjects, or in those exhausted by previous disease. It disappears mostly in a few days, but is apt to return when the exciting causes are renewed. Under very unfavorable circumstances, however, it may increase and extend into the general form of affection.

2*d. Tremor Saturninus universalis.*—In this the tremors are not confined to the arms, but appear in the legs and muscles of the head and trunk. The patient presents a peculiar and pitiful aspect. When at rest, his back is bent like an old man's, his head is bowed, and the chin falls upon the breast; in walking, the legs are rotated tremulously as in paralysis agitans. In advanced cases the jaws, and indeed all the muscles of the head and body, are the seat of the uncontrollable tremors which characterize the disease.

CAUSES.—The general tremor never results except from deeply-rooted lead-poisoning. It is preceded by repeated outbreaks of the severer forms of the lead disease, and progresses hand in hand with the lead cachexia. All the causes of the cachexia predispose to it, and the tendency is increased by an excitable nervous constitution, together with lax fibre and weak muscular system. Age has no appreciable influence. The saturation of the system with lead is the only exciting cause of the disease.

The COURSE of the general disease is chronic and very protracted. Months or even years elapse before permanent improvement takes place. Often a radical cure is impossible. Frequently, also, the tremor becomes associated with some form of anæsthesia, or ends in complete paralysis.

The PROGNOSIS is consequently almost always unfavorable. Only when the disease is partial, and follows a violent colic, or an acute attack of convulsions, can a speedy favorable issue be anticipated. Under all other circumstances, lead tremor is a most serious affection, and is apt to be the precursor of more sudden and dangerous disorders, such as paralysis or cerebral disease.

PATHOLOGY AND MORBID ANATOMY.—No specific lesion has yet been pointed out in the brain or spinal cord. The affected muscles have been found altered, but this is of subordinate importance. The pathology is doubtless analogous to that of mercurial tremor, and idiopathic paralysis agitans.

The DIAGNOSIS is sufficiently determined by the symptoms of the lead cachexia which accompany the tremors, and by the absence of any source of mercurial poisoning.

The TREATMENT must be directed to neutralize the lead poison, and to strengthen the nervous centres, which are the chief seat of the disease. The energetic employment of *sulphur baths*, cold water *douches* to the spine, and the internal use of *nervine tonics*, nux vomica, or strychnia, valerian, quinine, &c., are the appropriate means. Sudorifics, sarsaparilla, &c., have also been used, and Melsens recommends the iodide of potassium on the same grounds as in the case of mercury. Brockmann states that he never concludes the treatment without the persevering use of baths of aromatic herbs, and the cold plunge bath, as well as the internal administration of chalybeates. In obstinate cases electricity and galvanism must be resorted to, but frequently without much success. The most essential part of the treatment consists in the removal of the patient from his unhealthy employment, and placing him in free pure air, with a nutritious animal diet, and a moderate allowance of good wine and beer.

In poisoning with arsenic, zinc, or bismuth, tremors frequently occur, but they only form part of a general group of symptoms, and do not require particular description in this article.

B.—PARTIAL DISEASES OF THE NERVOUS SYSTEM.

1. Diseases of the Head.

a. Meningeal Diseases:—

Simple Meningitis.
Tubercular Meningitis.
Chronic Hydrocephalus.
Meningeal Hemorrhage.
Adventitious Products.
Congenital Malformations.

b. Cerebral Diseases:—

Congestion of the Brain.
Cerebritis.
Softening.
Adventitious Products.
Cerebral Hemorrhage.
Abscess.

SIMPLE MENINGITIS.

By J. Spence Ramskill, M.D.

Definition.—By Meningitis is generally meant inflammation of the pia mater and arachnoid. Inflammation of the dura mater is described separately. Attempts have been made to separate inflammation of the arachnoid from that of the pia-mater, and some, as Lallemand, Parent-Duchâtelet, and Martinet, have even gone so far as to apply the term Arachnitis to inflammation of the arachnoid, on the ground that the serous membrane was the one chiefly affected. But an analysis of the cases given by the very advocates of that opinion shows most conclusively that the pia mater is in all cases affected, and always bears more marked evidences of inflammation than the arachnoid. There is no symptom which, during life, could help to distinguish between inflammation affecting the pia mater and inflammation involving the arachnoid alone; and as the treatment in either case would be the same, there would be no practical advantage gained by such a distinction. Cerebral fever is a name given by Trousseau to various acute affections of the head in children, amongst which he includes Meningitis. Meningitis may be primary or secondary; uncomplicated or complicated; acute or chronic.

Acute Meningitis. Symptoms.—In some rare cases, certain prodromata precede the invasion of the disease, in the shape of slight but increasing pains of the head, irritability of temper, sleeplessness, and general malaise. But, as a rule, the invasion of the disease is decided, and from the outset its gravity is not to be mistaken. Its course may be divided into three stages:—1st, a period of excitement; 2d, a period of transition; 3d, the stage of collapse. These three stages are not always present, nor are they always distinctly marked. When the disease is very violent, the first stage may rapidly pass into the third, or comatose period. In old and feeble people the first stage may either be absent altogether, or be so little marked as to escape observation. Again, the third stage may be absent, from life being abruptly cut short by violent general convulsions in the second period; and lastly, the first and third stage may coalesce during the transition from one to the other, and may present mixed phenomena of delirium alternating with coma.

Stage First. Period of excitement.—A well-marked rigor, with pallor of the surface and cutis anserina, opens the scene, and is very quickly followed by intense

febrile reaction. In very young children a paroxysm of general convulsions may be the first symptom; in adults, however, convulsions are the exception. The fever is very high; the skin is hot and dry; the pulse frequent, sharp, and hard; the face flushed, particularly about the malar bones: sometimes it is alternately flushed and pale. The eyes are glistening, the conjunctivæ injected; the pupils in this stage are usually contracted; there is photophobia; and, in order to keep the light out, the patient keeps his eyes firmly closed, and resists all attempts at opening them on the part of the practitioner. There is sometimes strabismus on one or both sides, particularly in children. Acoustic dysæsthesia distresses the patient: the least sound, the lightest footstep about the room gives him pain. Cephalalgia of the most acute character sets in from the first. It is referred to the forehead, vertex, temples, or occiput, or to the head generally: pressure on the scalp increases, and movement intensifies it; hence, in order to prevent his head from moving, the patient holds it between his hands. Sensorial impressions of light and sound also exaggerate it. The pain is continuous, but presents also frequent exacerbations, during which the patient, especially if a child, utters a peculiar, loud, piercing cry. The headache may precede the other symptoms for a day or two, or for a few hours only, or it may appear simultaneously with them. It is the most striking symptom of the disease; it is present in nearly every case, but not in all; and Andral has related in the "Clinique Médicale," cases in which it was absent from first to last; and, in one of these, sero-purulent effusion was found in the lateral ventricles after death. The intensity of the pain does not bear any relation to the stage of the inflammation and the nature of its products. Thus, it has been found as severe in cases where a post-mortem examination disclosed mere injection and increased vascularity of the meninges, as in cases of serous or purulent infiltration of the membranes, or when false membranes had time to develop. The extent over which it is felt is not proportionate to that of the inflammation, for it may be felt all over the head, and yet the Meningitis be partial only; on the other hand, it may be exactly limited to one particular spot, and yet the inflammation be general. When partial, it does not always correspond to the exact seat of the inflammation, although when an individual complains of a fixed pain in a spot never varying, the probability is, that the meninges are inflamed at that point. The cephalalgia of Meningitis differs from that of continued fevers in its intensity, and in the fact that the patient does not wait, as in the latter, till asked whether he has any pain in his head, before speaking of it himself, and craving for relief. The character of the pain varies; it is described by some as a heavy weight pressing on their brain; by others, as consisting in violent shooting pains, either continuous or recurring at intervals. Sometimes, again, it is compared to the sensation of an iron band encircling the forehead, or of the head being squeezed in a vise. Vomiting is another well-marked symptom of acute simple Meningitis. It is purely sympathetic, and is unattended with epigastric pain or tenderness on pressure, or with nausea. It recurs very frequently for the first day or two of the disease, and may then cease; but in some cases it persists unto the end, either continuously, or with more or less prolonged intermission. The matters vomited are abundant in quantity, and are mixed with bile. The tongue is natural, generally moist, and occasionally covered with a white creamy fur. Constipation is the rule, and the discharges, when there are any, are dark and offensive.

The intellect is always affected from an early period. The temper is extremely irritable; there is marked somnolence or constant wakefulness, or the one may alternate with the other for several days. Delirium sets in early; it is generally of a wild, fierce character, the patient shouting and vociferating, and tossing himself about. In some cases, on the contrary, the first sign of intellectual disorder is obstinate mutism, the patient burying his head under the bed-clothes and refusing to answer questions. Sometimes the delirium reaches at once its maximum on its first occurrence, but otherwise it is slight at first and gradually becomes more and more marked. Sometimes, again, it is only nocturnal at the outset, and does not become diurnal until after a few days; and again, a patient who has been wildly delirious for a few days may recover his reason before death, although all his other symptoms grow worse.

From the beginning the patient staggers when walking, and his gait resembles that of a man under the influence of drink. When he has taken to his bed, he is extremely restless, and keeps constantly shifting his position. The muscles of his face and limbs, even in this stage, may begin to twitch involuntarily. When convulsions have opened the scene, as they frequently do in young children, they recur in frequent paroxysms one upon the other with scarcely any intermissions. The general sensibility is usually heightened in this stage, although it has sometimes been known to be normal. To sum up, the characteristics of the first stage are high fever, cephalalgia, an occasional sharp piercing cry, vomiting and constipation, general hyperæsthesia, sensorial

and cutaneous, and fierce delirium. This stage may last only a day or two, but generally extends over a week, and sometimes over two weeks. It then merges into the second or transition period.

Stage Second.—The delirium becomes quieter; there is carphology, picking at imaginary flies in the air, or on the bedclothes; and the patient's eyes become dim and lustreless: his pupils dilate, or they oscillate at first before they dilate, and become insensible to the influence of light. Vision is impaired; hearing gets dull. The patient complains less often of his headache, because he is less sensible; occasionally, however, he utters still a loud cry, he grinds his teeth, moving his jaws as if he were chewing, and rolls his head from side to side, boring his pillow with his occiput. Somnolence sets in, to be soon followed by a comatose condition. The pulse is less frequent, and occasionally intermits. The respiration is very irregular; at one time it is very rapid, and the next moment it is slow and suspirious, made up of a long, deep inspiration, followed by a slow and long-sustained expiration. The urine is retained, or there may be stillicidium after retention. The constipation persists; and the abdomen is apt to become retracted, sunken, and almost boat-shaped. The general hyperæsthesia is now replaced by hypæsthesia, which soon makes way for complete anæsthesia. The muscular twitchings increase; there is subsultus tendinum, and actual convulsions often set in. These may be general or partial, usually the latter, and they then attack different parts successively. The muscles most commonly affected are those of the eyeballs, producing strabismus, or rolling upward of the globes; the muscles of the face and lips; and lastly, of the extremities. The tongue itself may be the seat of convulsions. There may be rigidity in one or more limbs, and the head may be bent backwards, or be permanently inclined to one side. Spasm is apt, after a time, to alternate with paralysis, or the latter may be gradually or abruptly established. In some cases the paralysis may come and go: in others, a limb may be paralyzed, whilst its homologue is the seat of violent convulsions. The characteristics of this second stage are generally prostration, convulsive movements, gradually developing coma and total paralysis, motorial and sensory, a thready pulse, and an irregular, suspirious respiration.

Stage Third.—The collapse is now complete, and the coma profound. The pupils are widely dilated, and are insensible to light, the eyes are half open, the face sunk and ghastly, the skin cold and clammy. The sphincters relax, the urine and feces are passed involuntarily, and the pulse becomes frequent again, indeed more so than before; it is small, filiform, and uncountable; the breathing is stertorous, and the patient at last dies in a state of complete coma.

Acute Meningitis in old persons, Dr. Machlachlan asserts, seldom occurs suddenly, and rarely exhibits the acute symptoms of Meningitis which affects persons of middle age, or of a younger period of life. It commences insidiously, and without premonitory rigors; it may exist some days without the most experienced eye detecting it. The pulse is natural, the tongue clean, the bowels regular, and there is little or no vascular excitement, local or general. The temper is peevish and irritable, with more or less confusion of thought, inattention, and forgetfulness. The patient makes strange mistakes, takes possession of another's bed, uses the spittoon instead of the chamber-pot. When addressed, his replies are rational, but there is a peculiarity in his manner and expression of countenance, an apparent slowness of comprehension, and a vacancy of eye. The appetite meanwhile is normal, and there is no feverish reaction. Yet there is great restlessness, unsteady gait, a trembling hand when the patient lifts anything to his mouth. After from twelve to sixty-two hours there comes on slight feverish reaction, expressed by increase of temperature only, and not by flushing of face or increase of pulse. Next, wandering, low delirium, and incessant talking become frequent and characteristic symptoms. Maniacal excitement is uncommon: reverie generally passing into coma constitutes the rule. Headache is not a permanent symptom. The patient moans, but never complains. Unless the patient is pointedly asked about headache, there is never any allusion made to it, or to tinnitus aurium. Dr. Machlachlan takes especial notice of this absence of headache; for even in the most acute pus-forming or false-membrane-forming Meningitis, headache may be entirely absent from the beginning. The eyes are suffused, the pupils slightly contracted, or natural. Knitting of the eyebrows, intolerance of light, acuteness of hearing, and vomiting, are comparatively rare in the aged. The only objective evidence of increased vascular action within the cranium may consist in a hotter scalp than natural, and suffusion and injection of the eyes. There is great thirst usually, the patient will drink freely when liquids are offered to him, but he will seldom ask for drink, whilst he is very apt to refuse all food. In the worst cases, nervous twitchings and convulsions are observed, and these symptoms may be induced when otherwise absent, by raising the patient in bed. When coma is present slight convulsions of the limbs may be present. The general aspect of the

senile Meningitis resembles typhus fever rather than Meningitis. The dryness and brown color of the tongue, the muttering delirium, excessive prostration, injection of conjunctivæ and heat of scalp, equally belong to either affection; but in Meningitis the livid hue of the surface, and the mulberry rash characteristic of typhus, are wanting, whilst the diffused heat of skin of the latter disease is absent, or limited to the forehead and scalp in Meningitis.

Varieties as to Seat.—Meningitis may be partial or general. When partial, it may be limited to a small portion only of one hemisphere, generally the anterior lobe, or to the whole of one hemisphere alone, in that case stopping abruptly at the median fissure; or it may affect the convexity of both hemispheres, or be restricted to the base alone. In some cases it affects the base and the convexity of the hemispheres simultaneously. Meningitis of the base alone is, in the immense majority of cases, of tubercular origin. Parent-Duchâtelet and Martinet regarded very profound coma, not preceded by delirium, as characteristic of this variety of the disease; but Andral has conclusively shown that they were mistaken. As regards the relative frequency of the partial and general form of Meningitis, Parent-Duchâtelet and Martinet say, that in ninety-one out of a hundred and seventeen cases, the inflammation affected the convexity of both hemispheres, and in twenty-six, one hemisphere alone.[1]

A very rare form of Meningitis is that which is confined to the membrane lining the interior of the lateral ventricles, a membrane which is so very fine and delicate, as to be indistinguishable when in a healthy condition, and the existence of which has even been denied by Kölliker. Andral relates five cases of this variety, in one of which the granular condition of the membrane pointed to its tubercular origin, a view which was supported by the presence of tubercle in one lung; whilst in another of these cases, the intra-ventricular effusion occurring in the course of general anasarca and ascites, was probably one of the results of albuminuria. The first case of the series, however, seems to be an instance of genuine inflammation of the membrane lining the lateral ventricles, which cavities were found after death to contain a sero-purulent fluid. The symptoms of this rare form of the disease do not differ from those of Meningitis in other parts. There is the same acute cephalalgia, generally referred to the supra-orbital region, followed by delirium and coma, and in some cases attended with violent agitation, convulsive movements, and tetaniform rigidity of the limbs.

Cerebral complications are sometimes apt to arise in the course of an attack of acute articular rheumatism. Of these, an affection resembling Meningitis seems by far the most frequent. In his work on "Diseases of the Brain," Abercrombie relates a few instances of this variety of the affection in a chapter headed "A dangerous modification of Meningitis, which shows only increased vascularity." In France, where the affection seems to be more common than here, the subject has been very much discussed of late years, and has given rise to a good many predictions. So far back as 1835, however, Sir Thomas Watson had called attention to these head complications, which he ascribed to a disturbance of the cerebral circulation, arising from the cardiac disease, which so often supervenes in the course of acute articular rheumatism, and which he stated to denote an inflammatory condition of the brain or its membranes.

There is no doubt that cases of rheumatic fever have occurred in which, from the character of the symptoms, the existence of Meningitis has been diagnosed, whilst post-mortem examination disclosed no such affection. Several such cases are mentioned by Dr. Fuller, in his work on Rheumatism, pp. 303, 304.

The invasion of the disease is, as a rule, very sudden; mostly appearing in the middle of the night. There may or may not have been some premonition of the coming complication in the shape of a feeling of anxiety, of terror, of strange forebodings of evil, a fear of impending death, expressed by the patient (Bourdon and Vigla); or, for a few hours before the actual attack, there may have supervened sudden mistiness of vision, as in a case related by Trousseau, in his "Clinique Médicale." As a rule, the disease does not occur at the commencement of the rheumatic attack, but is always preceded by pain and swelling of one or more joints. The invasion itself is characterized by great increase of temperature, restlessness and jactitation, extreme loquacity, and wild delirium, soon followed by coma, with intervals of muscular twitchings or slight convulsions. In some cases, the delirium is remarkable for the obstinate taciturnity of the patient. The pulse suddenly becomes very frequent, small, and irregular. The respiration is sometimes hurried, and sometimes slow. In the majority of cases, the swelling of the joints goes down, the redness disappears, and the cerebral symptoms seem to be attributable to a real metastasis. In some cases, however, the articular disease persists undisturbed. As to the pain in the joints, it is no longer complained of; but this is no proof that it has ceased, it

[1] Parent-Duchâtelet et Martinet, De l'Arachnitis, p. 94.

merely shows that the intellect is so affected that the patient is unconscious of pain, or at least no longer takes cognizance of it. One of the most distinctive features of this form of Meningitis is the absence of headache and the absence of vomiting; two symptoms which are so prominent in simple acute Meningitis. In the latter affection, the course of the disease is generally rapid, but its rapidity is much less than that of rheumatic Meningitis. The patient may die in a few hours; more often from two to five days. If he lives beyond that time, the probability is that he will escape; and this prognosis will be all the more certain if the swelling, pain, and redness of the joints again make their appearance. Recovery, however, is rare; but when it does occur, convalescence sets in rapidly. In some cases the affection terminates in insanity, but the rule is, that death takes place; according to Vigla,[1] thirty out of thirty-nine cases terminated fatally.

The causes of this peculiar complication of acute articular rheumatism are very obscure. It is said to occur more frequently in cold weather, in the cold months of the year; and there is no doubt that exposure to cold is often an exciting cause. Vigla's assertion that patients who perspire very profusely, and who show confluent sudamina, are more liable to the disease than others who perspire less and have few sudamina, can be easily explained by the greater liability of the former category of patients to catching cold. The disease has been attributed to the perturbing influence of certain methods of treatment. Repeated bloodletting has, by some, been regarded as the cause of the disease, on account of its weakening the system and causing a preponderance of the amount of fibrine over the other constituents of the blood (Beau and Briquet). Others, on the contrary, have ascribed it to the large doses of quinine which, in France particularly, are administered in acute rheumatism. The well-known influence, they say, of quinine in large doses, in producing tinnitus aurium, giddiness, and deafness, explains how its administration may, in acute rheumatism, render the brain liable to be affected by the rheumatic poison circulating in the blood. This influence of quinine in the causation of rheumatic Meningitis is far from being proved, and Trousseau denies it emphatically. This author cites a case of Dr. Beau, in which the symptoms of Meningitis began to show themselves, after the commencement of the quinine treatment, when small doses only of the drug had been given, while it completely disappeared on continuing the treatment, and giving larger doses of quinine.

According to Trousseau, habitual excess in drinking is the chief cause predisposing to this affection, and also an hereditary tendency to neurotic affections of any kind, and chiefly the various forms of insanity. The post-mortem appearances found in such cases are generally a considerable injection of the membranes of the brain, and in some cases sub-arachnoid effusions of serosity are met with; in very rare ones, pus has been found over the hemispheres, as in three cases by Watson.[1] Trousseau regards the affection as a neurosis, and totally discards the opinion that it is really constituted by an inflammatory condition of the meninges, and it is now well known that in the majority of cases of acute rheumatism terminating by high temperature and head-symptoms, nothing at all resembling Meningitis has been discovered upon post-mortem examination.

Meningitis occurs also in individuals suffering from tertiary syphilis; for just in the same way as nodes and gummy tumors form under the periosteum, in different parts accessible to view, similar deposits are found in the substance of the dura mater. In some cases, the membrane is not inflamed in the vicinity of these growths, but in others the dura mater is thickened, and adherent to the brain, while itself participates in its superficial layer, in the chronic inflammation. The symptoms indicating the presence of such deposits are intense and constant cephalalgia, with nocturnal exacerbations; in some cases with convulsions, obtuseness of the intellectual faculties, and sometimes paralysis. The previous history of the patient, the peculiar sallowness of his complexion, and the presence in many cases of periosteal nodes, either on the head itself or on the bones of the leg, sufficiently attest the nature of the case. In less patent cases, the history of the patient will often decide its character. According to Robin and Lebert, these deposits can be recognized to be of syphilitic origin by the characters which they invariably present, and the following is a description of their microscopical structure as given by Robin.[2]

First.—They consist of an amorphous, transparent, grayish, granular blastema.

Second.—This blastema is traversed in spots by lamellar fibres which lie deeply in it, and are often difficult to see, and which are accompanied with fusiform, fibro-plastic bodies, not numerous on the whole.

[1] Actes de la Société Médicales des Hôpitaux de Paris, 1855, Troisième fascicule.

[1] Principles and Practice of Physic, p. 302, vol. ii. Fourth edition.

[2] In Zambaco, Affections Nerveuses, Syphilitiques, p. 80.

Third.—In the portions that are whitish, the amorphous blastema is scattered over with a certain number of fat granules, and some drops of oil.

Fourth.—The most abundant elements of the tissues are cytoblasts, which make up four-fifths of the whole. They are uniformly distributed in the amorphous blastema, and between the fibres of the lamellar tissue; they are separated from one another by a substance which scarcely equals their own width. From their number, and their mode of distribution, they make the blastema look of a remarkable uniform composition. Free nuclei are found in great abundance. A few cells are also met with, with pale, transparent, finely granular bodies; they are almost spherical in shape, some are ovoid, a few angular: the nuclei have a distinct and generally dark contour. They have no nucleolus.

Fifth.—A few rare embryo-plastic elements.

Sixth.—A few globules of pus.

Treatment consists in the administration of large doses of iodide of potassium, of mercurial baths, or, when pain and sleeplessness are constant, of calomel and opium at night, with salines and iodide of potassium during the day.

Inflammation of the Dura Mater is exceedingly rare, as an idiopathic affection, and generally comes under the cognizance of the surgeon as the result of a blow or a fall on the head. Abercrombie gives one case of spontaneous inflammation of the membrane which had come under his own observation; but even there, the disease had spread to the arachnoid. Inflammation of the dura mater is apt to supervene in cases of chronic otorrhœa, an affection which frequently sets in after scarlatina, and sometimes also after measles and variola. There is at first merely a thick mucopurulent discharge from the ear, with some tenderness about the mastoid process, and this goes on for a long time, when suddenly the patient becomes dull and drowsy, complains of intense pain in the head, he then becomes delirious, and lastly passes into a state of coma. After death, the petrous portion of the temporal bone is found carious and softened, and the dura mater overlying it is seen to be detached, inflamed, and generally bathed in pus.

The same series of phenomena may also occur in cases of chronic disease of the ethmoid bone; or inflammation may spread to the dura mater from the membrane lining the cavity of the orbit in cases of wounds of the eyeball or fractures of the orbit.

The symptoms of the meningeal complication are well marked: there is considerable rigor recurring in paroxysms, followed by high fever, and so marked are the intermissions that the disease simulates an attack of ague. Headache is complained of, and the discharge of matter from the ear does not give ease. Vomiting is often present, together with hyperæsthesia of the retina. There may be slight convulsions, but these are never general, and never so marked and so violent as in cases of pure Meningitis. When there is inflammation of the sinuses in the head, which are formed by duplicatures of the dura mater, secondary abscesses show themselves in distant and various parts of the body, in the joints of the big toe, the knee, the hip, the wrist, &c. The aguish aspect of the symptoms in such cases is extremely marked: there are strong rigors, followed by heat and clammy perspiration.

The treatment should have for its object to prevent, or at least to limit, the extension of the inflammation, by the application of leeches to the mastoid process, as soon as it is found to be tender, and subsequently by the use of blisters. The ear should be carefully syringed with warm water, and the pus allowed a free escape. When there can be no doubt that the meninges are attacked, the treatment to be recommended in Meningitis should at once be adopted. When secondary pyæmic abscesses have formed, the treatment should be of a stimulating character, combining the administration of wine and strong nourishing broths, with the use of bark and acids, quinine and iron, &c.

Instances of recovery have been recorded in cases when head symptoms have pointed to an extension of disease from the internal ear to the dura mater, and Abercrombie has related a case of a young lady who, after the usual symptoms, lay for three or four days in a state of perfect coma. Her medical attendants thought her condition utterly hopeless, and continued to visit her as a matter of form. One day, however, they were agreeably surprised to find her sitting up and free from complaint; a copious discharge of matter had taken place from the ear with immediate relief, and she subsequently perfectly recovered.

Progress, Duration, and Termination of Acute Cerebral Meningitis.—The progress of the disease is always continuous; there may be slight remissions, but never those intermissions which form such a remarkable feature of tubercular Meningitis, during which the apparent improvement is so great as to mislead the inexperienced into the belief that the patient is getting well.

The duration of the complaint is extremely variable. It has been known to be fatal in thirty-six hours (Rilliet and

Barthez), but as a rule, death only follows at about the end of the first week, although it may take place at the end of the second, third, and even fourth week.

The termination of the disease is generally in death: very few cases recover, and only when active treatment has been employed at the very outset. It is doubtful whether any acute cases ever pass into the chronic form.

Pathological Anatomy.—The post-mortem appearances necessarily vary according to the stage of the disease in which death has occurred. Thus, there may be only intense redness from increased vascularity of the membranes, which are also remarkably dry; or, if the disease has lasted a few days, there may be fluid effused on the free surface of the arachnoid, in the interior of its sac, and in the meshes of the pia mater. The effusion may be serous, sero-purulent, or entirely purulent. It is rarely abundant enough to produce a distension and prominence of the anterior fontanelle, although a case is related by Abercrombie[1] in which, "at an early period of the complaint, there was observed a remarkable prominence of the anterior fontanelle; in the second week this increased considerably; and in the third week it was elevated into a distinct circumscribed tumor, which was soft and fluctuating, and pressure upon it occasioned convulsion. It was opened by a small puncture, and discharged at first some purulent matter, afterwards bloody serum. After death the opening which had been made through the fontanelle was found to lead to a deposition of thick flocculent matter mixed with pus, between the dura mater and arachnoid, and covering the surface of the brain to a considerable extent."

When death takes place at the end of the first week, the pus is no longer liquid; its more fluid portion has been absorbed, and false membranes of a bright yellow color are now found, which are not yet adherent to the arachnoid. The pus infiltrates the meshes of the pia-mater, and dips between the convolutions. The concrete pus is found in greatest abundance around the larger bloodvessels, and in the sulci between the convolutions of the upper and lateral portions of the brain. Sometimes, as in a case detailed by Rilliet,[2] one hemisphere may be seen covered with pus or false membranes, whilst the pia-mater on the opposite side is merely infiltrated with serosity. The brain, in uncomplicated Meningitis, is not affected; it is generally of firm consistency, and sometimes even more firm than usual. If death has occurred at an early period of the complaint, between the second and fifth day for instance, the gray and white substances present scarcely any traces of injection. After that time, they may still be perfectly healthy, although in the majority of instances the gray substance is of a somewhat pinkish hue, whilst the cut surface of the white matter shows numerous red points. The whole mass of the brain is always firm, but the peripheral layer of the convolutions may be softened; and when stripped off, the pia-mater which adheres to it carries away some portions of it. The longer the duration of the disease, the greater the risk of this complication, although there may be exceptions to this rule. In very young children, according to Rilliet, the whole mass of the brain is sometimes soft throughout, and he ascribes this softening to œdema of the brain. The lateral ventricles may be found empty, or they may contain transparent serosity, or even pus, and, in rare cases, false membranes. According to Andral (Clinique Médicale, vol. v. p. 140), the presence of serosity should not be regarded as the result of a morbid process, unless the quantity amount to more than *one ounce* of fluid in each lateral ventricle.

Etiology.—A. Predisposing causes:—

First, Age.—According to Guersant (Dictionnaire de Médecine, art. Méningite) simple acute Meningitis may occur in the fœtus in utero, and is pretty frequent in new-born infants. After the age of two up to fourteen, it becomes rare, and yields in frequency to tubercular Meningitis, the two being then in the proportion of two of the former to twelve of the latter. After fourteen, it again increases in frequency, and particularly attacks individuals whose ages range from sixteen to forty-five.

Second, Sex.—It is considerably more frequent in male adults than in women; according to Parent-Duchâtelet and Martinet, in the proportion of three males to one female.

Third.—Those trades or occupations which expose the individual to atmospheric changes seem to predispose to the disease. Thus masons, carpenters, soldiers, &c., seem to be more liable to it than other men.

Fourth.—The sanguine temperament, a short thick neck, hypertrophy of the heart, a very irritable temper, are said also to predispose to the disease, as well as the abuse of alcoholic liquors, excessive grief, and mental work.

B. *Exciting Causes.*—The most common are blows on the head, falls, and concussions, &c.; and more frequently exposure to a hot sun in tropical countries. The

[1] Abercrombie, Diseases of the Brain, p. 57.

[2] Rilliet, De la Méningite franche chez les Enfants. Archives Générales de Médecine, 1846, vol. xii.

sudden disappearance of a chronic eruption about the scalp, *e. g.*, chronic eczema or impetigo, has been known to be followed by acute Meningitis; but this cause is not so frequent as it has been held by some authors.

DIAGNOSIS.—It is extremely difficult to distinguish acute Meningitis from acute Cerebritis—as the two affections so frequently coexist; inflammation of the membranes having a tendency to spread to the substance of the brain, or the reverse obtaining. In simple Cerebritis, however, uncomplicated with Meningitis, the excitement is not so marked, the delirium is not of the same wild, fierce character, the pulse either does not rise above its natural standard, or falls below it, even down to sixty or fifty; it is, besides, irregular and varies considerably in its rate of frequency. There is also *tonic* rigidity of one or more limbs, followed by paralysis, which is permanent. In every case, however, the limitation of the inflammation can at best be merely suspected.

From *Delirium Tremens*, acute Meningitis may be distinguished by the absence of headache in the former affection, the peculiar trembling, the hallucinations and spectral illusions and fears of the patient, the character of the delirium, and the abundant, clammy perspiration. The previous history of the patient, besides, usually tells a long story of inebriation.

Typhoid Fever may be separated from acute Meningitis by the headache being less intense, by the frequency of the pulse, the presence of diarrhœa, the infrequency of vomiting, if at all present, the gurgling in the iliac fossa, and abdominal tenderness, the leaden tint of the countenance, and, after the fifth day of the disease, by the characteristic rose spots.

The points of distinction between the simple and the tubercular forms of Meningitis will be given when treating of the latter affection.

TREATMENT.—The treatment of acute Meningitis is only successful when employed very early in the disease, and carried out with energy. It resolves itself into three great remedial measures: first, blood-letting; second, hard purging; third, application of cold water or ice to the head.

Blood-letting.—The patient is to be bled in the sitting posture, from a large opening in a vein in the arm, and continued until syncope is induced. The bleeding is to be repeated as often as the symptoms require it, or to be followed by the application of leeches behind the ears and to the temples. Continental practitioners often prefer bleeding from the dorsal vein of the foot to opening a vein at the bend of the elbow. According to Guersant, in very irritable individuals who are very sensitive to pain, especially in very young children, the application of leeches to the head increases the restlessness and the headache, and he therefore recommends that the leeches should in such cases be applied round the anus or about the ankles.

When it is not considered advisable to repeat the bleeding, compression of the common carotids in the neck, as originally suggested by Dr. Blaud (of Beaucaire),[1] might be had recourse to, so as to cut off for a time the supply of blood to the head.

The application of leeches to the interior of the nostrils, or scarifying the membrane with a lancet, is a favorite practice with some of the German physicians, and must directly relieve the circulation in the head on account of the inosculation between the vessels which ramify in the pituitary membrane and those at the base of the brain.

[My experience very positively confirms the above recommendation of venesection in the early treatment of simple meningitis. The now common opposition to it proceeds upon theoretic grounds, from practitioners who have never made trial of it. I have not, however, found difficulty in the use of leeches, instead of or after general bleeding. They may be applied either to the temples or the back of the neck; in children, most conveniently the latter. When much restlessness exists, cut cups have the advantage of requiring less time than leeches in their application.—H.]

Purgatives.—Active purging possesses considerable efficacy in the early stage of the disease, and materially aids bleeding in producing its full effects. Calomel, jalap, and scammony are the purgatives usually selected, but croton oil seems by far the best, from the ease with which it may be administered, even to children, and the certainty and rapidity of its action.

Mercury.—Apart from its purgative effects, it is a most valuable remedy in Meningitis. It should be administered in small and frequently repeated doses, so as to bring the system under its influence quickly; and this is best effected by combining, with its internal administration, the use of mercurial inunctions in the groin and axilla.

The application to the head of cold, in its various forms, should never be neglected. There is no remedy so effectual in lowering the heat of the head, calming the headache, and subduing the violence of the delirium. Simple compresses, kept

[1] Bibliothèque Médicale, vol. lxii. See also Valleix, Guide du Médecin practicien, vol. ii. p. 49.

wet with cold water, are the least useful form of using cold, as they are soon heated and become dry, and the alternation of heat and cold thus produced might be injurious by attracting more blood to the head. A bladder, containing pounded ice, or a mixture of common salt and ice, is an excellent mode of applying cold, because of the facility with which it adapts itself to the shape of the head. The most effectual method, however, is *irrigation*, i. e. allowing a small stream of water to run on the head from a small vessel placed above it. The effect of this is almost magical, but it should be used with great caution, particularly in children and aged persons, so that its sedative influence might not be too powerful. Previous to using any applications on the head the hair should be cut close or shaved, and this simple measure is sometimes attended with great relief to the patient. Simultaneously with these applications revulsives should also be employed at the opposite extremity of the body by wrapping up the calves of the legs in mustard poultices, or in blankets wrung out of hot water and sprinkled with turpentine.

When the disease has passed into the third stage, that of coma, the above treatment is no longer admissible. Blisters applied to the nape of the neck and behind the ears, are exceedingly useful; and if the coma be very profound, a cap of blistering ointment applied over the whole skull has been known to rouse the patient. Flying blisters, applied in rapid succession to the inner aspect of the thighs, the calves of the legs, or mustard poultices even, are then useful also to rouse the system. When collapse has set in, mercury and purgatives should, of course, be discontinued, and stimulants, ammonia, and bark had recourse to. The bladder should be frequently examined to prevent the accumulation of urine and its consequent evils.

[The disparagement of blisters by some late authors induces me to add confirmation, from my own experience, of their value, after the early stage of high febrile excitement has passed, the other symptoms not having given way. I have seen several recoveries under apparently the most unpromising circumstances, when simple meningitis was actively treated, with bleeding, purgation, cold to the head, and vesication, either of the back of the neck or over the whole shaven scalp.—H.]

Diet.—The diet, in the first stages, should be low; no solid food is to be given.

In the third stage, however, strong broths given in small quantities repeatedly and wine become essential.

The room in which the patient lies should be kept cool and dark, and well ventilated, and free from the slightest noise.

Sometimes after the acute stage of the disease has passed, and convalescence has begun, the delirium is apt to return.

The practitioner should carefully guard against the error of mistaking this condition for one of recrudescence; it is due to exhaustion, and, as such, requires a judicious stimulating plan of treatment. It may be known by the coldness and pallor of the surface, and the weak compressible state of the pulse.

When occurring in the course of acute rheumatism prophylactic measures should always be adopted, such as the avoidance of cold, clothing the patient in flannel, and carefully watching the condition of the joints: if there be retrocession of the swelling and redness and pain in the joints, while the patient becomes restless and loquacious, we must try and bring back the rheumatism to the joints by wrapping them up in mustard poultices or applying blisters. Opium and musk have been recommended, and Trousseau declares that he has cured three patients by the combined administration of these drugs, although he adds that two others got well without any active treatment having been employed. In any case, the alkaline treatment for rheumatism should be continued, and careful nursing and the administration of unstimulating food adopted.

Chronic Meningitis.—This is a very rare affection, and it is generally recognized after death only from the pathological appearances met with in the meninges—namely, thickening and opacity of the arachnoid, cellular adhesions between it and the pia-mater, development along the falx cerebri of the so-called glandulæ Pacchioni, which from their absence in infancy and youth are generally regarded as evidences of chronic meningeal irritation. In some cases, plates of osseous tissue have been found in the membranes; whilst during life, the symptoms presented by the patient did not point to any mischief in the head.

Troublesome headache, a disposition to somnolency, sometimes convulsive twitchings, and in children, vomiting, are regarded as symptoms which should excite suspicion of the existence of chronic Meningitis. Of that form of the affection—which is complicated with chronic inflammation of the superficial layer of the cortical substance of the brain, and is symptomatically characterized by lofty ideas, hallucinations, paroxysms of maniacal excitement and embarrassment of speech, followed by gradual general paralysis of motion, sensibility being scarcely affected, and terminating in idiotcy—we have

nothing to say here, as the affection is always treated of in conjunction with insanity.

Although chronic Meningitis in middle age and in early life is of rare occurrence, it is not so rare in old age. Concerning it Machlachlan observes, "The disease may be of a chronic nature, *ab initio;* chronic in regard to the subdued and insidious nature of its symptoms, while at the same time it pursues a strictly chronic course, seemingly, now and then existing one or two years, and never following an acute attack of the disease. It is not an unfrequent result of albuminuria and repeated attacks of delirium tremens, or it follows gout and rheumatism. Chronic Meningitis in the aged is almost uniformly accompanied with great impairment of the mental faculties, frequently with thickness of speech, and paralytic weakness of the lower extremities, the gait being tottering and feeble. The energies of the system are reduced; all movements of the limbs are performed awkwardly, slowly, and with uncertainty. The appetite remains good; but digestion is slow, bowels are inactive, and the various excretions vitiated. Vertigo, singing in the ears, marked loss of memory, slowness of comprehension, periodical fits of passion, and occasional attacks of headache, with or without signs of high vascular excitement, are frequently observed. Sooner or later the invalid takes to his bed reluctantly. There he lies uncomplaining, vegetating, and gradually sinking, dying often in consequence of sloughs on the nates."

Treatment. — The diagnosis of this affection being so uncertain and obscure, it is clear that little is known regarding the mode of treating it. If the symptoms in the least show a tendency to assume the acute form, the application of leeches behind the ears, cupping the nape of the neck, and administration for a short time of small doses of a mild mercurial, would be called for. Otherwise the use of repeated blisters applied to the nape of the neck, and the internal administration of iodide of potassium, and occasional purgatives, seem to be the most rational treatment that can be employed. In the form occurring in old age, cold lotions to the head, and an occasional brisk purge followed up by an enema, are most advisable. The condition of the bladder must be frequently examined, and the catheter employed should retention arise; when, on the contrary, there is dribbling, a proper apparatus must be worn. Good nursing, cleanliness, the hydrostatic bed, are also essential. In the later stages of the disease, when the vital energies begin to part and fail, wine will be essential, but until then the treatment should be strictly antiphlogistic; the patient avoiding also all mental excitement, and, if not secluded, he should be kept tranquil both in body and mind.

Secondary Meningitis. — This affection sometimes shows itself in the course of one of the eruptive or exanthematous disorders, as typhoid fever, measles, scarlatina, and variola.

When Meningitis occurs in the course of an eruptive fever, some of its ordinary symptoms may either fail entirely, or be masked by those of the primary disease. Thus, the intense headache of acute primary Meningitis may be absent, or it may be slight only, and there may be no vomiting. The invasion of the superadded disease may, however, be recognized by a sudden slackening and irregularity of the pulse and respiration, by the pallor and anxious look of the face, and the extreme jactitation which ushers in the delirium.

TUBERCULAR MENINGITIS.

By Samuel Jones Gee, M.D., F.R.C.P.

By tuberculosis we mean that disease which is attended necessarily by the formation of miliary tubercle. Inflammation of the brain and its membranes, occurring as a consequence of the progress of tuberculosis, is called Tubercular Meningitis.

Causes. — This is not the place in which to set forth the knowledge which has been gained of late years respecting the etiology of tuberculosis in general. A few conditions which predispose to Tubercular Meningitis are all that need to be dwelt upon.

Be the case as it may with regard to the adult, I think that most physicians will agree that the tubercular disposition in the child is strongly hereditary; it will be found that a large majority of children attacked by Tubercular Meningitis come of families in which there are, or have been, sundry manifestations of a tendency to scrofulous or tubercular diseases. Yet in many cases no tendency of the kind can be discovered.

The influence of sex, season, or social position upon the occurrence of Tubercular Meningitis is quite insignificant.

Tubercular Meningitis may set in at any age. No doubt the disease is more common before puberty than afterwards: but it is, at present, impossible to procure numerical proof of the fact, and this on account of the comparative absence of children from the general hospitals into which adults are admitted.

Age.	Cases.	Age.	Cases.
Six weeks	1	16 to 20 years, inclusive .	10
Two months . . .	1	21 to 30 " .	12
2 to 4 years inclusive .	12	31 to 40 " .	5
5 to 7½ " " .	11	41 to 50 " .	3
8 to 10 " " .	13	51 to 60 " .	2
11 to 15 " " .	9	68 years	1
Total . . .	47	Total . . .	33

However, there can be no doubt that Tubercular Meningitis is comparatively much more common under two years of age, and much less common after eleven years of age, than these figures would make out. At the Hospital for Sick Children the following cases of Tubercular Meningitis were examined *post mortem* between August, 1862, and March, 1871, inclusive:[1]—

Under	1 yr.	2 years.	3	4	5	6	7	8	9	10	11	12
Males . .	...	3	13	10	9	6	5	1	...	1	...	...
Females .	...	6	5	9	3	2	2	4	2	...	...	...

Being 48 males and 33 females. Seven cases of non-tubercular cerebro-spinal meningitis were examined in the same space of time; whereof six were males (at 6 months, 2, 3, 6, 8, 9 years of age), and one was a female of 20 months. No case of simple cerebral meningitis, not traumatic, during that period. After puberty, both acute tuberculosis and Tubercular Meningitis are much more common in the female than in the male sex.

Symptoms.—The symptoms of Tubercular Meningitis sometimes break in suddenly upon what has seemed to be, so far, a state of perfect health; sometimes they are preceded by several weeks or months of indistinct poorliness: both groups of cases (seeing that in both the sure and certain symptoms of distinct disease are cerebral from the very first) may be classed together under the head of Primary Tubercular Meningitis. But, on the other hand, Tubercular Meningitis sometimes attacks a person who has already exhibited symptoms and signs of either acute general tuberculosis, or of local tuberculosis, acute or chronic: this may be called Secondary Tubercular Meningitis. Ordinarily its existence is apt to be overlooked, unless its special symptoms have been watched for; sometimes, indeed, the disease is wholly latent, and discovered *post mortem* only.

I. Primary Tubercular Meningitis:
   1. With premonitory symptoms.
   2. Without them.

II. Secondary Tubercular Meningitis, preceded by manifestations of
   1. General Tuberculosis.
   2. Local Tuberculosis: i. cerebral;[2] ii. thoracic; iii. abdominal.

The primary form of the disease with premonitory symptoms is the typical form.

Primary Tubercular Meningitis in the Child.

1. *Premonitory Symptoms.*

Their Character.—i. Loss of flesh is the most constant precursor; indeed, is almost

[1] Children under two years of age are not usually admitted.

[2] *I. e.* Tubercular tumors of the brain which have produced symptoms.

constant; often the first, sometimes the only symptom: the child's face being very much spared, it is when the nurse comes to undress him that she finds the limbs to be losing their roundness, and the flesh to feel flabby. This loss of flesh mostly proceeds continuously; sometimes, however, the child will seem to pick up for a time and afterwards begin to waste again. ii. Loss of color concurs; it is sometimes masked by a false color in the cheeks, due to permanently dilated capillaries; it may be only now and then that this color leaves the cheeks, and then the real paleness of the child is obvious. iii. The child is drowsy by day and restless at night; he is easily tired, and in the midst of play will lie down on the floor and fall asleep for a short time; at night he grinds his teeth, sleeps with his eyes half-open, starts, and cries out; he is sad, fretful, peevish, taciturn, and wants to be let alone: if for any reason a young child has been put to bed for a day or two, he will not take to his feet again. iv. Headache is frequently absent, rarely a prominent symptom, but mostly present to a certain degree: very young children will be observed to put their hands to their heads, and to toss their heads on their pillows; older children will say that their heads ache, and this especially after some exertion of mind or body; a child will go to school and come home complaining of his head: the headache is rarely severe, and mostly frontal: a strong light increases it. v. Feverishness is not always present; when it is present, it is noticed chiefly in the evening. I do not myself possess any thermometrical observations made during this period, nor do I know of any that have been published.[1] vi. Loss of appetite is common; vomiting uncommon; the bowels are confined, or relaxed, or quite regular in action.

These symptoms are grouped in every manner possible; any of them may be absent, and how many soever be present, they do not justify more than a fear least the condition should terminate in Tubercular Meningitis.[2]

They are sometimes due to progressive tuberculosis, but sometimes certainly to a catarrhal state or to simple nervous depression. The pyrexia is the most important symptom. When a child suffers, day after day, from a slight elevation of temperature above the normal, that elevation of temperature may be due to tuberculosis. Inasmuch as a catarrhal fever does not usually last longer than ten or twelve days; if the pyrexia do last longer than that time, and if the presence of continued fever or of a local inflammation can be excluded; the elevation of temperature is very probably due to tuberculosis. But in actual practice we are not often called upon to study the precursory symptoms of primary Tubercular Meningitis so closely. This is certain, that the most careful mother will often fail to observe any feverishness before the day of invasion, even though she have been disquieted for weeks or months by her child's steady loss of flesh and strength. The poorliness which follows acute specific or other diseases often passes uninterruptedly into the premonitory period of Tubercular Meningitis. Measles, so far as I have seen, is the most common antecedent, hooping-cough next, occasionally an attack of diarrhœa and vomiting, bad sore-throat, or hip disease.

The *Duration* of this period is different in different cases. Sometimes, as mentioned before, there are no prodromata at all.[1] There are all grades between this extreme and the other in which a child loses flesh for four, six, or even more months before the invasion. One or two months may be regarded as the average; sometimes the prodromata last two or three weeks only. Again, in some cases, the precursory stage is interrupted by a temporary improvement in the health of the child.

2. *Invasion.*

By the invasion of Tubercular Meningitis I mean the period at which there occur such new symptoms as enable us to pass from the uncertainties of the prodromal stage, and to declare most positively that from this date, at all events, the child has sickened with hydrocephalus. The invasion symptoms are the first which make the friends of the child think him dangerously ill; the poor, as a rule, only now begin to seek advice.

Character of Symptoms.—(1) Vomiting is by far the most common special invasion symptom. In the majority of cases the frequent repetition of the vomiting makes it seem to be the most important symptom of the onset; but sometimes, although serving to mark the invasion, the sickness is subsidiary in urgency to the other symptoms. In the former group,

[1] The reader will bear in mind that *primary* Tubercular Meningitis only is under consideration.

[2] Refer to section on Diagnosis.

[1] It may afford some notion of the frequency with which the prodromata occur to mention that out of twenty-six cases of primary Tubercular Meningitis (the diagnosis having been confirmed by a post-mortem examination in all), there were only two in which premonitory symptoms had not been noticed. That sometimes the prodromal stage is absent, or so slight as to be unnoticed, even in children carefully looked after, I have no doubt.

the child may vomit incessantly with and without taking food; in the latter, the child may be sick only once or twice. (2) Convulsions are the next most frequent (though much less frequent) special symptom of the invasion; they, like the vomiting, may be repeated several times or not. Sometimes the attack is epileptiform, sudden with complete unconsciousness, and yet without convulsive movements. It will give some notion of the comparative frequency of the different modes of invasion to mention that, out of twenty-five cases in which the invasion symptoms were carefully ascertained, vomiting without convulsions occurred in nineteen; vomiting followed by convulsions in one; convulsions without vomiting in two; several attacks of general rigidity, succeeded by vomiting on the third day, in one; in one an attack of temporary unconsciousness (epileptiform), followed by vomiting; and in the remaining case the invasion was marked by a notable and comparatively sudden increase in the severity of the premonitory symptoms (headache, drowsiness, loss of flesh), without vomiting or any motorial symptoms. The most important concomitant symptoms of the invasion are: the first occurrence or the increase of headache, or of the pyrexia, or of drowsiness; the co-existence of constipation as a rule with an occasional exception; there is often a change in the temper of the child, or some odd unreasonable behavior; the character becomes morose, irascible, and obstinate.

Period of Occurrence.—From seven to twenty-one days elapse between the invasion and death; the average is fourteen days. "When the meningitis sets in suddenly without prodromata, its duration is from twenty to thirty days, rarely less, provided that no complication modify the course of the disease" (Rilliet, iii. 487)—a very necessary qualification.

3. *The Established Disease.*

It is well known that Dr. Robert Whytt first described the course of what he rightly called "the most frequent species of the hydrocephalus internus;" and so described it that his successors have not been able to add much that is useful to his description.[1] The disease depicted by Whytt is a clinical entity of the most definite kind; but when we come to use the phrase Tubercular Meningitis, and to frame a species of disease characterized by certain anatomical changes (namely, tuberculosis and inflammation of the meninges), we find that we have to do with a more comprehensive notion than that intended by Whytt. The disease of Whytt corresponds to primary Tubercular Meningitis, involving the base of the brain, and occurring in children. But Tubercular Meningitis may be secondary; and, when primary, may not involve the base of the brain, or may attack adults: to these forms of the disease Whytt's description ceases to apply. Nevertheless, the set of symptoms pointed out by Whytt remains the most common and best marked manifestation of Tubercular Meningitis during life, and may be deemed the typical form, and will therefore be first described.

The peculiar symptoms of Whytt's disease depend upon the fact that the meningitis affects the base of the brain; the tubercular nature of the meningitis plays no part in the production of these symptoms. For both tubercular and non-tubercular meningitis of the base are attended by precisely the same symptoms; and Tubercular Meningitis involving the convexity alone is not accompanied by the symptoms which are peculiar to meningitis of the base. It must therefore be clearly understood that the form of disease which will be first and most fully described is, in reality, dependent upon basilar meningitis; and that, in a given case, finding this condition to be present, we assume it to be tubercular also, because basilar meningitis (unaccompanied by spinal meningitis) always is tubercular.

A. Meningitis of the Base.

Whytt's first stage of dropsy of the brain includes the premonitory and invasion periods, previously described. The subsequent course of the disease he divides into two stages; the one being an earlier period during which the pulse is infrequent, and the other a later period of frequent pulse. This division of Whytt's is true to nature, but the pulse is a fallible criterion. In order to recognize that a patient is in one or other stage of the disease, the physician must look at the symptoms in a comprehensive spirit. There is an earlier period (the second of Whytt), in which the brain may be regarded as reacting under or against the disease: the symptoms are *sthenic*; headache, delirium, exalted sensibility, infrequency of the pulse, consciousness being retained. The disease goes on to produce destruction of the brain; the symptoms become such as are due to a steadily increasing *paresis* of the animal functions; stupor, insensibility, paralysis, frequency of the pulse—the third period of Whytt. To repeat: the observer must not narrow his view to the variations of a single symptom, or he will often fail to perceive the

[1] Works of Robert Whytt, M.D. Edin. 1768.

stages of Dr. Whytt, or even be inclined to reject them. In the second of the ensuing paragraphs I have noticed the variations of the pulse somewhat minutely, so as to show how little it is to be trusted as a certain sign of the stage of the disease.

(1) Digestive Organs.—i. *Vomiting.*—As already mentioned, the vomiting of the invasion is ordinarily repeated several times. Sometimes the symptom is very urgent, occurs whether food has been taken or not; and this may be so every day for a week. When once the tendency to vomit has ceased for twenty-four hours, it does not ordinarily recur. ii. *Constipation.*—In the great majority of cases the bowels are constipated throughout the whole course of the disease; yet sometimes they are spontaneously relaxed throughout; more frequently (especially when hard purging has been part of the treatment at the beginning of the disease) they are relaxed without the use of drugs, towards the end. The constipation is not often obstinate; it is easy, for the most part, to procure an action of the bowels by common means. iii. *Retraction* of the walls of the belly is a symptom which will be met with at some time or other in nearly every case: sometimes present for a day or two only, and at no certain period; sometimes present all along. Acute diseases affecting the brain are the only acute diseases of children which, as a rule, cause great excavation of the belly. iv. *The Tongue* has no fixed character; it may be moist and clean. Aphthæ sometimes occur as death draws nigh.

(2) The Circulation.—i. *The Pulse* affords symptoms which have been much studied, and with good reason, for they are most important. *a.* Its frequency is diminished in the earlier and increased in the later part of the disease: this is the rule. The increase mostly sets in during the second week; sometimes much later (*e. g.* boy, of four years, eighteenth day = 84; 116, 128, 128, 168, on successive days, death on twenty-third), or earlier (*e. g.* girl, eight years, sixth day = 140). Sometimes the increase occurs only three or four days, sometimes eight or ten days, before death. Sometimes the increase takes place suddenly (*e. g.* boy, two years four months, seventh day = 72; eighth day = 164), sometimes gradually. After the pulse has been very frequent, it may again become comparatively infrequent (*e. g.* girl of one year and ten months, eighth day = 162; ninth = 144; tenth = 100; eleventh, day of death, = 180.) This infrequency may persist and increase up to the day of death (*e. g.* girl, two years and six months, fifteenth day = 120; sixteenth = 140; seventeenth = 76; eighteenth = 70; *i. e.* day of death: cases of this kind die in a state of algidity unsurpassed in any other disease.[1] As to the absolute frequency, I have not known the pulse to fall below 64. At the invasion of the disease "the pulse is not much accelerated: 108, 112, 120 at most; sometimes it is even already slackened" (Rilliet, iii. 480). The frequency is at all times easily increased by movement of the body. *b.* Irregularity in the rhythm and inequality in the force of the pulse are two symptoms upon which great stress is laid in the diagnosis of Tubercular Meningitis. They coincide with the period of infrequency; not that an infrequent pulse is irregular at all times, but it will be found so, at least, now and then. During the period of increased frequency the irregularity is less easy to detect, and is probably really less common; yet a pulse of *e. g.* 170 will sometimes be found distinctly irregular. ii. The modifications in the *cutaneous circulation* are well displayed in the face. If habitually pale (as it sometimes is from beginning to end of the disease), a flush is easily produced by excitation of any kind—by moving the child, giving him to drink, brushing the hand over the cheek, and so on. Sometimes the flushing is spontaneous: limited to one cheek, or general as regards the head; constant, or alternating with pallor. The highly characteristic facies of acute brain disease[2] is chiefly effected by the congestion of the face. What is best seen in the face is observed to a less marked degree in the skin of the trunk and limbs; that is, the skin is injected at times. Spontaneous injection is often made evident by the unusually distinct white ring left by the pressure of the end of the stethoscope. As in the face, so in the skin of the trunk and limbs, injection, when not present, may be easily procured: draw the finger across the skin, and, in a few seconds, a red streak will appear along the track of pressure. This is what Trousseau has called the "tache cérébrale": for my own part I believe it to be a sign which does not possess any diagnostic value.

(3) The *Respiration* also yields symptoms deserving careful study. The frequency is increased, diminished, or at the natural rate. Increased frequency of pulse and respiration sometimes go together, but not always (*e. g.* boy of five years, P. = 190, R. = 24; day before death). Irregularity and inequality are often very striking in later stages of this as well as of all other acute cerebral diseases of childhood. Sometimes the respiration is irregular only, sometimes unequal only, often both irregular and unequal: the child may remain for many seconds as if he had forgotten to breathe, then follow a few rapid respirations, then

[1] Refer to paragraph on Heat of Body

[2] See paragraph (8).

another pause, and so on. A careful examination will sometimes be necessary to detect irregular breathing. Sighing expiration is particularly common when children become half-insensible from Tubercular Meningitis.

(4) *Heat of Body.*—The first three or four days of the established disease are attended by what seems to be (judging by the hand) a distinct increase in the heat of the skin. This is followed by a period of low pyrexia, during which the temperature only occasionally exceeds 101°. I do not say that the temperature is not sometimes persistently higher, but I do not happen to possess notes of any cases in which it was so; whilst, on the other hand, for days together the temperature may vary between 96° and 98°. As death approaches (say for about the four days preceding death) cases have seemed to me to group themselves into three classes: in one, the state of moderate fever continues up to the very day of death; in another, the fever greatly increases before death; in a third, the body-heat falls below the standard of health. An example of each class will make this more clear:—

Day before death.		Third.		Second.		First.		Day of death.	
Type 1.	Boy: 4 years	99·5	102·0	99·0	101·0	100·5	101·5	99·0	101·5[1]
Type 2.	Boy: 2½ years	97·0	99·3	99·2	101·0	100·0	103·0	104·4	107·25[2]
Type 3.	Girl: 2½ years	97·8	96·6	96·2	93·0	82·8	82·1	80·5	79·4[2]

The minimum and maximum temperatures of each day are given.

The peculiar variety of ardent fever referred to the second type (the lipyria of Galen) is, perhaps, more common in Tubercular Meningitis than in any other disease.[1] "Heat of the viscera, as if from fire, but the external parts cold; the extremities—that is to say, the hands and feet—very cold" (Aretæus). A thermometer in the arm-pit will prevent our being misled by the coolness of the exposed parts. In the third type the algidity involves the viscera themselves; the temperature in the example adduced was taken by means of a thermometer kept permanently in the rectum; and, as the hyperpyrectic cases might deceive the hand applied to the limbs only, so might the state of algidity deceive the eye. The example chosen one hour before her death, when her temperature was 79·8°, her breath cold to the hand, and her pulse imperceptible at the wrist, still kept a little color in her cheeks, and (except that her eyes were half-open) it would have been impossible for one merely looking at her to have said that she was not a tolerably healthy child calmly asleep. The pulse, as a rule, agrees with the temperature, rising in frequency as the temperature rises, and falling as it falls: no proportion is kept in the amount of the rise and fall. Excessive frequency of pulse may concur with a moderate elevation of temperature; or, reversely, the temperature may be high and the pulse infrequent (*e. g.* temperature 103°, pulse 72—a ratio really observed, and such as would serve to clench the diagnosis of acute disease of the brain). Heat of the head greater than of the rest of the body is a symptom far from always observed in the earlier stages of the disease, and still less frequently in the later.

(5) Nervous System.—i. *Headache* concurs with the invasion, or, if present previously, is much increased then. The pain is mostly referred to the top of the frontal bone. The headache is tolerably constant, subject to paroxysmal exacerbations, and lasts until stupor sets in. The temporary increase of headache is sometimes made known by the moaning of the child; sometimes he cries out, "Oh! my head," or shrieks,[3] or holds his hands hard on his head; he greatly dislikes any disturbance, for that increases the headache. But it must not be supposed that headache of this severity is present even occasionally in all cases. ii. *Eye-symptoms* are very important. One pupil is often distinctly larger than the other: this state is present at some time or other in every instance of the disease, does not occur at any special period, and is not always constant; thus the inequality in size, present in the earlier part

[1] See article on Scarlet Fever, vol. i. p. 83.

[1] One hour and a half before death.

[2] At very moment of death.

[3] I copy the following particulars relating to the "hydrocephalic cry" from Trousseau (Clin. Méd. 2me édit. vol. ii. 239):—"It is a single, violent cry, resembling the cry of a person suddenly exposed to great danger: the expression of the face is not that of suffering: any period of the disease may be attended by this cry, which may occur every hour, half hour, or even every five minutes." Rilliet (iii. 503) does not consider this to be either a common or a special symptom—an opinion with which my own experience would lead me to coincide.

of this stage, not rarely disappears later on in the disease, in order, it may be, that the relation of size may be reversed for a day or two before death: or, sometimes, the variations are much more rapid. Dilatation and sluggish action of the pupils is the rule towards the end of the disease, but the absolute size of the pupils is of small value in diagnosis. Squint is present sooner or later in every case. Hemiopia may occur so early in the disease as to be discoverable (Trousseau, ii. 236, 237). Oscillation of the eyeballs, or of one eyeball (the other being fixed), is common in the later period.[1] iii. *Paralysis of the Face*, one eye opened less widely than the other, one nostril being rounder than the other, one corner of the mouth less acted upon by the muscles than the other, one side of the upper lip straightened—these are frequent concomitants of the later period. iv. *Paralysis of the Limbs.*—Quite towards the end of the disease we often observe one or more of the limbs to be unmoved, relaxed or feebly rigid, flexed or extended. v. *Convulsions and Rigidity.*—These have been already mentioned as occasional invasion symptoms. As terminal symptoms they are equally frequent, occurring on the day of death, the child perhaps dying immediately after a convulsion. Yet they are not to be trusted as a sign of impending death, or even of death likely to occur in a day or two. When convulsions have been invasive they do not necessarily recur. The following may be taken as an example of the state of a child in convulsions towards the end of the disease: he lies unconscious, whole skin injected, eyeballs drawn upwards and to one side, pupils large, one side of face more wrinkled than the other, teeth clenched, limbs rigidly extended—except the hands, the fingers of which are flexed—slight twitching movements of face and limbs, more marked on one side than the other, respiration labored. As the child comes round the unconsciousness diminishes, pupils become smaller, he is left bathed in sweat. Sometimes the clonic movements are more marked. Sometimes the limbs are relaxed throughout, and the twitchings limited to the face and eyeballs. Permanent feeble spastic rigidity of one or more groups of muscles is common during the latter period; sometimes the rigidity is much stronger. Opisthotonos may be present during the last few days of life; it may be paroxysmal and last only a few minutes, or it may be continuous and last until death. In such cases I have not found any signs of inflammation about the cord or its membranes. Tremulousness of the limbs is very common. A shudder may be often observed to pass through the body from head to foot. Many other niceties of motorial symptoms might have been described: enough has been said to show the infinite variety present in Tubercular Meningitis. vi. *Sensation.*—Tenderness of the skin can be demonstrated to be present in some cases; it is often very obviously present in the scalp when a barber is employed to shave the head. Pains in the limbs are sometimes complained of early in the disease. Dislike of light is common at the same period. Blindness is difficult of recognition, because occurring late. All these symptoms are sometimes unilateral. vii. *Consciousness.*—The children soon become somnolent: they lie with their eyes shut or half-shut, reply to questions in a dry short way or by a nod: when raised up in bed they complain much, knit brows, throw head back, and slip down in the bed. They dislike disturbance extremely: will clench their teeth against food. As the somnolence increases, the children cease to speak, but they will put out their tongues when shaken and pertinaciously asked to do so; they then relapse willingly into their former soporose state. By degrees, or sometimes suddenly, the sopor becomes deeper; but not until near the very end, and not always even then, does the coma become so deep that the child will not withdraw his limbs (provided they are not paralyzed or rigid) when pinched, and also give other signs of being discommoded. Inability to swallow accompanies the coma. Retention of urine is sometimes rather an early symptom. The consciousness may be perfect the day before death. The semi-coma may be continuous from the invasion to the end of the disease. Delirium is common, but is not a symptom of much value in diagnosis, prognosis, or treatment.

(6) *Physical Signs* of tuberculosis of the lungs are not often to be detected in cases which, by reason of their course, are arranged under the head of Primary Tubercular Meningitis. Yet occasionally, and that even when the foregoing poorliness has not been greater than usual, I have detected the signs of a cavity under one or other clavicle. Sonorous râles may be met with, sometimes a little mucous râle; and in exceptional cases, which are, nevertheless, cases of Tubercular Meningitis as opposed to acute tuberculosis, all the physical signs are present of that very fine capillary catarrh which, in children, is nearly always indicative of the coexistence of tubercle or pneumonia. This sign I have observed in Tubercular Meningitis, and in no other disease: namely, the chest heaves equally well on both sides, and yet over a very large part, or even the whole of one side, no respiratory sound is heard

[1] For the ophthalmoscopic appearances, refer to paragraph (6).

by the stethoscope. In a few hours this sign will have passed away. It is probably due to a slight pulmonary catarrh concurring with the respiratory unconsciousness of the brain disease. When the fontanelle is large, it is mostly distended; when small, the distension cannot be perceived.

Of late years the ophthalmoscope has taken an important place among the means for discovering Tubercular Meningitis. Actual tubercles may be occasionally seen in the ocular choroid during life; but this is not a common occurrence, and if the ophthalmoscope were useful in this way only, its use would be very small. Choroidal tubercle was discovered, in one case, six weeks before the invasion of Tubercular Meningitis: Fränkel, Virchow's Jahresbericht, 1869, p. 621. Steffen found choroidal tubercle in four out of five cases of Tubercular Meningitis, and in three cases of the four during life: eod. loc. 622. It is by detecting changes in the vascularity of the retina that the ophthalmoscope renders real service. Whenever meningitis is basilar, we find congestion of the retinal vessels, and sometimes optic neuritis. It will be obvious that there is nothing peculiar to meningitis in these signs; and also, that to find a state of retina which is probably dependent upon intracranial causes must be a very important item in the diagnosis, when we are doubtful whether a convulsion or an attack of vomiting be due to meningitis or not.[1] Meningitis which affects the convexity of the brain, and spares the base, is unattended by any unnatural condition of the optic disks: of this fact I have seen two instances.

(7) *Urine.*—In the case of a boy, aged four years, in whom it was necessary to employ the catheter, I had an opportunity of examining the urine. The following was the result:—

Day of disease.	Water.	Urea.	Ch. sod.	Phos. acid.
19–20	122 c. c.	5·07 grammes	0·195 gramme	0·432 gramme
20–21	122 c. c.	5·51 grammes	absent	0·367 grammes
21–22 (day before death)	171 c. c.	7·34 grammes	absent	0·583 gramme
Mean of 3 days in healthy boy of same age	431 c. c.	15·27 grammes	3·062 grammes	0·967 gramme

Weight of body—case of meningitis . . . . . 19½ lb.
" " healthy child . . . . . 28 lb.

(8) *General Appearance: Summary.*—Although the individual symptoms which have been now described are grouped in almost every possible manner in the different actual examples of Tubercular Meningitis which we meet with, yet it may be well to recapitulate the chief matters in what has gone before, and so to arrange them as to form a sort of idea or type of primary Tubercular Meningitis in the child.

A boy of five years old, in whose parental antecedents there are signs of a tendency to tuberculosis, begins to feel poorly, to lose flesh, and to complain occasionally of his head; he is restless at night, and languid by day; his bowels are rather confined; he is subject to irregular feverish attacks. These symptoms last two months, and then, one day, the child vomits for the first time; during the next three or four days the vomiting is repeated several times; afterwards it ceases; at the same time the fever runs higher, the headache increases, the nights are noisy, the constipation is obstinate. About the time that the vomiting ceases, other symptoms pointing to cerebral disease appear; say, for example, on the sixth day after the first vomiting, he looks thin and pale; skin hot and dry; temperature 100·8° (evening); pulse 84, irregular; respiration 20, regular; tongue dry, red tip, light fur elsewhere; bowels not open; belly natural; converging strabismus of one eye; pupils of middle size, mobile, one larger than the other; he says he has headache, and points to his forehead as its seat; physical signs of chest are negative; tache cérébrale uncertain; the boy is quite rational, moves about in bed, sits up, answers questions, and the expression of his face is not peculiar: the diagnosis rests (and rests surely) on the previous history, the pulse, and the condition of the eyes. On the seventh day he is much the same; belly rather retracted; pulse 108, very irregular; respiration regular; temperature 100·4° (morning) and 101° (evening). Eighth day: no marked change, rather lower; pulse 112, still irregular; temperatures, morning and evening, 100·2° and 100·6°: all the other symptoms remain unchanged. Ninth day: clearly much worse; consciousness failing; does not cry out; probably can still see; tache cérébrale easily produced; cheeks, habitually pale, easily flush; cannot sit up; no special

[1] See especially Dr. Allbutt's papers "On Optic Neuritis as a symptom of disease of the brain and spinal cord." Med. Times and Gaz. 1868, vol. i. pp. 495 *et seq.*

expression in face; swallows well; pulse, 160, regular, weaker; eyes as before; arms very tremulous; temperatures, 101·2° and 103°. Tenth day: still worse, semi-stupor, cannot be made to speak: eyes only half-open; passes excreta under him; lies fidgeting and picking with tremulous hands; pulse, 168, regular; temperatures, 101·8° and 102·4°. Eleventh day: stupor greater; he occasionally moans; whole surface much injected, face and head greatly flushed, dusky; and as the child lies on his back, motionless, with his half-opened and prominent eyes, their corners filled with thick secretion, and the corneæ dusty and filmed, he has a look quite characteristic of hydrocephalus; pupils dilated; one eye fixed, and probably blind; swallows pretty well; belly greatly sunken; pulse, 180, regular, very weak; respiration, 15, irregular; temperatures, 101° and 103°. Twelfth day: stupor deeper still; moves limbs of one side feebly; those of the other side are somewhat rigid; swallows badly; pulse so frequent and feeble that it cannot be counted; feet cold; temperatures, 101·5° and 103·6°. The next morning he dies.

Duration. — The duration of primary Tubercular Meningitis with prodromata is from seven to three-and-twenty days. It has been already mentioned, on the authority of Rilliet, that when the prodromata are wanting the duration is from twenty to thirty days.

Remission in the gravity of certain symptoms is not uncommon in the acute cerebral diseases of children. The direct nervous symptoms are the most variable; the squint, the unequal or dilated pupils, the rigidities, and the somnolence. The variations in the last symptom are the most striking and deceptive, semi-stupor passing away so as to leave the intellect perfectly clear. But when once the physician has satisfied himself of the existence of meningitis, he should not let his diagnosis be easily shaken. As Rilliet observes, "the improvement does not show itself in all the symptoms;" the pulse remains irregular, it may be, the squint or inequality of pupils persists, and though the remission should last a day or two, the child will die as surely and as early as if all the symptoms had been continuous.

Termination. — Tubercular Meningitis, running the course which I have now described, has but one termination, and that is death. But it has been suspected, and with good reason, that recovery sometimes takes place in the earliest stage of the disease.[1] The probability of this opinion has been greatly increased by the result of ophthalmoscopic examinations. If a child become febrile, and convulsed, generally or partially; if it vomits, or complains of headache, and at the same time the optic disks be found congested, it is very likely that it has basilar meningitis. All these symptoms may pass quite away: but occasionally the patient is left more or less imbecile, or epileptic, or partially paralyzed, or with progressive atrophy of the optic disks. In these cases, however, a recurrence of the disease is to be greatly dreaded.

[The following is the report[1] of a case occurring in my practice, of a sufficiently exceptional character to be given in full:—

"A strong predisposition on the part of the patient, was proved to exist toward cerebral disease, by the facts that the child's grandmother has been for eight years hemiplegic after apoplexy; that one of her sons has been insane, and another died of disease of the brain; that the child's mother died a few months since at the Pennsylvania Hospital, having been there under treatment for chronic mania; and that the elder brother of the patient, aged ten years, having attended the funeral of his mother, was attacked by a convulsion on the same day, and, in spite of prompt treatment, died in thirty-six hours.

"H. S., a boy,—aged five years—had enjoyed tolerable health during the past summer, with the exception of occasional diarrhœa. Never, however, had he appeared to be a robust child. About the first of October he became languid and peevish, with loss of appetite, and soon afterwards vomiting, with deranged character of the fecal discharges, and a tendency to costiveness, alternating with slight diarrhœa.

"On the 9th of the month, he was brought to me, complaint being made by his nurse of his vomiting repeatedly through the day, and seeming fretful and miserable. His face was very pale, and lips bloodless. Some mild stomachic treatment was advised, the nature of the case not being then suspected.

"The next day, pain in the head came on, with great restlessness; the forehead being hot, and pulse full and about 100. The vomiting continued; two dozen American leeches were applied to the nucha, his bloodless aspect of the previous day, and other indications of an enfeebled condition, forbidding venesection. The leeches drew blood freely. Citrate of magnesia was directed, as the bowels were constipated. In the evening, the heat of the head was lessened, but opisthotonos had come on; the head was retracted so that he lay at times in a per-

[1] See Dr. Allbutt's papers "On the diagnostic value of the ophthalmoscope in 'Tubercular Meningitis.'" Lancet, 1869, vol. i. pp. 596 and 599.

[1] Transactions of Phila. College of Physicians, New Series, vol. ii. No. 7, p. 343.]

fect arch, with the top of the head upon the pillow; even during the short periods of his sleep, this unnatural contraction of the muscles continued.

"On the morning of the 11th, as the citrate of magnesia had failed to operate, a laxative enema was administered. Through this day, the opisthotonos persisted, and the child lay without signs of intelligence, screaming and sleeping alternately; the cry being mostly sharp and not prolonged, the 'hydrencephalic cry' of authors. His pulse had now become slow—and the heat of head was very moderate. On this evening a blister, four inches square, was made to vesicate the back of his neck.

"The 12th found him with some temporary amelioration. The tetanic arch of the neck had partially relaxed. He was still indifferent and restless, however, especially at night, at which time some fever was said by his attendants to come on. Bowels costive—moved only by injection. Urine retained twenty-four hours, and very dark colored; warm fomentations and sp. æth. nit. dulc. were used for the relief of the retention. The great nervous excitement caused by the effort to overcome his resistance against medicine, deterred me from advising anything further at this time.

"On the 13th, I find it stated on my notes that he appeared to be a little better. No decided alteration, however, occurred, and the night was much disturbed by his restlessness. Vomiting had at this time ceased. His nourishment was thin tapioca, made with milk. The bowels still required movement by injection. During the 14th, 15th, and 16th, no important difference in his symptoms occurred; the costiveness, retention of urine, indifference through the day, and restlessness at night, being the leading symptoms. Pulse, at the time of my visits, about 80; head not hot.

"On the 17th, eight days after I had first seen him, and about twice that interval since his first signs of indisposition, a violent *general convulsion* occurred, lasting fifteen or twenty minutes. Pediluvia, and cold to the head, were resorted to, and a hyoscyamus and asafetida mixture was given by the mouth. After the convulsion, the pulse was slow, the pupils dilated, and consciousness more obscured than before.

"On the 18th, his nurse reports that he had during the night numerous convulsions, at intervals of less than half an hour. He screams frequently, and rolls from side to side. Eyes entirely vacant in expression. The convulsions continued through this day in the same manner, affecting chiefly the left arm and leg, and the muscles of the face. The pulse at this time, and for the four following days, was slow and irregular; the irregularity being very strongly marked. The bowels and bladder were opened during the spasms repeatedly, and, when in the interval, involuntarily and unconsciously, in the bed. A warm bath was at this time employed, his resistance while conscious, having interfered with it.

"During the 19th, the same condition was maintained. His father told me that he had a spasm every fifteen minutes—but feebler. The right limb occasionally moves during the convulsion, but is motionless otherwise. Pulse has not lost its strength or irregularity. He refuses to swallow—or chokes when compelled to receive fluid into the mouth. No evidence, whatever, of sight or hearing exists; the breathing is almost stertorous, and gritting of the teeth nearly incessant. Through this night also the convulsions continued. I noted, in my memoranda, that he would certainly die, and gave nearly the same opinion in reply to inquiries of some of the family.

"On the 20th, at my request a renewed attempt was made to induce him to swallow, although the attendants declared that he could not open his lips. He was raised up, and drank a few teaspoonfuls. Some slight appearance of improvement in his aspect, with the subsidence of the convulsions, induced me to urge, although without any hope, the renewal of the blister. This was attended to; he was nourished with tapioca; the bowels were once more emptied by injection; and sponging briskly all over with warm spirits and water was advised and practised. The father continued the use of the hyoscyamus mixture, avowing that it calmed his restlessness.

"21*st*. No more of the convulsions. Pulse still irregular. He certainly looked decidedly better, turning his eyes towards us with a conscious and attentive expression. The right arm and leg, however, were *paralyzed*, and his apparent efforts to speak proved abortive. The blister raised well.

"22*d*. All the symptoms have improved, except the paralysis. Repeated stimulating frictions were used to the affected limbs.

"23*d*. The right leg already begins to show some return of control. Speech is yet very slowly and imperfectly accomplished, but his senses appear to be acute, and consciousness perfect. He receives nourishment with avidity, and sleeps well. He continued to improve from that date, and, by the 30th of October, had entirely regained the use of both the limbs which were paralyzed, being, in all respects, so far as I could discover, well.

"I had, during its progress, supposed this to be a case of Tubercular Meningitis, from the slowness of its approach and ad-

vance, with but moderate circulatory excitement, and most obstinate cerebral symptoms. Apart, also, from the difficulty of diagnosis between this form of the disease and simple meningitis, I had founded an unfavorable prognosis upon the same facts, with the knowledge of the child's inherited predisposition. The symptoms which especially pointed to this expectation were, the great *frequency of the convulsions* (according to the child's father, at least every twenty minutes during the night of the 17th, the day and night of the 18th, and the day-time of the 19th—two days and two nights); the occurrence of these convulsions, also, at a *late period of the disease*—between the eighth and the sixteenth day; the *irregular pulse*, observed steadily during four or five days; the total absence, through most of the same period, of sensation or any evidence of consciousness; the involuntary discharges; and, lastly, the *hemiplegia*.

"Rilliet and Barthez assert that when, with symptoms of meningitis, general convulsions occur frequently or with violence, they almost always coincide with tubercles of the substance of the brain. 'Perfect paralysis,' says Dr. Gerhard (*Amer. Journ. of Med. Sci.*, May, 1834, p. 107), 'did not occur, unless immediately before death.' And Dr. Meigs terminates his list of the signs which most positively indicate the near approach of death in meningitis with the phrase, 'particularly general convulsions.'

"The same writer (Dr. J. F. Meigs) alludes, without details, in his work on the *Diseases of Children*, to a case which I suppose to have been somewhat similar; and, while treating of Sir Benjamin Brodie's plan of mercurial inunction, he refers to a parallel instance, occurring under that treatment, recorded by the editor of *Braithwaite's Retrospect* (vol. iv. 1846).

"But I do not, in any of the works I have had opportunity to consult, find such definite statements, particularly with regard to the possible time of duration of convulsive symptoms, in cases which recover, as enable me to judge, authoritatively, of the degree of rarity of such an occurrence as has just been narrated. Charpentier asserts that, in the *first* period of tubercular meningitis, a cure is possible. In the second stage, when no doubt can exist as to its nature, Guersant believes that he has seen one recovery in a hundred cases. Of those which arrive at the third period, he has seen none recover, *even temporarily*.

"It is, then, my duty to record that the above-mentioned child, H. S., continued in good health, with excellent appetite, digestion, and spirits, and increasing in flesh, until November 26th, about a month after the date of his previous recovery. He was at that time, while returning in an omnibus from Frankford, attacked with a convulsion, which did not leave him entirely until the middle of the next day, the 27th, when he died.

"Must not this, however, be considered a *new* and separate attack from the one above described?

"On referring to Guersant's elaborate article on 'Méningite Tuberculeuse,' in the *Dictionnaire de Médecine* (1839), I find a record by him of two cases somewhat similar in their mode of termination.

"The one, he says, having been treated in the Hôpital des Enfans, 'en était sorti dans un état de demi-convalescence, lorsqu'au bout de cinq semaines il fut repris de nouveau de tous les symptômes de la maladie aiguê, a laquelle il succomba: les caractères anatomiques de la méningite tuberculeuse furent parfaitement constatés par la nécropsie.' The other case resembled this, except that the period of apparent convalescence lasted two months, with a good appetite, and 'ayant repris l'embonpoint;' after which he was reattacked, and died.

"May we not imagine, however, that, if such a convalescence could last two months, it might, in a case affected with nearly similar lesions, be prolonged indefinitely?"—H.]

B. Meningitis of the Convexity.

No doubt Tubercular Meningitis usually affects the base of the brain; but it is equally certain that this is not always the case. And inasmuch as the most characteristic symptoms of ordinary Tubercular Meningitis are in fact the symptoms of basilar meningitis, it follows that when the meningitis is not basilar, it is not attended by those symptoms. That is to say, the vomiting of the invasion period, the constipation, the infrequent and irregular pulse, the unequal pupils, the ophthalmoscopic signs of disease, the strabismus and other local paralysis, are absent from meningitis which does not involve the base of the brain. A state in which general convulsions are either present or imminent, the intervals between the convulsions being occupied by tremblings and twitches of the limbs and face, turning of the thumbs in upon the palms, clenching of the fists, stiffness of the back, neck, and limbs—in short, a convulsive state, which is constant (except perhaps quite at the close of the disease)—this is the prominent symptom of Tubercular Meningitis of the convexity. Add moderate pyrexia, and a pulse which is frequent and very variable in its frequency.[1] The

[1] I wish it to be understood that I do not speak of cases of cerebro-spinal meningitis.

clue to diagnosis is to be found in the acuteness of the disease, the convulsive state, and the constant pyrexia: the evidences of basilar meningitis and of cerebral abscess being wanting. Meningitis of the convexity runs a more rapid course than meningitis of the base: two weeks, one week, or even less, commonly see the fatal termination.

Secondary Tubercular Meningitis in the Child.

As before explained, meningitis is called Secondary when its symptoms have been preceded by manifestations not to be doubted of tuberculosis elsewhere. It has also been mentioned that, with this condition, the onset of the meningitis is, as a rule, obscure; a fact which will not surprise the reader when he considers the nicety of the premonitory symptoms, and the slight prominence of the commoner invasion symptoms appearing in the midst of a state of tuberculosis already existing. A state of *acute* tuberculosis, we ought rather to say; for when the tubercular disease is of that chronicity which we sometimes see, lasting for years (insomuch that tuberculization having probably ceased, the patient suffers from its permanent effects merely), the recurrence of actual tuberculization is only somewhat less marked than its supervention upon a healthy state. The small number of cases then (with regard to children, very small) which belong to the latter class may be dismissed from further consideration, differing as they do from primary Tubercular Meningitis only in this, that they run more rapidly to death. On the other hand, in a case of acute tuberculosis the cerebral lesion may have reached the point of complete softening of the septum lucidum and fornix, and not have produced any symptoms of hydrocephalus which could be discovered even by the observer watching for them. Between these extremes with regard to curtailment of symptoms there are all possible grades.

Tubercular Meningitis, when secondary to cerebral tubercle, is attended by symptoms which are for the most part distinct enough. This, indeed, would almost follow from the law before laid down; for cerebral tubercle (that is to say, a tubercular tumor), which has caused symptoms whereby it has been recognized, must be so chronic that the onset of the meningitis is well marked. If, on the other hand, the symptoms of tumor have been so slight as to have been insufficient for its diagnosis, then the case is, for clinical purposes, primary Tubercular Meningitis. Masses of yellow tubercle are often found embedded in the brains of children dead of hydrocephalus acutus, whereof neither the prodromata nor the symptoms had led us to suspect the presence of anything more than the constant accompaniments of the latter disease.

It is uncommon for meningitis to supervene upon chronic phthisis in children; when this does happen, the new disease has been, so far as I have seen, easy of discovery; the more easy, the more chronic the precedent disease. Contrariwise, meningitis which occurs in the course of acute tuberculosis of the pulmonary form is mostly latent; when not so, very rapid in inducing death.

When tubercular peritonitis which has been diagnosed is complicated by meningitis, the latter is of the curtailed kind, apt to be overlooked, being, as it were, rather the harbinger than the cause of death—affording another proof of the truth of that aphorism which may be here repeated under another form, that the more tuberculosis has involved the health at large, the more obscure are the signs of a sequential meningitis; its premonitory and invasive symptoms have been anticipated. I have not known meningitis to supervene upon tabes mesenterica of such gravity as to have been a disease by itself.

The recognition of the occurrence of meningitis in the course of acute tuberculosis, which has been previously known to exist, depends greatly upon the degree to which the brain becomes implicated. Cerebral symptoms may be well marked (though shortened in duration, reduced to a week or less) even when they have been preceded by such grave symptoms, independent of the brain, as have not permitted us to doubt the existence of acute tuberculosis. Acute tuberculosis, not primarily cerebral, assumes for the most part one of two forms, namely, the typhoid form, or the pulmonary form. I cannot do better than quote Rilliet's description of the *typhoid* form when it precedes meningitis:—"In rare cases the invasion symptoms are more acute and febrile than usual, the skin is somewhat hotter, the pulse somewhat more frequent. The child complains of his head and belly at the same time; he does not vomit, but his bowels are obstinately constipated; he does not shriek, nor sigh, nor grind his teeth. The symptoms last from six to twelve days; fever continued; tongue covered with a thick fur; belly somewhat swelled and tender. The child is drowsy, but easily roused; answers sensibly; no photophobia; pupils natural; pulse regular, equal, 120 or more; no spots or sudamina anywhere; facies not that of hydrocephalus. This state is followed by the second (established) stage of meningitis." Let me add, that I have remarked, in such cases, the tongue to be pointed, with

a central white fur and red tip and edges, and the bowels to be spontaneously relaxed. The *pulmonary* form is more common. The child sickens with what seems to be a bad cold; rapid loss of flesh and strength; fever rather high. The catarrh continues, fever increases, dyspnœa and lividity ensue; the râles heard in the lungs become more and more abundant, fine, sharp, and metallic; the percussion note is high-pitched and hard, without losing in resonance. After two or three months from the beginning, symptoms of meningitis appear, more or less distinct, therein following the rule already several times laid down.

Tubercular Meningitis in the Adult.

It will be no small gain, if, by treating separately of Tubercular Meningitis as it occurs in the adult, a single reader be put upon his guard against supposing that acute hydrocephalus is a disease peculiar to childhood. As a matter of fact Tubercular Meningitis in the adult is not often diagnosed; yet were every one to study acute tuberculosis in the child, and then to transfer the knowledge acquired to the investigation of the diseases of the full-grown, there would probably be no special difficulty in the recognition of Tubercular Meningitis at any age.

Meningitis, when intercurrent in the course of chronic phthisis, is characterized by more or fewer of the following symptoms:—Headache, complained of for the first time; or, if previously present, greatly increased in severity; mostly, but not always, very painful; frontal. Vomiting is an early symptom, occurring in almost every case: vomiting in uncomplicated pulmonary phthisis is uncommon, except when brought on by the violence of the cough. Convulsions, occasionally, mark the onset of the meningeal disease. Delirium, of a quiet talkative kind, ensues. Sometimes the patients become speechless; they make ineffectual efforts to answer a question, or they look steadily at the speaker for a few moments, and then, without any expression of face, turn the head away. Numbness, paralysis, rigidity, of a limb, or of some other part, may be a very early indication of the affection of the brain. At the same time, the symptoms, so far as the chest is concerned, "abruptly improve or actually disappear." Then follow: comparative infrequency of the pulse, and irregularity both of pulse and respiration; squint; inequality of the pupils; the patient lies in a meditative, semi-unconscious state, then becomes more and more unconscious, while motorial symptoms, of any kind, ensue. The phthisis is not often advanced.

Primary Tubercular Meningitis is at least as common, in the adult, as secondary; and, like as in the child, the symptoms may be nearly wholly cerebral from the first, or may assume a typhoid character. In the latter case, the disease is rather acute tuberculosis than Tubercular Meningitis; the non-cerebral symptoms predominate, at least at first. In the other case, the symptoms do not differ from those previously described, as occurring in the child, either in their character or their order of appearance; headache, at the beginning, is mostly very severe, but is not always so. Vomiting, strabismus, diplopia, more or less loss of power over some part of the body, convulsions, numbness, dilated pupils, infrequency of the pulse, early delirium, the presence of any of these symptoms in an adult suffering from an acute illness, should suffice to put us on our guard: the physical examination of the chest does not often help the diagnosis. The duration of the disease is from eight to fifteen days.

Diagnosis.—The diseases which are confounded with Tubercular Meningitis may be divided for practical purposes into two classes: the first comprehending those diseases which simulate the earlier, and the second those which simulate the later, periods of meningitis. And it so happens that the resembling diseases of the first class are not attended, and of the second class are attended, by organic lesions of the nervous centres, or their appendages: this, speaking generally. The difficulty is greatest in the diagnosis of the earlier, the premonitory and invasive, periods of Tubercular Meningitis from the diseases of the first class; and the reason of the difficulty is obvious, namely, that the premonitory symptoms of Tubercular Meningitis are common to many diseases; so that the physician, full of a just dread of tuberculosis, and not wishing to be confronted by meningitis unawares, is continually suspecting tuberculosis when it is not present. To have treated incipient hydrocephalus slightingly is a mistake which, once made, is not readily forgotten; the patient's friends, at any rate, will remember the failure in prognostics. Loss of flesh going on steadily is a symptom to which it is wise to give the worst possible meaning. Repeated vomiting, in the child or the adult, occurring as a new symptom during a state of good health, or after a period of poorliness, is worthy of all our attention. Very carefully do we examine a child who has had a convulsion, lest it should be the first warning of the existence of incurable disease.

The *First Class* of diseases includes:—1. Simple Exhaustion; 2. Derangement of the Alimentary Canal; 3. Typhoid Fever; 4. Scarlet Fever and Smallpox;

5. Hysteria; 6. Simple Convulsions; 7. Pleurisy and Pneumonia.

1. Simple exhaustion of the vital powers (Morton's nervous atrophy) sometimes occurs so acutely and reaches such a pitch as to be mistaken for tuberculosis, or, indeed, actually existing Tubercular Meningitis. The exhaustion may be *primary:* a child, without any obvious cause, or perhaps in consequence of a slight catarrhal state or change of diet, loses its appetite, and therewith its flesh; becomes pale, languid, and restless; there are no distinct dyspeptic symptoms; the nurse fancies that the child is feverish; the pupils are large, and do not act very readily; a convulsion, or a series of fits, may occur —no other of the symptoms of meningitis being present. Wary in our prognosis, we submit the child to the test of treatment. We order pounded meat, milk, wine, or brandy, the aromatic confection, or a mixture of muriatic acid, cinchona, and chloric æther; the next day we shall be able to prognosticate much less dubiously; possibly, in the end, we may really have warded off Tubercular Meningitis. The exhaustion which is *secondary* to acute diseases, and especially to the longest acute disease, typhoid fever, is sometimes so great as to be mistaken for established hydrocephalus. It is chiefly in the houses of the poor that we see children, wofully mismanaged during their illness, wasted past belief. The alimentary canal ceases to perform a single natural function; the disgust for food is complete, the children are not even thirsty; forced to swallow broth, or food which is called light by a foolish metaphor, vomiting ensues; the child is somnolent, yet extremely restless, lies rooting with his head in the pillow, tossing from side to side, waving his arms in the air, or constantly passing his hand over one side of his head; incessantly whining, occasionally screaming, and, if old enough, complaining, when asked, of severe pain in the head; the tongue rolls from side to side, the lips are dry and peeling; the eyesight becomes dim, the somnolence deepens into unconsciousness, and the child dies. *Post mortem* we find the marasmus has invaded the brain; it is small and very bloodless; the pia mater is watery. The sketch is from nature, and the possibility of mistake is more than a mere possibility. The diagnosis will depend upon the previous history of the case, upon the character of the symptoms, and the order in which they have been developed.

2. Derangement of the alimentary canal.—i. *Acute Dyspepsia* causes symptoms which are almost identical with those of the earlier periods of Tubercular Meningitis. To take an example: a child of four years old, of a phthisical family, suddenly vomits several times, becomes feverish, complains of pain in his head, has no appetite; coughs a little; is very irritable, thick-looking, and heavy; greatly dislikes being touched; the bowels are confined. Occasional vomiting continues; in the course of a few days (measured by the thermometer, the pyrexia in such a case may last a week) the fever diminishes, the pulse becomes irregular and much less frequent. But, happily, at the same time, the child begins to look brighter, and to sleep better. It is important to bear in mind that during convalescence, even from so trivial a complaint as dyspepsia, the pulse of many children becomes actually infrequent and very irregular. To increase the difficulty, I have known one pupil to become larger than the other at the same time, and to remain so several days. A diagnosis off-hand is often impossible; there is no help for it but expectation—expectation of the active kind. The patient is visited more often, examined more minutely, and treated more carefully than if there were no doubt; at least he does not suffer, probably he derives benefit, from the uncertainty of the physician. ii. *Gastro-intestinal catarrh.*—A little child, who had lost appetite and flesh for several weeks past, has one day a fit, which lasts, say, a quarter of an hour; on the same day her bowels become loose; they remain so for a week, then she vomits several times. All this time there is more or less pyrexia; what heaviness there is, caused by the diarrhœa, tends to complicate the diagnosis. From one example the reader will learn all: diarrhœa, although no doubt an uncommon, is not an impossible, accompaniment of Tubercular Meningitis. When dentition coincides, the pain caused thereby is not always distinguishable from the headache of meningitis: nor must we attribute too much to dentition; I have known the canines to pierce the gum and Tubercular Meningitis to break out at the same time.

3. Typhoid fever resembles not so much Tubercular Meningitis as acute tuberculosis. But typhoid acute tuberculosis is sometimes immediately fatal by way of meningitis. With regard to children, the physician, when in doubt, is far more ready to suspect acute tuberculosis than typhoid fever. The difficulty is caused by the aberrant forms of typhoid fever which we meet with; cases with confined bowels, with an empty belly, with spots which are small, dusky, and hard to the feel, or even vesicular at the apex; cases which have the facies of acute tubercle; cases complicated with consolidation of one or other apex of the lung, and attended by universal mucous rhonchi. In the adult, however, typhoid fever is far more likely to be suspected

than acute tuberculosis. The practitioner, if fully aware that acute tuberculosis does occur in the adult, will not fail of making the diagnosis as soon as it becomes possible. Diarrhœa may accompany acute tuberculosis. Acute tuberculosis, with or without meningitis, sometimes greatly resembles typhus fever.

4. Smallpox and scarlet fever, both of which invade by vomiting, may be attended at the same time by very severe cerebral symptoms.[1]

5. Tubercular Meningitis sometimes at first simulates the symptoms of ulcer of the stomach, or may assume a quasi-hysterical form.

6. Simple convulsions cannot *per se* be discriminated from those which are precursory of Tubercular Meningitis.

7. The vomiting of incipient pleurisy and pneumonia, if accompanied by convulsions, as may be the case, is apt to divert attention from the chest to the head. But acute tuberculosis tends to cause inflammation of all the serous membranes; and, as a matter of actual experience, I have heard the friction sound of pleurisy in cases of Tubercular Meningitis; so that, on the contrary, attention must not be diverted from the head to the chest. The râle of lobular pneumonia is less general than that which we hear in some cases of miliary tubercle of the lung. In the lobular pneumonia of children, chlorides are often present in the urine while the body temperature is still high.

The *Second Class* of diseases which simulate Tubercular Meningitis includes: 1. Simple Meningitis; 2. Abscess of the Brain; 3. Thrombosis of the Sinuses of the Dura Mater; 4. Caries of the Atlo-Axoid Joint; 5. Arachnoid and Sub-arachnoid Hemorrhages; 6. Intracranial Tumors; 7. Hypertrophy of the Brain; 8. Essential Brain Fever.

1. By simple meningitis is meant meningitis which occurs totally unconnected with tuberculosis. Simple meningitis of the convexity is not a common disease. The symptoms are the same as are described under the head of Tubercular Meningitis affecting the convexity of the brain;[2] but the course of simple meningitis is more rapid. Convulsions and pyrexia in children; headache, active delirium, and pyrexia in adults, are the symptoms present early in the disease. Unconsciousness ensues in a day or two; the duration of the illness does not often exceed a week.

Sporadic cerebro-spinal meningitis (the membranes of the base and ventricles of the brain, and the sub-arachnoid space of the spinal cord, being especially affected), totally unconnected with tubercle, has been, in my experience, comparatively common. Cerebro-spinal meningitis is frequently quite a chronic disease; when acute, it resembles Tubercular Meningitis of the base very strongly.[1]

2. Cerebral abscess occurs under several different circumstances, and differs accordingly in clinical details. The diagnosis of Tubercular Meningitis continually opens up the whole field of diseases of the brain; the ability to distinguish them greatly depends upon a full and minute knowledge of their history. The reader will refer to the special articles, and make the necessary comparisons for himself. i. Cerebral abscess of pyæmic origin may be dismissed at once as never complicating diagnosis. ii. Cerebral abscess due to suppuration of the pia mater, going on so far that numerous large collections of pus are protruded into the brain substance, which disappears by rapid atrophy, so far as I have seen, does not modify the ordinary course of simple meningitis. iii. Cerebral abscess (due to disease of bones of the skull or not) differs in its symptoms according as pyæmia is combined with it or not. Abscesses merely pushed into the brain, inasmuch as they are not commonly combined with disease of the sinuses, cause symptoms which are altogether those of an intracranial tumor. Abscesses which are separated from the diseased bone by a layer of brain tissue (often greatly altered), and which are therefore presumed to have originated in the very midst of the lobe affected, are for the most part attended by pyæmia, the symptoms being complicated accordingly. But pyæmia does not always accompany even these non-peripheral abscesses, and then the difficulty of diagnosis from Tubercular Meningitis is very great, especially if we bear these facts in mind: first, that it is not uncommon for otorrhœa to concur with Tubercular Meningitis; and next, that external otorrhœa, in cases of cerebral abscess due to disease of the pars petrosa, may not set in until a week before death, and may have been preceded by the gravest symptoms of that intracranial otorrhœa which destroys the patient. The pulse of cerebral abscess is more persistently infrequent than that of Tubercular Meningitis.

3. Thrombosis of the sinuses of the dura mater, when secondary to neighboring inflammation, does not admit of diagnosis, unless there be present pyæmial symptoms and some obvious possible cause of disease of the sinuses; caries of the pars petrosa is by far the most common. Thrombosis secondary to debilitat-

[1] See vol. i. pp. 132 and 85.

[2] Page 817.

[1] The symptoms are the same as those of epidemic cerebro-spinal meningitis, to the article upon which subject the reader is referred, p. 296.

ing causes may be suspected if signs of disease of the brain follow a profuse diarrhœa or hemorrhage in a young child, but could hardly be distinguished from the simple exhaustion before described; while, on the other hand, I have known a decolorized softening thrombus to occupy the whole bore of the upper longitudinal sinus, to be attended by large sub-arachnoid hemorrhages, and to have caused no symptoms during life.

4. Caries of the atlo-axoid joint may cause such brain symptoms as to lead to a suspicion of the possible existence of Tubercular Meningitis. It is well, therefore, in a doubtful case, to examine the cervical region carefully, so as to discover any thickening and swelling of the soft parts.

5. Arachnoid hemorrhage, according to Legendre, may simulate Tubercular Meningitis.[1] This can be only in exceptional cases of the former rare disease.

6. An intracranial tumor at the base of the brain, of the soft sarcomatous kind, which approaches nearest to cancer in general appearance and in rapidity of growth, may cause symptoms which resemble those of Tubercular Meningitis so closely, that, for a week or two from the beginning of the disease, it may be impossible to arrive at a diagnosis. In the case of the tumor, the pyrexia ceases for several weeks before death, and the disease becomes of a more chronic character.

7. Local hypertrophy of the brain is sometimes attended by symptoms which, at first sight, are like those of Tubercular Meningitis. We discover afterwards that the hypertrophy is, comparatively, very chronic.

8. Every practitioner, from time to time, will come across an acute febrile disease, accompanied by symptoms which seem to point unmistakably to some affection of the brain: there being every reason to exclude the notion of suppressed exanthemata or analogous disorders. After one or several weeks of coma, delirium, severe headache, or whatever may have been the prominent symptom, the patient recovers, and we are left quite unable to say what has been the matter with him. To go more into detail, I could not do otherwise than narrate a series of cases which would differ from each other in most important points, and have nothing in common excepting pyrexia and brain symptoms. There is, generally, something wanting which makes us suspect that we have not to do with Tubercular Meningitis. Brain fever is as good a name as any whereby to designate these different anomalies; cerebral congestion, which is more commonly used, involves an explanation which is probably often wrong, and certainly never proved to be right.

[1] Rilliet and Barthez, ii. 259.

Morbid Anatomy.—I shall describe the morbid appearances of Tubercular Meningitis, in that order wherein they are brought under view during a post-mortem examination.

Separation of the calvaria is easily effected as a rule. Miliary tubercle of the most undoubted kind was once seen by me upon the inner surface of the *dura mater*. Slitting up the longitudinal sinus, a pale narrow clot is seen in the posterior half: sometimes the sinus is filled with fluid blood and loose coagula, sometimes with a large black shining thrombus. Removing the dura mater, the great *arachnoid* sac is found to be destitute of fluid; the membrane itself is dry, and, what is more, sticky to the finger passed over it. Scrape the surface gently with a scalpel and the sticky matter will be removed, minute in quantity, and puriform in appearance. Reddish serosity has been observed in the arachnoid sac by Senn and Becquerel; transparent or turbid serosity by Rilliet and Barthez. This serosity, was it observed before or after the brain had been removed? If after, the observations are quite valueless, unless indeed certain precautions were taken which probably were not. The ordinary unnatural state of the arachnoid may be looked upon as the sign of a feeble inflammation; similar stickiness is common in incipient pleurisy and peritonitis. Empis once found the arachnoid sac obliterated by old adhesions in a patient who had probably passed through an attack of acute tuberculosis long before. Still more to the point: in Tubercular Meningitis there is almost constantly present adhesion, more or less firm, of the opposed surfaces of the great longitudinal fissure, especially just above the corpus callosum.

The *pia mater* affords more unequivocal signs of disease. First, as to vascularity. Sometimes there is obvious hyperæmia of the whole convexity of the brain: it looks rosy; examined minutely, the fine vessels are seen to be injected everywhere; the body having lain on the back, the injection is nearly as well marked over the anterior as over the posterior lobes. More commonly, the capillaries are not much injected; what color the surface has being derived from large veins full of blood. Sometimes capillaries and veins both are emptied of blood, so that the brain has a most striking appearance, exactly resembling in color painters' putty. These differences depend for the most part upon the amount of pressure from within to which the surface of the brain has been subjected. Secondly, as to œdema. Excess of clear serosity is commonly met with in the meshes of the pia mater between the convolutions; sometimes the effusion is semi-opaque and lymph-like. Thirdly, as to tubercles. Examine the

membranes of the lateral region of the brain, corresponding in position to the temporal fossæ, and almost certainly miliary tubercles will be seen; not that they are absent elsewhere, but they are most common at the spots indicated; they are common at the bottom of the great longitudinal fissure also. These tubercles are beneath the arachnoid, often adherent to its under surface; those exposed to pressure against the skull are more or less flattened. Alongside the branches of the middle cerebral artery it is common to find a firm, grayish, semi-transparent material, which is probably confluent tubercle. Minute opacities of the pia mater are sometimes seen in the same region, most numerous by far in the neighborhood of the miliary tubercles, and possibly tubercular in nature, a sort of white "tubercular dust." Sometimes the tubercles are yellow at their centre, sometimes all of them are yellow throughout, remaining crude. The number of tubercles present may be very large; there may be none at all.[1] Raising the membranes from the surface of the brain, small portions of the brain substance adhere to the membranes so as to be removed with them; not that this is always the case; the difference depends upon the degree which the softening of the cortex has reached. The amount of vascularity of the pia mater and the degree of cortical softening are not always in direct proportion. The *convolutions* of the brain are more or less flattened, the intervening sulci narrowed. Proceeding to slice the brain, we perceive that the color of the cortex is increased in depth if the pia mater be hyperæmic, or diminished *vice versâ*, or remains natural. As a rule, the *centra ovalia* are anæmic, sometimes exceedingly so. Sometimes the texture of the whole brain is obviously softened. When we reach the *lateral ventricles*, they are found to be distended with fluid,—a colorless serosity, of low specific gravity, mostly clear, but becoming faintly turbid when agitated; slightly albuminous, containing chlorides and phosphates. The quantity of the fluid is from one to four ounces; sometimes more. In one case I found a drachm of fluid in the fifth ventricle, the septum lucidum being everywhere perfect. The foramen of Monro is dilated. The lining membrane (ependyma) of the ventricles is toughened, sometimes obviously opacified in places, especially in the sulcus between the corpus striatum and the thalamus opticus. Viewing the surface of the lining membrane sideways, we see that it looks as if it had been sprinkled with the finest dust. It seems probable that this condition is mostly due to small heaps of cells, a commencing suppuration of the lining membrane;[1] sometimes the dusty look, in part at least, is due to a minutely wrinkled state of the ependyma, resulting from the stretching it has previously undergone. Occasionally we see larger granulations than those described, grayish elevations, something midway, in every respect, between the sandy specks and miliary tubercles. The whole ependyma down to the fourth ventricle may be thus granular, or this sanded appearance may be quite absent. The vessels of the ventricles, the choroid plexuses and veins of Galen, with their tributaries, are sometimes obviously full of blood, but more often not so, and sometimes almost empty, the plexuses being quite pale. Softening of the cerebral matter beneath the ependyma is almost always found; the septum lucidum and under surface of the fornix are reduced to a pulp; the corpus callosum, walls of the posterior cornua, and other parts are often similarly affected. The question naturally arises,—What are the causes of these lesions of the ventricles—the dropsy and the softening? We cannot suppose that the brain substance will soften by passive imbibition of fluid; were it possible, there would be no reason why softening should not occur in health. But is the fluid forced by its excess into the brain substance? This is not so, because the ventricles are sometimes nearly empty when their walls are thoroughly softened. Mechanical congestion might conceivably be the common cause of the dropsy and the softening; but, in the great mass of cases, it is impossible to discover any impediment to the return of blood from the ventricles. The blood may be made to flow from the veins of Galen back into the straight sinus with perfect ease; moreover, as stated above, the plexuses are often quite pale, and the veins nearly empty. It would be difficult to explain the effusion of serosity into the pia mater, and the cortical softening, by mechanical congestion. Inflammation will account for all the conditions. We have already seen that the ependyma presents an appearance common in inflammation of surfaces,—namely, proliferation of cells. The connective tissue which underlies the epithelial layer of the lining membrane is gradually lost in the neuroglia or interstitial non-nervous tissue of the brain. And hence, inflammation of the ependyma leads to effusion of serosity both in the ventricles and into the brain matter; the softening being rendered complete by the mal-nutrition which ensue in consequence of the more or less arrested capillary circulation. To proceed: the velum interpositum is natural, or œdematous, or

[1] See the article on Scrofula, in this volume.—H.]

[1] Löschner and Lambl: Aus dem Franz-Josef Kinderspitale, 1860; S. 82. Prague.

thickened and somewhat opacified; studded, it may be, here and there with miliary tubercles. The condition of the *third ventricle* resembles that of the lateral ventricles: the distension is ordinarily less because of the resistance of the thalami optici: in front of these masses the dilatation sometimes goes so far as to expose the pia mater of the base: the commisura mollis is often more or less torn and speckled with capillary hemorrhages. The *fourth ventricle* also is distended. I have several times examined the cerebro-spinal opening *in situ*, and have always found the membranes about it perfectly healthy. When the *spinal canal* is laid open before the calvaria has been removed, to puncture the sub-arachnoid space (internal arachnoid of Hilton) causes the escape of a certain quantity only of fluid, merely the excess in the spinal canal; when the calvaria has been removed before the spinal sub-arachnoid has been touched, the cerebral ventricles can be drained completely by opening the membranes of the cord. The spinal internal arachnoid is distended with fluid, especially around the cauda equina. I have never observed any other morbid condition within the spinal canal, but then it has been examined in a minority of cases. Removing the brain from the base of the skull, we occasionally find adhesions of the two surfaces of the great arachnoid about the circle of Willis. The membranes at the *base of the brain* are sometimes greatly injected, sometimes much less so. Miliary tubercles sometimes swarm in the Silvian fissures, interpeduncular space, round the crura cerebri, and on the top of the cerebellum: ordinarily they are not in very large numbers; occasionally there is only a tubercle here and there; still more rarely no unquestionable tubercles can be found. Sometimes the tubercle is of the crude yellow variety. Other parts of the membranes of the base than those mentioned sometimes present tubercles. Besides being tuberculized, the membranes (*i. e.* pia mater, sub-arachnoid) undergo those changes which have caused the name of meningitis to be given to this disease. The meshes of the pia mater are filled with serosity (clear or turbid), or with lymph-like material, or with puriform: sometimes the membrane seems to be merely thickened, toughened, and opacified. The inflammation of the pia mater is most marked in the interpeduncular space, but tends to spread forwards along the optic and olfactory nerves, sideways into the Silvian fissures, and backwards round the crura cerebri on the upper surface of the cerebellum, or right over the pons and as far back, it may be, as the medulla oblongata. There is no proportion between the amount of tuberculosis and of meningitis. Softening of the brain cortex is usual; the under surface of the anterior lobes, and the under surface of the cerebellum, are affected with especial frequency. Softening of the optic commissure and of the smaller nerves is mostly found. The sinuses at the base present nothing abnormal.

The foregoing description applies to the commoner form of Tubercular Meningitis, in which the membranes of the base of the brain are involved. In the less common form of the disease, limited to the convexity of the brain, the morbid changes correspond. The pia mater of the affected part is infiltrated with puriform lymph; the membranes of the base being spared: there is no excess of serosity in the ventricles.

It is obvious to the naked eye that the tubercles are everywhere in the closest connection with the bloodvessels, especially the small arteries. And, in fact, it is in the sheaths of the vessels that the tubercles are formed. Both by their position (being seated upon the vessels), and by their structure, recent miliary tubercles of the pia mater have the closest resemblance to the Malpighian follicles of the spleen: indeed the similarity is so great that when the same bodies, which are recognized elsewhere as being recent miliary tubercles, are found in the spleen, it is often impossible to distinguish them from the Malpighian follicles. There can be no reason for doubting that the meningeal tubercles are not of the same nature as miliary tubercles elsewhere. It is true that the lymphatic character of the vascular sheaths is very well marked in the brain, but the vessels of many other parts are similarly constructed, to say nothing of the lymphatic tissue which exists beneath the serous and mucous membranes, and in other places. According to Dr. Bastian, the new formation in the sheaths of the vessels of the velum interpositum is sometimes so abundant as to cause the vessels to be obstructed.

Accidental Lesions.—1. Masses of yellow tubercle are often met with in examining the brains of children dead of Tubercular Meningitis. Sometimes the tubercle is softened. 2. Capillary hemorrhage coincides with the softening of the cerebral matter, when the softening has reached a certain point. Most frequently seen in the soft commissure, hemorrhage sometimes occurs in other parts, the brain proper, the pons Varolii, &c., leading to utter disorganization of the tissue. 3. I have seen miliary tuberculosis of the brain substance carried almost as far as can be conceived possible—a whole hemisphere of the cerebrum so much softened that it was easy to wash all the brain matter away; which done, there remained a close network of injected and dilated capillaries studded everywhere with mi-

liary tubercles. The meningitis in this case ran a very rapid course. 4. Meningeal apoplexy, and a decolorized thrombus of the superior longitudinal sinus, I observed in one case of tuberculosis of the meninges, unattended with obvious inflammation of them.

Other Organs. — Tuberculosis of the lungs, liver, spleen, lymphatic glands, kidneys, and ocular choroid, concurs with the brain disease. The tubercle is mostly miliary, sometimes yellow, crude, or softened. A girl of four[1] died on the tenth day of Tubercular Meningitis without prodromata; *post mortem* we found numerous miliary tubercles in the pia mater, great ventricular effusion, very little lymph at the base of the brain, and absolutely no tubercle in any other part of the body (which was carefully examined), excepting a small mass of cheesy material in each lung. Tubercular ulcers of the intestines are often present: also intussusceptions, easily reduced. The children will have frequently preserved a large amount of subcutaneous fat.

PROGNOSIS AND TREATMENT. — The prognosis must always be unfavorable; and when the disease has passed beyond the invasion period and has become established, recovery may be deemed hopeless. In the latter case, if we reckon twenty-one days from the invasion symptoms we shall probably cover the fatal termination. When the disease does not pass beyond what seemed to be the invasion symptoms of Tubercular Meningitis, the patient's recovery is not always complete; this has been already dwelt upon: moreover, a second attack sometimes ensues.

What then, is to be done by way of prevention of the disease? The prophylactics and ordinary hygienics are the same—animal food, change of air, warmth to the surface, moderate exercise; to which may be added cod-liver oil and cinchona. The bad prognosis of confirmed Tubercular Meningitis does not belong to acute tuberculosis. No doubt many persons recover from acute tuberculosis: knowing this, any patient suffering from what is possibly acute tubercle should be treated very carefully, so as, if possible, to stop the disease and prevent affection of the brain. If the patient be seen during the invasion period, he should be put into a dark and quiet room; be carefully and regularly fed; symptoms should be treated; constipation relieved; a convulsive state diminished by full doses of bromide of potassium. There can be no doubt concerning the powerful depressing influence exerted by continuous cold applied to the head: this means should be therefore employed if the disease be seen in its earliest stage. Cod-liver oil may be tried at the same time. Later on there is not much that can be done. Sufficient liquid food should be given to the patient, by means of a syringe placed between the teeth, if need be. Leeches, active purging, blistering, and such-like measures, will rather hasten the advance of death. It is best not to shave the head unless it be necessary to apply cold. If the corneæ begin to ulcerate, it is as well to keep the eyelids closed by means of a little sticking plaster.

[Since the discrimination in the early stage, between simple and Tubercular Meningitis, is not always easy or certain, the good results of active treatment in the former justify the moderate and careful use of similar measures, leeching, purgation, and blistering, at that period. After paralytic symptoms appear, all such treatment should be discontinued. The following is abstracted from an account of the case of a man, aged 23, reported by M. Dujardin-Beaumetz in *Le Progrès Médical*, 1879, p. 208:—

"For five days he had persistent cephalalgia, which appeared about to usher in an attack of typhoid fever; coma followed, and from the sixth to the tenth day the symptoms of Tubercular Meningitis, notwithstanding which the man recovered. The patient's antecedents were very unfavorable to this result, his father and mother having both died with phthisis. Examination of the eye-ground had shown exudation with tuberculous granulations. The treatment consisted in blisters, calomel, and ice to the head. M. Beaumetz's paper was read before the Société Médicale des Hôpitaux, and several other cases were brought forward by members showing a similar arrest of the disease in question."—H.]

[1] Under the care of Dr. West in the Children's Hospital.

CHRONIC HYDROCEPHALUS.

By J. Spence Ramskill, M.D.

This disease is a real dropsy occurring within the cranial cavity. The fluid may be collected in the sac of the arachnoid or in the ventricles of the brain, beneath the arachnoid membrane. The affection may be congenital or acquired. When congenital, it is generally, but not invariably, due to an arrest of development of the cerebral mass, although even in such cases the dropsy has been regarded by Rokitanksy and Vrolik, whose opinion is quoted and endorsed by Dr. West,[1] as not a mere passive dropsy, but as the result of a slow kind of inflammation of the arachnoid, especially of that lining the ventricles, which may have existed during life. Such inflammation may also attack the child after its birth, and "each year," says Dr. West, "leads me to estimate more highly the share of inflammation of the lining of the ventricles in the production of Chronic Hydrocephalus. Acquired hydrocephalus begins to show itself about the period of the first dentition." According to Dr. West (p. 124), out of 54 cases, 18 of which came under his own observation, some indications of the disease were obtained in 50 before the child was six months old; in 14 the symptoms existed from birth, and in 21 more they appeared before the completion of the third month. In some rare cases, the disease attacks children seven, eight, or nine years old, who until then had seemed to be free from all cerebral complaint. In some extremely rare cases, this affection has been known to attack persons of advanced life. Sir Thos. Watson[2] cites several instances of the kind, one of which occurred under his own observation. A young and distinguished lawyer of his acquaintance had one or two attacks of rather sudden loss of consciousness, while engaged in the Court of Chancery; by degrees he became dull, stupid, forgetful, and at length insensible. In this condition he died. A large quantity of serous fluid was found distending the ventricles of the brain. No other alteration could be detected. A case of Dr. Baillie's is quoted by the same author, the patient being a man fifty years old. The celebrated Dean Swift died of this complaint at the age of seventy-eight, three years after the commencement of the disease.[1]

Gölis also mentions three instances in which this affection began in advanced life: two of the patients were about seventy years of age; the third, who was a physician at Vienna, likewise died in the decline of life, having suffered under the disorder for ten years. When hydrocephalus shows itself some time after the birth, it is generally accounted for by the presence of a tumor (cancer, tubercles, or cysts). The dropsy in such cases is produced, as was pointed out by Dr. Whytt long ago, by the same mechanism as ascites in cases of schirrus of the liver, of the spleen, or of the pancreas. Any deposit compressing the veins of Galen, which bring back the blood from the ventricles of the brain, is sure to lead to accumulation of serosity within those ventricles.

Dropsical effusion within the sac of the arachnoid is sometimes the result of a former hemorrhage into that cavity, as pointed out by Legendre, and supported by Rilliet and Barthez. In some very rare cases, Chronic Hydrocephalus seems to be a result of the acute disease. Two cases of this kind are recorded by Rilliet, one in his work on "Diseases of Children," p. 162, and the other in the "Archives générales de Médecine," for Dec. 1847. Dr. West also relates a case in which the first link in the chain of morbid processes seems to have been an injury to the head; the child, when five months old, having fallen out of the arms of the person who was nursing her, and on the same day she had a fit, and remained stupid and senseless for hours.

Anatomical Characters.—1. *Ventricular Hydrocephalus.* — The quantity of fluid varies from a few ounces to a few pounds. In a case mentioned by Trousseau,[2] the head measured a *mètre* (39·3 inches) in circumference, and about thirty pounds of fluid were found in the ventricles. The same author cites another case from Franck, in which the fluid amounted to fifty pounds.

As a necessary consequence of the ac-

[1] Diseases of Children, p. 121.
[2] Practice of Physic, 4th edit. p. 464.

[1] Practice of Physic, 4th edit. p. 464.
[2] Trousseau, Clinique Médicale, 2e édit. p. 247.

cumulation of fluid, the ventricular cavities are considerably enlarged, the openings through which they communicate with one another are considerably dilated, although in some instances, from the pouring out of lymph, these apertures may get closed, and the fluid may therefore accumulate in one part more than another, producing an unsymmetrical enlargement of the head. Thus, Vrolik[1] has related the case of a young man who died from Chronic Hydrocephalus at the age of twenty, and in whom a false membrane had occluded the foramen of Munro through which the two lateral ventricles communicate.

The walls of the dilated ventricles may be of normal consistence, or even of greater consistence than normal. Rilliet and Barthez state that they have been able, in some cases, to dissect the condensed mass into several layers. In other cases the walls felt softer, and œdematous for some little distance. The brain mass above the ventricles becomes thinned and unfolds itself. The convolutions are flattened out, and the sulci between them disappear. The cerebral substance looks pale and anæmic. In some cases it happens that the commissures of the brain yield, and that the whole, or a portion of the fluid which it contains, escapes into the cavity of the cranium. This appears to have taken place in the well-known case of Cardinal, whose skull contained seven or eight pints of fluid, while "the brain lay at its base with its hemispheres opened outwards like the leaves of a book."[2] When the accumulation of fluid has resulted from inflammation of the membrane lining the interior of the ventricles, that membrane is found thickened and rough, and in some cases in a granular condition.

2. *Intra-arachnoid Hydrocephalus.*—When the result of hemorrhage into the arachnoid sac, the fluid is found more or less yellowish in color, and may be even more or less mixed up with thin, serous blood. When it has been poured out to fill up the vacuum in the skull due to defective development of the brain, it is perfectly limpid and clear. The sinuses of the dura mater in this, as in the preceding form of hydrocephalus, are either empty, or are found to contain blood, both liquid and coagulated. The fluid of hydrocephalus, when tested by heat and nitric acid, is found to contain albumen; chloride of sodium, soda, and traces of salts of lime and potash have also been found in it. Urea was detected by Dr. Bostock in his examination of the fluid found in Cardinal's head.

[1] Traité sur l'Hydrocéphalie interne. Amsterdam, 1839.

[2] Bright's Reports, vol. i. part i. p. 433.

Condition of the Bones in Chronic Hydrocephalus.—They are generally found to be considerably thinned, and transparent; if the union of the sutures has been completed, the bones are found to be less firmly united than usual, with less dovetailing; and there are numerous ossa triquetra found in the lines of the sutures. In some cases, the bones have been found of normal thickness, and in rarer ones they have been of greater thickness than normal (Rilliet and Barthez), hard, compact, and resisting.

Symptoms.—When the disease is congenital, signs of cerebral disturbance manifest themselves very soon after birth. There may be either strabismus and rolling of the eyes alone, soon followed by gradual enlargement of the head, or convulsions recurring pretty frequently may set in.

According to Dr. West,[1] "enlargement of the head is by no means invariably the first indication of Chronic Hydrocephalus. In twelve out of forty-five cases, fits, returning frequently, had existed for some weeks before the head was observed to increase in size; in six, the enlargement of the head succeeded to an attack resembling acute hydrocephalus; and in four other instances it had been preceded by some well-marked indication of cerebral disturbance. In the remaining twenty-three cases no distinct cerebral symptoms preceded the enlargement of the head." Failure of nutrition is almost invariably present, although Rilliet and Barthez assert that "the nutritive functions are as a rule well performed in hydrocephalic children, unless they be in an advanced stage of tubercular cachexia, or chronic intestinal catarrh. Except such cases, the children are plump and well nourished, and even have sometimes an abundance of fat which is certainly morbid."[2] The cases, however, in which nutrition is unaffected, form the exception, not the rule. The child sucks well, voraciously even, and yet does not grow: he may even waste. His bowels are generally constipated, and his motions are unhealthy. The gradually increasing head soon attracts notice, and the peculiar physiognomy and aspect of a hydrocephalic child soon develop themselves. The fontanelles enlarge, and the anterior one is seen often to pulsate, and grow tense and prominent; and at such times there is heat of the head, and the child is more restless than usual. The sutures of the head widen, and the head by degrees assumes a globular shape. The forehead is round and prominent, the orbital plates of the frontal bone gradually become slanting, and the eyeballs become half

[1] Diseases of Children, p. 121, 5th edit.

[2] Mal. des Enfants, p. 161.

hidden under the lower eyelid, so that the cornea cannot be seen until this is depressed. The parietal bones being pushed outwards and their edges being last to ossify, there is a considerable increase of the sagittal suture, whilst the occiput is driven downwards and backwards, in some cases to such a degree as to be almost horizontal. On applying the hand over the opened sutures and fontanelles, a distinct sensation of fluctuation is perceptible. The hair grows very scantily on the head, on which very large distended veins are seen to ramify. The face is small, and contrasts remarkably with the large size of the head; and looks triangular, with the apex of the triangle at the chin. The child's expression is dull and stupid, and he has a very aged look; he cannot sit up, or hold up his head, but lies down constantly. As the fluid continues to accumulate, and the disease progresses, the sight becomes impaired, and is completely lost after a time; the eyes are bright and shining, but restless and oscillating. Hearing is as a rule preserved much longer, but is lost at the close of the disease. Paralysis often sets in; contractions and rigidity of the limbs and trunk are not very rare, according to Rilliet and Barthez, particularly in very young children (p. 160). Occasional attacks of laryngismus stridulus are not infrequent, and they may even come on before there is much enlargement of the head (West). That form of Chronic Hydrocephalus which results from the transformation of a cyst, the result of hemorrhage into the arachnoid sac, may be recognized, according to Legendre, "by its being never congenital; by generally beginning about the tenth month, that is to say, about the time when the teeth begin to appear. The head, indeed, enlarges gradually, but does not acquire so large a size as in internal hydrocephalus; while, lastly, it is always preceded by convulsions, or by some other form of active cerebral disturbance, which marks the date of the occurrence of hemorrhage."[1] At best, however, the diagnosis can be but hypothetical. When hydrocephalus becomes developed after the sutures are united, the bones, being subjected to pressure, become thin, and in some cases the sutures have been known to give way. Such cases are spoken of by Rilliet and Barthez, who also quote from the *London Medical Journal* (for 1790, p. 56) the case of a child who at the age of nine years, and eleven months before his death, became affected with chronic cerebral symptoms. Nine months and a half after the first manifestation of the disease, the sutures of the cranial bones, chiefly the coronal, began to open. At the time of his death the distance between the edges of the coronal suture measured half an inch, and at the spot where the lambdoidal joins the sagittal suture there was a marked opening, so that the occipital bone was completely free.[1] As a rule, however, when hydrocephalus begins after the sutures are united, the head does not enlarge considerably, although it may do so in some rare instances, as in a case mentioned by Rilliet and Barthez (p. 165), of a child nine years old, who from the age of eight exhibited the symptoms of hydrocephalus, and whose head became enormously enlarged in spite of the ossification of the fontanelles. The size of the head in Chronic Hydrocephalus varies considerably; it has been known to measure two and even three feet in circumference. In the Museum of the Faculty of Medicine of Paris there is a hydrocephalic skull which measures 39 inches round. The shape of the head is generally globular and flat at the top, but in some rare cases it is conical, shaped like a sugar-loaf.

The termination of the complaint is generally in death, which occurs either from some intercurrent affection, hydrocephalic children being always weakly and unable to resist disease, or from an attack of laryngismus stridulus, or from convulsions due to passing congestion of the meninges, or lastly from gradual exhaustion, from positive asthenia. The disease extends at least one or two years, but it may last from four to ten years. Cases have even been recorded of individuals living to an advanced age who had been hydrocephalic from infancy. Thus Dr. Bright's patient, Thomas Cardinal, lived to nearly thirty. Franck, cited by Trousseau,[2] speaks of two individuals, the one aged seventy-two, and the other seventy-eight, who had been hydrocephalic from infancy. Strictly speaking there is no cure of the complaint, but merely an arrest of its progress. Fluid may be no longer poured out, but that which has been already effused is not absorbed. The sutures and fontanelles ossify and close, and a good many ossa wormiana are then found along the lines of union; these are like nuclei for the formation of bony matter. In some instances it has been said that a real cure takes place; that there is increased activity of the nutrition of the brain, producing hypertrophy of that organ, the fluid being absorbed and new matter deposited in its stead (Otto).[3]

[1] Legendre, Recherches Anatomo-pathologiques, p. 135.

[1] Sir Thos. Watson (p. 464, 5th edit.) also cites two similar cases, one from Dr. Baillie—the patient was a boy, seven years old; and the other from Dr. Yeats' work on Hydrocephalus—a boy nine years old.

[2] Clinique Médicale, p. 247.

[3] In Rokitansky's Pathologische Anatomie, 1st edit. vol. ii. pp. 749–769.

Such cases, however, must be quite exceptional, and the rule is that the fluid is unabsorbed and remains in the cranial cavities. The patient's intellect and senses are not perfect it is true, but are still sufficient to enable him to perform the ordinary duties of life, although he is apt to be fretful and irritable, and somewhat childish in his ways.

Diagnosis.—1st. Congenital hydrocephalus has to be diagnosed from encephalocele and perforating fungus of the dura mater. In encephalocele the feel of the swelling is doughy and elastic, not fluctuating; it is local and not general, and it is not transparent. In cases of fungus of dura mater, which has perforated the cranium at birth, the general size of the head is not affected, the perforated spot can be easily detected, and it is over the central parts, not near the sutures or fontanelles; the mass feels doughy, elastic, quasi-erectile, and when it is compressed, symptoms of irritation are produced. Acquired hydrocephalus has also to be distinguished from a merely excessive development of the head apart from any disease. The absence of all cerebral symptoms is sufficient in such cases to establish the diagnosis. Sometimes hydrocephalus may be suspected where none exists, because of the disproportion between a small, emaciated, triangular face and largely developed skull. Rilliet and Barthez candidly confess to an error of this kind. 2d. From abnormal thickening of the bones of the skull, which sometimes obtains in rickets. In such cases the diagnosis may be made by a careful inspection and palpation of the bones of the head. The development of the skull is not uniform; it seems as if flat bumps had been superadded to the centre of the frontal and parietal bones, and we can detect with the finger the exact spot where the bone begins to thicken.[1] The swelling of the articular ends of the bones of the limbs, which is characteristic of rickets, will at once awaken suspicion, for rickets and hydrocephalus do sometimes coexist. 3d. From hypertrophy of the brain. This is an exceedingly rare affection, in which the head enlarges without exhibiting any symptoms at first; and when these show themselves after a time, they run an acute course which soon terminates in death.

Treatment should be persisted in for a long time, without the adoption of any violent measures. The plan recommended by Professor Gölis, of Vienna, seems to be one of the best. He advises the head to be shaved, and a scruple or two of mercurial ointment, mixed with ointment of juniper berries, to be rubbed on the scalp twice a day. The child should wear a woollen cap, to prevent the risk of the perspiration being checked by the cold air. From a quarter to half a grain of calomel should be administered twice a day; if it purges too much, the inunction of mercurial ointment must be alone employed. This treatment is to be persevered in for thirty or forty days, when, if there be some improvement, the remedies may be gradually diminished; but the cap is to be worn after the inunction has been discontinued. If there be no marked improvement after six or eight weeks, some diuretic, acetate of potash or squills, for example, may be added; and a couple of issues may be inserted in the occiput. Blisters to the nape of the neck may be advantageously substituted for these. Whenever there is heat of head, and the child grows fretful, restless, and irritable, a couple of leeches behind the ears will be found of service. Gölis affirms that under this plan of treatment he has known the circumference of the head decrease by half an inch, or an inch, in a period of six weeks to three months. He thinks that convalescence, when once begun, may be accelerated by small doses of quinine. Dr. Gower's plan of treatment, which is said to have been successful in many cases, consisted in giving ten grains of crude mercury mixed by rubbing with about a scruple of manna and five grains of fresh squills. This was one dose, and it was to be repeated every eight hours. The medicine induced great prostration of strength, loss of flesh, and profuse action of the kidneys, without ptyalism.

Chronic Hydrocephalus has been treated by two *mechanical* means; by bandaging and tapping. Bandaging, which has been particularly advocated by Mr. Barnard, of Bath,[1] seems to be chiefly useful in pale flabby children, whose bones are loose and yielding: strips of plaster, about three-quarters of an inch wide, are made to encase the head; they are to be applied circularly, transversely, and diagonally. Trousseau, who was at one time an advocate of this plan, has given directions for properly carrying it out in the *Journal de Médecine* for April 1843. But this eminent practitioner had good reasons for changing his views, and did not latterly advocate this plan. In his *Clinique Médicale*, second edit. p. 250, he says that he has given it up completely, since a child aged five months, whom he treated in that way, died suddenly on the fluid making its way through the ethmoid bone and the nasal fossæ.

The second mechanical mode of treatment, namely, by tapping the skull, and

[1] Rilliet and Barthez.

[1] Cases of Chronic Hydrocephalus, &c., by T. H. Barnard. London, 1839.

letting out the fluid accumulated in its interior, has been opposed by such men as Gölis, Richter, and Dupuytren. Dr. Conquest has been the greatest advocate of the operation in this country, and a paper on the subject may be found in the *Medical Gazette* for March, 1838. Sir Thos. Watson gives the sanction of high authority to the procedure; and although Dr. West speaks rather doubtfully on the subject, yet he does not regard the operation as unjustifiable in some cases; when, for instance, there is good ground for believing that the hydrocephalus is external, or where the enlargement of the head has not been attended by indications of active cerebral disease. The operation itself does not seem to be attended with any very great immediate risk of life, if performed carefully. The best spot for puncturing the skull is about an inch, or an inch and a half, from the anterior fontanelle, near the edge of the coronal suture, taking care to avoid the longitudinal sinus, and some of the large veins which empty themselves into it. The trocar should be a small one, and it should be introduced perpendicularly. The fluid should be let out very slowly, a few ounces at a time, and the skull supported by bandages, both at the time and subsequently. If the child turns pale and faints, a few drops of ammonia, or of brandy, will be found useful. If any inflammatory action should be set up a day or two after the tapping, cold lotions to the head and leeches behind the ears, and small doses of mercury, will be required. The administration of iodide of potassium internally, and of iodine lotions to the scalp, has been advocated by Trousseau; and, when more active measures may not appear justifiable, some hope in the way of arrest of the further progress of the disease may be entertained from the use of these remedies. In addition to them I have found great assistance from the use of syrup of iodide of iron, cod-liver oil given in small doses, and bone-earth. The dose of cod-liver oil should be limited to a teaspoonful, the object being not to increase, but to improve nutrition. The iodide of iron is usually very well borne by hydrocephalic children, unless there be a tendency to congestion, or to inflammatory action. Amongst the children of the poor the combination of the oil and the syrup of iodide almost always gives the most satisfactory results. Bone-earth mixed with fine sugar, administered with every meal, sprinkled on the surface of milk, or of other food, has appeared to me to possess a tonic action beyond that possessed by any chemical compound of the phosphates. It has an increased value in cases associated with rickets or imperfect nutrition of the bones; and a diet, of which lentil flour forms part, has appeared to me highly advantageous. Good food, given in limited quantities, and at small intervals, is absolutely necessary, and I object to the use of stimulants. When the patient appears faint and languid, beef-tea will prove a better and more permanent stimulant than wine or ammonia. The usual hygienic measures should be adopted,—warm clothing for the extremities, the head being kept cool. Bathing with sea-water is useful, taking care that the limbs are rubbed to produce warmth and redness of the surface after the bath. The patient should, if possible, spend the summer months on the sea-coast, or in some elevated district, and he should almost live in the open air.

MENINGEAL HEMORRHAGE.

By J. Spence Ramskill, M.D.

The term Meningeal Hemorrhage is used to denote extravasation of blood either into the cavity of the arachnoid, or beneath this serous membrane, and into the meshes of the pia mater. Hemorrhage occurring between the dura mater and the bones of the cranium is extra-meningeal; and as it is usually the result of a blow or a fall on the head, in which case it often takes place on the side opposite to that of the injury, by *contre-coup*, it comes within the province of the surgeon and not of the physician.

In his valuable work on diseases of the brain, Abercrombie, at p. 238, relates a most curious instance of "extravasation in a cyst, formed by separation of the laminæ of the dura mater, from rupture of the middle meningeal artery." The patient, a man aged forty-eight, about the 12th of November, 1814, was assisting a neighbor to carry a heavy load up a

high stair, when he felt a sudden attack of headache. He was from that time troubled with headache and giddiness, increased by stooping; and after these symptoms had continued rather more than a fortnight, he became sensible of some imperfection of vision. When seen by Dr. Gairdner, on the 2d of December, he complained of violent headache. The pulse was forty in the minute, and feeble. The pupils were at this time sensible to the light, but after a few days became insensible. He sank very gradually into coma, without any remarkable symptom, and died on the 13th. *Inspection:* On the left side of the head, a cyst was found in the course of the middle meningeal artery, occupying the region of the lower part of the parietal and upper part of the temporal bone. It was formed by a separation of the laminæ of the dura mater, and contained about four ounces of coagulated blood. The portion of the dura mater forming the cyst was considerably thickened and very vascular. There was a depression on the surface of the brain, corresponding to the cyst, and the ventricles contained a considerable quantity of serous fluid. There was no other morbid appearance.

True Meningeal Hemorrhage is an affection which is found generally at the two extremes of life, in infancy and old age. It occurs in new-born infants, after severe and protracted labors, and, from the discoloration of the skin attending it, is often mistaken for cyanosis. It may be distinguished from this malformation, however, by the absence of cardiac murmur, which is almost always present in the latter.

The blood may be diffused, as we have said, into the arachnoid sac itself, or under it, and in the pia mater. A third variety has also been described, in which the blood is said to be effused between the dura mater and the arachnoid; but recent researches have made it more than doubtful that the extremely delicate visceral layer of the arachnoid can be separated without being torn from the dura mater; and Baillarger[1] has shown that the error arose from the rapid formation of a false membrane resembling the arachnoid, which isolated the effused blood. An instance of this variety, of traumatic origin, is related by Sir Robert Carswell. A man fell on his head, was stunned for some little time, but afterwards went to work as usual. Three weeks afterwards he applied to a hospital, but was refused admission because he had no fever, and he was suspected of malingering. On leaving the hospital he drank some hot spiced wine on his way home, became delirious, and died in thirty-six hours. A post-mortem examination showed six ounces of blood effused between the dura mater and the arachnoid, part of which was in a coagulated and part in a fluid state.

Two cases of the same form of hemorrhage, but of spontaneous origin, are given by Andral in his "Clinique Médicale," occurring in two men, aged respectively seventy and seventy-three. Other instances are recorded by Rostan, Blandin, Ménière,[2] Cruveilhier.[3]

Hemorrhage into the arachnoid cavity.—This may be traumatic or idiopathic. When the latter, the blood accumulates in the cavity of the arachnoid, and is equally diffused over the brain, not accumulated at the base. It is generally more fluid anteriorly, and more coagulated posteriorly. The arachnoid and dura mater are colored by imbibition. After a time, the blood is enveloped in a pseudo-membrane, and in old cases cysts are found with yellowish contents and smooth walls; in some cases the two layers of the false membrane are found agglutinated, leaving no doubt as to the possibility of a perfect cure occasionally being made. With regard to the source from which the blood comes, there exists a discrepancy of opinion. According to most authors, the extravasation results from the rupture of a bloodvessel, but Prus[4] maintains that intra-arachnoid hemorrhage is always the result of exhalation. Hemorrhage by exhalation is, however, a pathological phenomenon not accepted nowadays, and for which cases of molecular rupture of bloodvessels used to be mistaken.

SYMPTOMS.—In persons of advanced age, there are sometimes certain premonitory symptoms observed in the shape of drowsiness, vertigo, general malaise, diminution of motor power, loss of speech, &c. All these symptoms do not show themselves in the same case, but one or other of them is generally present. Cephalalgia is a symptom which is usually met with in old people when hemorrhage has occurred.

Some authors regard the false membranes as being of inflammatory origin, and as the first step in the morbid process — the hemorrhage being only the second.[5] There may be such cases,

[1] Baillarger: De Siége de quelques Hémorrhagies des Méninges: Thèse. Paris, 1837.

[1] Rostan: Recherches sur le Ramollissement du Cerveau, p. 396.

[2] Anatomie Topographique. Paris, 1834.

[3] Anatomie Pathologique du Corps humain, livres vi. viii. xvii.

[4] Prus: Mémoire sur l'Apoplexie méningée (Mém. de l'Acad. Royale de Médecine), Paris, 1845, t. xi. p. 18.

[5] Consult Virchow; Die Krankhaften Geschwülste, Berlin, 1863, p. 140; and Lancereaux in Archives générales de Médecine, Paris, 1862, pp. 526–679, and 1863, vol. i. p. 38.

doubtless, in the adult and the old, but that they are very rare, in children especially, is sufficiently proved by the suddenness of the symptoms.

There may be sudden paralysis of motion on the side opposite to that of the extravasation; and when this is considerable in amount, both sides of the body may be affected, or paralysis may begin in one side and extend to the other. Sensation is rarely affected. Motor paralysis is not a constant symptom, and deviation of the tongue and of one angle of the mouth, and strabismus, are of very rare occurrence in adults, whilst they have never been observed in children: according to Legendre,[1] paralysis occurs only in one out of nine cases; Rilliet and Barthez say, in one out of seventeen.[2]

Contractions, rigidity of the limbs, and convulsions are, on the other hand, almost always present. There is at first somnolence, which gradually merges into coma; and this, when once established, persists, as a rule, unto the end. About the third or fourth day of the attack, there is intense fever lighted up, accompanied by the other symptoms of meningitis.

The course of the disease is exceedingly irregular; death may take place early or not until the end of a month. The prognosis is not necessarily fatal, and according to Legendre serous cysts may be formed, which give rise to a form of chronic hydrocephalus. Hemorrhage may also occur beneath the arachnoid, between it and the pia mater. In some cases, it may take place suddenly; in others, it may be preceded by some headache, drowsiness, redness and heat of the scalp and forehead. When the extravasation has taken place there is generally headache, but not very acute, and having no fixed seat. Paralysis of motion is rare, probably from the thinness of the layer of blood effused. It has been said that when the blood is derived from a ruptured artery, motor paralysis is more apt to occur than when it proceeds from a ruptured vein, the difference being explained by the rapidity with which the blood escapes from the artery, and the incidental shock to the brain. Sensibility is not affected as a rule. The intellectual faculties are merely enfeebled, not perverted. After a time coma sets in, which persists until death.

On inspection after death, which seems to be an invariable termination of the disease, the blood is found in a liquid state, showing no tendency to coagulate, or to form pseudo-membranes. From the fact that no old cysts are ever discovered between the arachnoid and pia mater, it is inferred that sub-arachnoid hemorrhage is invariably fatal. In his memoir on "Diseases of the Lateral Sinuses," Tonnelé[1] has related instances of rupture of a sinus giving rise to sub-arachnoid extravasation of blood. In a case reported by Dr. Mullar,[2] the blood came from the right lateral sinus, which was ruptured at its point of entrance into the torcular Herophili: death occurred in twenty-four hours, the symptoms preceding it having been those of cerebral hemorrhage. According to Aitken, arachnoid hemorrhage occurs when the extravasation bursts through the pia mater and arachnoid into the space between the membranes; and he says such an affection cannot be distinguished from ventricular extravasation. If, however, the extravasation is immediately arachnoid at first, and of limited extent, it may be approximately diagnosed: first, by the nature of the symptoms having partaken of meningeal inflammation, such as by severe pain in the head, with impaired intelligence and loss of power of movement; second, the attack is less sudden than in cases of congestion or of cerebral hemorrhage, and the symptoms are progressively developed.

The following are the combinations of symptoms which indicate sub-arachnoid hemorrhage:—

First.—Complete and profound coma without paralysis, or with general paralysis slightly developed.

Second.—Complete loss of consciousness without paralysis, but combined with rigidity or clonic contraction of limbs.

Third.—Paralysis of hemiplegic distribution, as regards the limbs; but without deviation of the features, the muscles of the face not being implicated.

Fourth.—An apoplectic attack without anæsthesia.

Fifth.—Imperfectly developed coma with general paralysis.

Sixth.—An apoplectic attack, of which the symptoms are somewhat interchangeable or remittent.[3]

The TREATMENT of arachnoid hemorrhage must be guided by symptoms present. When there is perfect coma with full, hard pulse, which is possible, a hot head, flushed face, turgid veins of the neck, and a hot general state of surface, with a slow, deep respiration, we may open a vein and take away ten ounces of blood with advantage; but in by far the majority of cases there will be no such

[1] Legendre: Mémoire sur les Hémorrhagies dans la Cavité de l'Arachnoïde (Recherches anat. path. et clin. sur quelques Maladies de l'Enfance). Paris, 1846, p. 130.

[2] Maladies des Enfants, p. 257.

[1] Journal hebdomadaire de Médecine, Paris, 1829, tome v.

[2] The Lancet, June, 1849.

[3] Reynolds: Diagn. of Dis. of Brain, &c., p. 101.

opportunity. If the tendency be to death by syncope, the pulse small or feeble, the surface cold, the face pale and head cool, if there be signs of disease of the aortic or mitral valves, of kidney disease, or a general appearance of anæmia, we should do mischief by abstracting blood. The administration of a turpentine and castor-oil enema, and the application of an ice-cap or cold lotion to the shaved scalp, will generally limit our power of treatment, until the period of shock has passed away, or until consciousness has returned. Then the propriety of applying leeches must be measured by the degree of pain in the head, and of heat of the scalp. In the majority of cases even this will be found unnecessary. Free purgation will always be advisable, and the continued application of cold to the head. After a time, if the patient survives, the continued administration of the iodide of potassium promises the most hope of good. With respect to the abstraction of blood, it is right to say that the most eminent authority in Paris, Professor Trousseau, never saw any reason to order it. He denied the slightest advantage to be gained by it, either in arachnoid or in any other cerebral hemorrhage.

ADVENTITIOUS PRODUCTS IN THE MENINGES.

Under the heading of Syphilitic Meningitis the presence in the dura mater of so-called gummata has been adverted to. Calcareous deposits are also found sometimes in the substance of the dura mater; in some cases the falx cerebri has been found completely ossified.

HÆMATOMA OF THE DURA MATER is hardly recognizable during life. The symptoms, according to Aitken, extend over several months, and consist in general weakening of memory and of intelligence, the occurrence of giddiness, and local pain in the head. A chronic form of idiopathic inflammation of the dura mater is set up. At a later period an aggravation of all the symptoms occurs, with transitory losses of consciousness. Somnolence and apathy prevail, and generally one-sided paralysis of the extremities, which may soon disappear; eventually the case terminates with symptoms of apoplexy. On post-mortem examination, sanguineous and flattened masses, composed of fine layers of fibrine, spread to a greater or less extent over the dura mater, are discovered, accompanied by small extravasations which are converted into pigment. By repetition of the process, numerous layers come to be deposited one on the other. Numerous and larger bloodvessels form in these layers; and from these vessels renewal of the hemorrhage occurs (Virchow, Weber). The lesion is sometimes described as due to intra-meningeal apoplexy, with false membranes on the dura mater; but the false membranes, which are the result of chronic inflammation, precede the apoplectic phenomena. The hæmatoma often attains considerable size. It may be four to five inches long, by two and a half broad, and one-half to three-quarters of an inch thick. It is generally of a flattened circular form, with a central elevation. The long diameter is parallel to the falciform process. The tumor generally occurs on one side only, or if bilateral, one is more developed than the other. The affection occurs only in the adult, and usually after the age of fifty. It has been clearly made out that the hemorrhage which gives rise to the formation of the hæmatoma takes place between the layers of false membrane, and becomes encysted there. Treatment must be tentative. Iodide of potassium is our chief remedy. Any symptoms which indicate a recurrence of the chronic inflammation must be met by the application of cold to the forehead, by purgatives and revulsives. If we can succeed in preventing the recurring attacks, there is fair ground for believing the newly-formed membranes may undergo a retrograde change and finally disappear.

Tumors have not been unfrequently found springing from the dura mater, varying as to their character and the nature of their contents. They are sometimes *fatty* and *encysted*, and have been known to contain hair; and Morgagni (in Epist. Anat. xx.) speaks of an adipose tumor with hair in the substance of the tentorium. But sometimes also the tumor is of cancerous nature, constituting what has been termed "fungus of the dura mater." The celebrated French surgeon, Louis, has written a most important series of essays on the subject, published in "Mém. de l'Académie de Chirurgie," vol. v. p. 1, Paris, 1774. The cancer may be of the *encephaloid* or of the *scirrhous* variety: the former is the more frequent of the two, and indeed, when tumor has made its way outwards by perforating the bones of the skull, it has often been mistaken for hernia cerebri. The tumor may spring from the outer or the inner lamina of the dura mater, and in some cases there may be tumors co-originating on both surfaces of the dura mater, as in cases reported by Chelius and by Dr. Bright. When they spring from the inner surface of the dura mater, they have a tendency to grow inwards and depress the surface of the brain. But in comparatively rare cases they press on the skull, cause absorption of its substance, and protrude externally. In one case, de-

scribed by Cruveilhier, protrusion had actually occurred; in another, perforation was in progress. In connection with the dura mater are sometimes also found *fibrous* tumors, which, on microscopical examination, are seen to possess the same curvilinear stromal arrangement as the common uterine fibrous tumor.

The bony plates found on the inner side of the dura mater have by some been regarded as growth belonging to the arachnoid, but that they are not so is sufficiently proved both by the position in which they are found, and by the property of periosteal tissues, to which class the dura mater undoubtedly belongs, to generate bone.

In connection with the choroid plexus (that intra-ventricular appendix of the pia mater) the adventitious products found have been indurated yellow bodies, the remains of former hemorrhagic effusions, and more frequently round or oval bodies of a yellowish tinge, apparently formed of concentric laminæ, which only become more apparent on the addition of acetic acid. They are generally microscopic, but sometimes accumulate into masses of the size of a pea or small nut. They have been called by Virchow *corpora amylacea*, and by Dr. H. Jones *concentric corpuscles*.

Small *cysts* are also pretty frequently found on the choroid plexus, which have by some been erroneously spoken of as hydatids, but there is no evidence to show that they belong to those parasitic formations. They rather seem to be due to a condensation of the epithelial covering of the plexus, and an accumulation of fluid beneath it, limited by an effusion of plastic matter.

CONGENITAL MALFORMATIONS OF THE MENINGES.

The dura mater is the one generally affected, and such cases are of extremely rare occurrence. Sometimes the falciform process is entirely or partially deficient, as is also the tentorium.

The falx is, of course, absent when the cerebrum is undivided; or if the cerebrum be single in front and divided behind, the falciform process begins to appear where the division is, namely, at the coronal suture. In monsters, in which the posterior lobes of the cerebrum are deficient, the tentorium is also deficient. In a girl, seventeen years old, who was idiotic and motionless from birth, the hinder part of the tentorium was deficient.[1] The Reports of the Pathological Society, 1847 and 1848, p. 178, contain the account of a very rare defect of the falx cerebri exhibited by Mr. Shaw. Dr. Bright also gives a similar case, in which no trace of the process was visible anterior to the tentorium, and it was assumed that the defect, which occurred in a lady of thirty years of age, had existed from birth.

CONGESTION OF THE BRAIN.[2]

By J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.

Under this name there are to be included several forms of disease very different from each other in the general character of their symptoms. In one of these the patient is feverish, and his attack is sometimes regarded as "brain fever;" in another the case is described as an "apoplectic" or "paralytic stroke;" in a third, as a fit, or seizure of "convulsions;" and in a fourth, as an attack of "delirium," or of "wandering." Patients taken with symptoms of disturbance, in any one of these forms, often die; and upon post-mortem examination there may be found but one departure from healthy appearance of the brain, viz., congestion. We cannot but suspect that in many cases there have been alterations of nutrition which have escaped our notice, and that the locality and nature of such alterations have determined the form that the malady has taken. We may infer in others that, if the congestion has been the sole cause of symptoms, the character of the symptoms which accompanied it was determined by the situation of the excess of blood. But we cannot

[1] Gilbert, in Edinburgh Medical and Surgical Journal, No. 95, April, 1828.

[2] The sections on Pathology and Morbid Anatomy are written by Dr. Bastian.

yet demonstrate the truth of these surmises or inferences, for we cannot see the brain while the symptoms last, and the most characteristic often pass away before the patients die. The premonitory, and even the earlier developed symptoms of cerebral congestion may be closely similar in many cases; they then speedily pass into one or another of the several groups enumerated above, and from the special characters which they then present the cases derive their names: but if the morbid state continue, and advance towards a fatal issue, the distinctive features of these several forms pass away; convulsion, delirium, and febrile action cease; and patients, who a few days before presented very wide symptomatic differences, look much like each other, and die in a similar manner. Such being the case, we should expect to find the traces of that which existed only a short time before death, and to fail in discovering evidences of those localized changes which must have determined the character of the previous symptoms.

It will be convenient therefore to describe first the premonitory symptoms of cerebral congestion, viz., those which are or may be common to its several forms; then to detail under four distinct categories the developed symptoms, with their modes of termination in recovery; and lastly, to describe again generally those which are final, and into which any one of the four varieties may pass by a rapid or a gradual progress.

Symptoms.—A. *Premonitory.*—There is often a mixture of two classes of symptoms—those which indicate both over-action and the reverse; sometimes the two coexist, at other times they alternate; in one case the former group is predominant, in another the latter; whereas in a few all the symptoms tell in the direction either of inaction or of undue excitement. It is possible sometimes to foretell, from the nature of these symptoms, the form which it is most likely that the disease will hereafter assume; but such forecast is uncertain in all cases, and useful in only a very small proportion.

The *mind* is changed in such manner that there is diminished intellectual power; thought becomes confused, and memory treacherous; the individual may be irritable, "put out about little things," worried, fanciful, peevish, or depressed; sleepy, and especially so after meals; at times indifferent and sluggish; he complains that he "cannot think," and that the forced effort to do so makes him worse; he talks at random, using wrong words—sometimes noticing his blunder, correcting it, and expressing his annoyance; sometimes not observing that he was wrong, and being greatly annoyed with any one who should attempt to set him right. He is usually worse after being in the recumbent posture, and after sleep; his sleep is heavy, and disturbed by dreams and nightmare; sometimes there are transient delusions—one person is mistaken for another—the past and the present are curiously intermixed, and the conversation is like that of a dream, a dream which goes on while the patient is awake, but from which he may be awakened still further by a loud voice or any other strong appeal to the senses.

The *senses* are dull: hearing is defective, and there are rumbling "noises in the head;" the sight is dim, and "black specks" appear before the eyes; sometimes diplopia is present; there is giddiness, and a feeling of oppression and fulness in the head, with "stupid headache," made worse by lying down. There is rarely "pain" in the head, but, as the patients say, "a confused, uncomfortable feeling." The limbs feel heavy, and there is often numbness or "pins and needles" in the toes and fingers. These sensations come and go, but between the periods of their recurrence there is a sense of general discomfort which it is often quite impossible for the patient to describe. Often it is that of "oppression about the breathing;" and great difficulty from this source is experienced in walking upstairs, uphill, or even a little more quickly than usual on level ground. Sometimes "feelings of faintness" are complained of, and with them nausea and increased vertigo.

The power of *movement* is diminished, and with it yet more notably the readiness of action. The limbs are dragged along sleepily, or sluggishly; the step loses its elasticity, is shorter than in health; the "general bearing" is changed; and sometimes, but rarely, the alterations in power and activity are observed on one side of the body more distinctly than on the other. The patient simply leans forward, and appears weak and lethargic; or he may lean to one side, hold one shoulder half an inch or an inch higher than the other when standing, and when sitting, collapse, as it were, on the lower side. Friends of such patients say, "He seems to go down on this side," and, "We are afraid that he will fall off his chair, or off the pavement," but the physician may observe no paralysis; for the stimulus given to voluntary effort by his presence is often enough to remove the trifling want of symmetry. The features are regular, the tongue is straight in its protrusion, and the grasp of the hand and the movements of the feet are as pronounced on the one side as on the other.

There are *other* symptoms than those of direct change in the nervous functions, such as redness, and often dusky redness,

of the lips, conjunctivæ, face, and scalp. The head is hotter than the cheeks, the jugular veins are distended, and the neck appears thick. On stooping, sneezing, or coughing, the veins of the forehead are too full, and the beat of the carotids is too distinct. The pulse is slow and labored, or quick and feeble; the tongue is foul, the urine small in quantity, and often loaded with lithates; the bowels are confined, and the extremities are cold. The heart may be found dilated, and there may be tricuspid regurgitation, shown by the pulsation of the jugulars, and systolic murmur at the ensiform cartilage.

These premonitory symptoms may exist for very variable periods of time; may appear and disappear; or may gradually increase and pass into one or another form of malady already hinted at, but now to be described.

B. *Developed Symptoms.*—1. *Apoplectic form.*—The attack usually takes place during some muscular exertion, such as lifting a heavy weight, blowing the nose, coughing, sneezing, straining at stool, or stooping to pick up something from the floor. Sometimes it cannot be traced to any one of these. But it rarely occurs during sleep; patients do not wake up and find themselves in a state of what is called "congestive apoplexy." They are more commonly doing their ordinary work, or trying to do a little more than they are able to accomplish, when the attack is made. Consciousness, sensation, and power of motion seem to be lost, and the patient is said to have "an apoplectic stroke;" but these faculties are not altogether lost, or if they are, it is for a few moments only, and the physician usually finds the following conditions:—

The *mind* is not in complete abeyance. There are indications that the patient knows, although but imperfectly, what is said to him; he makes some attempt to respond to questions, and to do what he is asked to do. He starts at a loud and sudden noise, looks round him, and gives signs of annoyance when he is disturbed. If at the moment of seizure he should appear to be in profound coma, this coma is of short, almost of momentary duration, and soon there are signs of returning consciousness: there is confusion of thought, bewilderment, and dulness of apprehension, passing sometimes into a mild delirium, but more often into a heavy sleep.

The *senses*, obtuse for a moment, are rapidly restored to a certain point. The patient shrinks from strong light, groans when pinched, starts when spoken to, but yet takes little or no notice of ordinary impressions.

The power of *motion* is so diminished that the patient falls down, and the limbs when raised fall heavily. All of them appear equally weak; but, in a few moments, occasional voluntary movements may be seen in them, and these are commonly more distinct on the one side than on the other. There are slight twitchings of the muscles, but there is no rigidity. The features are usually symmetrical, or if drawn to one side are speedily set straight again. There is no stertor in the breathing; the speech is clumsy, the words are clipped, and wrong words are used; but this is for a short time only, and the sphincters very rarely fail in their action.

The *pulse* at the moment of attack is sometimes suspended at the wrist, and the *breathing* is arrested; but soon the pulse is felt to be heavy and labored, and the respiration becomes tumultuous; and again, in a few seconds, both pulse and respiration go on as they did before. There is an exaggeration of the previous vascular fulness of the face, neck, and head; sometimes a bloodshot eye, or epistaxis; not unfrequently vomiting, with apparent faintness and a condition of collapse.

The symptoms of a first attack usually abate quickly: they may last for a few minutes, or for several hours, but most commonly they disappear within an hour; and the patient, although languid and perhaps alarmed, may feel better than he has done for some days before. Upon the repetition of seizure, however, the duration of symptoms is prolonged; the recovery of consciousness, sensation, and movement is less complete; drowsiness is more marked; and if there be some momentary awakening, it is momentary only; fresh attacks supervene, and each leaves the patient lower than he was before.

The apoplectic form of cerebral congestion is most common in advanced life, and has usually been preceded, and that to a marked degree, by the "premonitory" symptoms that have been described.

2. *The convulsive form.*—The paroxysms that occur have the general features of epilepsy (see Part I.); but they differ from the attacks of that disease in their general history and mode of onset. Congestive convulsions may occur at any period of life, but they are most frequently met with at the time of full maturity, or when that stage is passed. There are usually the premonitory signs of congestion, but these may be very slight; there may be no forewarnings, and the patient may be seized during sleep, or while making some unaccustomed effort. When the attack occurs during sleep it is difficult, and sometimes impossible, to say in what manner it commenced; but when it has come on while the patient is awake and friends are about him, it has been usually observed that much discomfort has preceded it, for a few seconds, minutes, or hours. A tight cravat, worn while making

some undue exertion; a sudden alarm; or an indigestible meal, rapidly swallowed, may be the immediate antecedents. The patient, more or less suddenly, becomes confused, then apparently half unconscious, makes some unintelligible sounds, turns red and then blue in the face, staggers for support, looks round him wildly or imploringly, and then sits down, or falls down, convulsed, and a paroxysm, epileptiform in character, supervenes. From this he recovers partially, exhibiting great confusion of mind, headache, muscular feebleness, and sometimes partial paralysis of one side, or of one limb. The attack is occasionally followed by quasi-maniacal excitement, lasting from half an hour to three or four hours; after which, the patient becomes exhausted and falls into a heavy sleep. From this state he may recover, or during sleep a second or third attack of convulsions may come on.

When congestion of the brain has assumed this convulsive form, the patients, so far as my own experience extends, have usually been in middle life, and have recovered. But in other instances, when the age has been further advanced, the attacks have recurred more frequently, the intervals between them have become of shorter duration, and the patients—less sensible and less reasonable after every paroxysm—have presented the appearance of those whose attacks have been apoplectiform at their commencement.

3. *Delirium* may be the most marked symptom of congestion of the brain in certain cases. This is observed almost exclusively in those who are of an advanced age, but it is not absolutely limited to the period of senility. It may occasionally be met with in middle or even early life, and is then commonly accompanied by some change in blood-quality. The attack may come on suddenly, may be induced by a fall or a fright, but when occurring spontaneously is first observed towards evening. Sometimes the attack is preceded by "depression of spirits;" the patient, after some hours or even days of undue taciturnity, becomes cheerful, or gay, and hilarious; he talks loudly and incoherently, but rarely exhibits any violence. He gets out of bed, wanders about his room or ward, opens drawers, puts on his dress, and is bent upon doing something which he cannot explain, or which, if expressed in words, is unnecessary, unaccustomed, and absurd. He is under a delusion, of no fixed character; and can usually be directed and managed without much difficulty. Sometimes, and this is especially observed in the aged, there may be hysterical crying; or, still more rarely, great irritability of temper and some attempts at violence. The latter occur almost exclusively as the result of bad management and rough thwarting of the delusive purpose.

The patient may complain of pain in the head, or of uneasy sensations in the limbs; and there may be twitching of the muscles, or weakness of the extremities. But none of these are complained of while the delirium lasts, although weakness and clonic spasm may be observed at the time of its occurrence. It is when the delirium has completely or partially subsided that these things are noticed, and that the general phenomena of cerebral congestion, viz., those which are described as premonitory symptoms, may be observed. Durand Fardel states that it is common to find "a mucous secretion, clear and viscid, produced on the eyelids, or in the interior of the mouth, and sometimes in extraordinary abundance, running over the whole face;"[1] but this is very common in other diseases of old people, and has no special relation to mere congestion of the brain.

The recurrences of delirium may be very frequent or very occasional: some old people present them nightly for many weeks, and know nothing about them on the following days; while others exhibit them after much longer intervals, and only when "upset" by the little occurrences of the day. The tendency, however, is towards increase—not so much in degree as in persistence of mental change—and the patients become gentler, but less rational. The mind is weakened at each onset of delirium, and does not recover itself; there is drowsiness in the daytime and wandering talkativeness at night; but the intellectual powers are seen to be failing day by day; the physical energy diminishes, and the patient keeps his bed, and gradually passes into the state hereafter to be described.

4. *Febrile Form.*—In the earlier periods of life, and especially in infancy and childhood, congestion of the brain may occur with marked elevation of temperature, a dry skin, thirst, and the restlessness and malaise of a pyrectic state. There is headache, not of great intensity, but of dull, oppressive character; the head is unduly hot, the cheeks and conjunctivæ are flushed, while the extremities are cold; the mental faculties are obscured, and the sleep is broken by dreams or transient and mild delirium. Usually there has been some distinct cause for such disturbances; there is no marked prostration, no initial rigor; there may be some vomiting, but it is not persistent, nor are the bowels obstinately confined; there is no photophobia, no intolerance of sounds, no eruption on the skin; the secretions may be foul, but they present no indications of organic disease; and the

[1] Maladies des Vieillards, p. 27.

patients usually recover speedily. Recovery is, however, not always observed; the distress may persist; there may be, alternately, convulsions and delirium, or there may be the changes from over-excitement to drowsiness, the latter gradually becoming relatively more marked, until the patient passes into a state of stupor from which he may never rally.

C. *Final Symptoms.*—Under whatever form congestion of the brain may primarily appear, its tendency, unless speedily recovered from, is to produce a condition of torpor and inactivity. The mind becomes a blank; there is sometimes profound coma, stertorous breathing, and involuntary evacuation of both bladder and rectum; sensibility both general and special is lost, and voluntary muscular power reduced to a minimum. Convulsions may occasionally disturb the calm, or there may be fitful and momentary muttering of unintelligible sounds, but usually, in this latter stage, the patient lies quietly, with labored pulse and breathing, and with flickering contractions of the muscles of the limbs, until he dies.

Causes. — Among the predisposing causes must be reckoned such physical conformations as should impede the return of blood from the head, and the most important of these is a morbid condition of the heart. Dilatation of the right side of the heart, with loss of both power and valvular competency, are commonly found, during life and after death, in those who succumb to cerebral congestion. It is in old age that such changes are usually discovered, and hence advanced age appears a predisposing cause. It is more common to find severe cerebral congestion in men than in women. Sedentary occupation and shortness of neck have been reckoned among the predisponents, but I think with insufficient reason; for attacks of cerebral congestion often occur in those of active habits and of healthy build, and indeed sometimes the worst forms of seizure that I have witnessed have been in persons of great mental and physicial activity, in those who have been overwrought, and who have continued in forced exertion beyond the bounds of reason and habitual practice.

The determining causes are to be found in all those conditions which entail sudden changes in the circulation. These are exposure to extreme heat or cold, and especially to the direct influence of the sun's rays; blows upon the head or trunk; violent exertions, such as make it necessary to "hold the breath;" rarefaction of the air, such as is encountered in balloon ascents, and in some mountaineering expeditions; violent emotion, or prolonged mental effort; an overloaded state of the stomach, and this especially after undue abstinence; the ingestion of large quantities of alcoholic stimulants; a sudden change of posture, such as stooping or lying down with the head too low; and tightness of the dress around the neck.

Besides the so-called predisposing and exciting causes of cerebral congestion, there are two general conditions of the organism which may have some causative relation to the symptoms, although neither of the preceding words fully conveys the nature of that relation. A full-blooded, lax-fibred, and fat man, in middle age, represents one of these conditions; a thin, pale, wiry old person, with rigid vessels, is an example of the other. Both are prone to suffer from disturbances, irregularities, inequalities in the circulation; and in either there may be cerebral congestion in a grave or fatal form. If these conditions be regarded as "predisponents," it must be remembered that they have no special relation to this locality of congestion, and further that the mode in which the one operates is quite distinct from that in which the other leads to its results. In the former cerebral congestion is but part of a general condition, and some accidental posture may determine that the brain shall be the organ upon which the weight of the burden falls; in the latter, feebleness of circulation power, and locally increased resistance in the walls of vessels, may be the main factors in the production of such partial congestion of the brain as shall give rise to an apoplectiform seizure.

Diagnosis.—Remembering the general character of the symptoms which were described as "premonitory," there can be but little difficulty in carrying the diagnosis up to a certain point, and in explaining them by the fact of congestion; but when the malady passes into either one of the four forms of "developed" symptoms, the diagnosis is sometimes difficult, and it is therefore necessary to consider it in detail with regard to each.

The *apoplectic form* of congestion resembles cerebral hemorrhage, acute softening of the brain, urinæmia, and syncope. From hemorrhage, it may be distinguished by the facts of its less sudden onset; its occurrence while the patients are awake rather than when asleep —patients do not wake up in the morning and find themselves paralyzed on one side, as they often do in cases of hemorrhage;—the attack of "congestive apoplexy" occurs during the day, and its onset is marked by the absence of the phenomena of shock; by the equality of disturbance usually noticed in regard of mind, sensation, and motility at the commencement of the seizure—each of them is affected generally, and to nearly the

same degree, but in no one direction is there entire and absolute loss of function except for a few moments; by the subsequent relative proportion of symptoms, such for example as partial paralysis of all the limbs with imperfectly developed coma, a combination not to be observed in hemorrhagic apoplexy; by the speedy restoration of the mental faculties; and by the equable and usually simultaneous removal of other symptoms.

From softening of the brain in its acute form, congestive apoplexy cannot be always distinguished at the outset, for in some cases of the former the attack is in reality due to the occurrence of the latter. The diagnosis can only be made after some little time has elapsed, and then it will turn upon a recognition of the following points: In congestion the mind speedily recovers, in softening it does not; in the former there is widely distributed but imperfect paralysis, in the latter limited, but more complete, loss of power; in the one the patient is generally powerless, in the other he is hemiplegic; in the one there is flaccidity of muscle, in the other there is rigidity; in the former the premonitory symptoms have been those of congestion of the brain, in the other those of chronic disease elsewhere and loss of power.

From urinæmia, the attack may be distinguished by regard to the premonitory symptoms; by the absence of œdema of eyelids or of lower extremities; by the absence of albumen from the urine; by the absence of marked rigidity of muscles; by the nature of the coma, its momentary profundity, rapid diminution, and want of that peculiar character which often attaches to blood-poisoning, viz. its apparent profundity in strong contrast with the ease with which the patient may be awakened up to a certain point; and further by the absence of a peculiar variety of stertor, occasioned apparently in the mouth or at the palate.

From syncope, congestive apoplexy may be distinguished by an examination of the heart, and the pulse at the wrist, the carotids, and the temples; by the color of the face and head; the premonitory symptoms, and the conditions which led to the attack.

The *convulsive form* of congestion may be confounded with epilepsy or with eccentric convulsions. From epilepsy the diagnosis may be made by a consideration of the previous history: in the one there have been the premonitory symptoms of congestion, in the other no such phenomena have presented themselves; in the former the patient is usually of middle or advanced age, in the latter he is young, and is either under twenty years, or has not far exceeded that period of life; in the one the period of most marked congestion is at the moment of onset of the seizure, in the other congestion of the face and head is most marked as the attack is passing off; in the former there may be some moaning sound, in the latter the "epileptic cry;" in the one there is the sudden onset of an acute disease, in the other the attack of a chronic malady.

From eccentric convulsions it is possible to distinguish congestive convulsions by regard to age and attendant symptoms. Eccentric convulsions are observed in infancy and early life, and when some definite source of irritation can be discovered in certain organs of the body; they are found most commonly in the weak, irritable, and nervous subject, and they are attended by no premonitory symptoms of congestion, and by little or no evidence of its presence during the attack. There is but trifling somnolency, and the seizures differ from those of epilepsy and of congestive convulsion in not passing through the stages which were described as proper to the former, and which are closely simulated by the latter.

Congestion of the Brain in the *form of delirium* is met with almost exclusively in old age; and it is necessary only to mention delirium tremens in order to prevent the possibility of their being confounded. From senile softening of the brain, when this is accompanied by recurrent delirium, the diagnosis may be made by regard to the intermediate state; for when only congestion is present the patient returns to his normal condition in the intervals of wandering, whereas when the brain tissue is undergoing degeneration, and is the cause of delirium, no such recovery is possible. There is, moreover, a progressive enfeeblement of all the nervous functions, and a general condition of depraved nutrition such as is not necessarily found in cases of congestion.

The *febrile form* of congestion may be distinguished from meningitis by the absence of acute pain, and of intolerance of sensorial impressions; by the milder character of the delirium, the dilatation rather than contraction of the pupils, the absence of persistent vomiting and of obstinate constipation, the generally milder character of the symptoms, and their early cessation.

PATHOLOGY.—The circulation through the cerebral vessels has been supposed to present certain peculiarities owing to the inclosure of the brain within an unyielding case, and its being, therefore, beyond the influence of atmospheric pressure. This was first alluded to by the second Monro. It was thought that no great alterations could take place in the total quantity of blood within the cranium at different times, although there might be an altered ratio as regards the respective

amounts of arterial and venous blood. It was even held by Dr. Kellie, that in animals which have died from hemorrhage there is no lack of blood in the brain; that where, on the contrary, we should expect to find a condition of cerebral hyperæmia, we do not meet with it; and that the quantity of blood in the cerebral vessels is not affected by gravitation, and thus is uninfluenced by the position of the head with respect to the body. These views were also supported by Dr. Abercrombie and by Dr. John Reid;[1] though they have been ably opposed by Dr. Burrows[2] and by Donders,[3] many of whose experiments go to establish the direct reverse of the results arrived at by Kellie. It seems by no means satisfactorily demonstrated that the contents of the cranium are so entirely removed from the influence of atmospheric pressure. Dr. Burrows says: "The numerous fissures and foramina, for the transmission of vessels or nerves through the bones of the cranium, appear to me to do away with the idea of the cranium being a perfect sphere like a glass globe, to which it has been compared by some writers." And the other dogma on which this hypothesis rests, and which Dr. Abercrombie supports when he says, "We may safely assert that the brain is not compressible by any such force as can be conveyed to it from the heart through the carotid and vertebral arteries," seems to be directly contradicted by a consideration of other facts.[4]

The observations of Robin,[5] and of His,[6] who have discovered a system of lymphatic sheaths inclosing spaces around the cerebral bloodvessels, are of great importance, and reveal a structural adaptation which seems especially calculated to permit of varying amounts of fulness of the cerebral vessels, within certain limits, without injury to or compression of the surrounding nerve pulp. Professor His has succeeded in injecting this system of perivascular canals, and has found them most obvious in the gray matter of both brain and spinal cord. He has found that the injections at first reach the surface of the encephalon and cord, and fill a vast system of lacunæ situated between the pia mater and the surface of the nervous centres; while, if pushed still further, he has found that they fill the lymphatics of the pia mater itself. Thus there is, as it were, a second series of vessels inclosing and surrounding with a fluid medium all the ramifications of the cerebral and spinal vascular system, whilst these two sets of vessels, containing and contained, are lodged in definite cylindrical canals permeating the nerve substance in all directions. The lymphatic sheaths are in contact with, though in general are easily separable from, the walls of these canals through the nerve substance. The diameter of the canal (and therefore of the lymphatic sheath) may be seen, in transverse sections, to be generally twice, and sometimes three or four times, as large as that of the contained bloodvessel. It will be easily understood that these two systems must have such a complemental relationship to one another, that an extra fulness of the one set of vessels will correspond with diminished fulness of the other set. That is to say, in order to make room for an increased amount of blood in the cerebral vascular system a corresponding amount of fluid must be driven out of the enveloping lymphatic vessels;[1] whilst, when the vascular supply is again diminished, a proportionate amount of fluid reenters the cerebral lymphatic canals.

Thus, we believe that the amount of blood existing within the cranium may be subject to great variation, and that the peculiarities of the cerebral circulation have been much overrated.

The conditions capable of bringing about a state of cerebral congestion are very various, and so also is the degree of hyperæmia met with, and the extent of its diffusion over the encephalon. In one class of cases, the congestions seem to be most obviously mechanical phenomena, due to some impediment to the proper return of blood from the brain, owing either to diseases of the heart or lungs, to pressure upon the great veins by tumers, or to their obliteration by thrombosis. In other instances, however, the condition of hyperæmia seems a more purely vital phenomenon, as when it is the result of prolonged study and over-mental work, or when it has an irritative origin, and is set up around some old clot, bony exostosis, or adventitious product in the brain. Then, too, alcoholic intoxication, great elevations and alternations of temperature, exposure to the sun's rays in hot summer weather, and the suppression of accustomed fluxes, whether menstrual or other, are all looked upon as occasional causes of cerebral congestion. In connection with inflammation of the meninges, congestion of the convolutional gray matter is doubtless the initial stage of what afterwards becomes diffuse superficial

[1] Physiolog. Anatom. and Path. Researches. No. xxv.

[2] Lumleian Lect. 1843, and On Disorders of the Cerebral Circulation, &c. 1846.

[3] Nederland. Lancet, 1850.

[4] Andral's Clinique Médicale.

[5] Brown-Séquard's "Journal de Physiologie," 1859, p. 527.

[6] Zeitsch. für wissen. Zoolog. 1865, Bd. xv. and The Journal of Anatomy and Physiology (Cambridge), No. 2, p. 347.

[1] Cambridge Journal of Anat. and Physiol., No. 2, p. 351, note 2.

cerebritis. Well-marked Congestion of the Brain is also met with very frequently in persons who have died whilst suffering from symptoms of delirium or coma during the course of the acute specific diseases, and in whom there may be no trace of meningeal inflammation. This is more especially common in typhus fever. From observations which I have made on the bodies of persons who have died from this disease, and also from the minute examination of the brain of a man who died delirious whilst suffering from acute phlegmonous erysipelas of the head and neck, I have been led to believe that these minute and wide-spread congestions are often due to embolism or thrombosis of the minute arteries and capillaries of the brain.[1] Wide-spread obstructions in the small vessels, however brought about, would cause much of the propulsive energy of the heart to be wasted and a consequent lagging of blood in the venous radicles.

Cerebral congestion is very intimately related to cerebral hemorrhage on the one hand, and to inflammation on the other. Hemorrhage is most likely to be associated with the congestions of mechanical origin, especially if these are brought about rapidly; and although such cerebral conditions generally give rise to well-marked brain-symptoms, still the groups of symptoms previously described are often related to congestions of a more active kind —such as are commonly spoken of as 'determinations' of blood to the head—and which may be said to commence rather on the arterial than on the venous side of the circulation. In these cases, perhaps by virtue of certain changes occurring in the nerve tissue itself, an increased flow of blood takes place to the brain, which may subside after a variable time and after the production of a certain set of symptoms, or which may occasion the death of the patient owing to the supervention of symptoms of a graver type. In certain other cases the congested condition of the membranes and cortical substance may gradually lapse over into a state of inflammation, and it will then be associated with tissue changes of a more marked character.

Morbid Anatomy.—Congestion of the brain tissue itself is almost invariably associated with a similar condition of the pia mater, and the amount of cerebral congestion is often judged of, in a loose way, by the degree of fulness of the vessels of this membrane. What many persons would consider to be a state of congestion is, however, natural to the vessels in this situation. Hasty opinions on this subject should, therefore, be especially guarded against. This fulness of the vessels of the pia mater is most notable in the occipital region, whither the blood gravitates, for the most part, after death. Occasionally, however, as suggested by Laborde, this occipital congestion may take place during the last days of life, so as to place it in the same category with hypostatic congestion of the lungs.

In some cases, where there has been every reason to believe that a state of congestion existed during life, it must be confessed that little or no traces of it can be recognized after death; though, on the other hand, when it has existed for some time and has been carried to an extreme degree, or when it has been often repeated, undoubted evidences of the present or previous existence of such a condition may be met with. In a young and middle-aged subject, in whom no atrophy has taken place, but whose brain has been subjected to an extreme degree of congestion during life, the organ frequently seems, after the removal of the calvarium, to be in a swollen condition. The dura mater is tightly stretched over the organ, and after its reflection the convolutions appear broad and flattened, with sulci less obvious than natural, owing to the effects of pressure against the interior of the skull. Then, there is not only the usual fulness of the large veins of the pia mater, but also a more tortuous and even varicose condition of these trunks, together with a more complete injection and turgescence of the smaller vessels than is usually encountered. The membranes may be stripped off the surface of the convolutions without tearing the gray matter, and on section this appears darker than natural, and dotted with bloody points in the situations of its loaded vessels. The white substance also shows an abundance of a certain number of the red points, which are usually gorged vessels pulled out for a certain distance so as to lie on the cut surface. These are only comparative signs, however, and their true value must be estimated accordingly, since all intermediate conditions may be met with between the ordinary healthy amount of fulness and the most marked degree of hyperæmia. It is extremely difficult to draw the line and say what is morbid and what is consistent with health.

If, however, the congestions have been often repeated or have lasted for any length of time, microscopic examination does enable us to discover evidence of this. The capillaries, and more particularly those of the gray matter, become

[1] "On the Clogging of Minute Vessels in the Gray Matter of the Brain as a cause of Delirium and Stupor in severe Febrile Diseases; and on other Symptoms of the 'Typhoid State:'" Brit. Med. Journ. Jan. 23, 1869.

twisted and varicose, displaying partial dilatations, or real aneurismal swellings, implicating either a part only of the calibre of the vessels, or dilating them in numerous adjoining parts in their whole extent, so as to constitute "l'état moniliforme" of Laborde.[1] But a still more certain mark of old congestion is afforded by the presence of a quantity of blood pigment (hæmatine) surrounding the vessels, though inclosed within the lymphatic sheath described by Robin. It is met with in the form of more or less rounded simple or molecular grains, mostly of large size. They may measure as much as $\frac{1}{2000}$" in diameter. They are usually of a dark olive or amber yellow color, and are sometimes composed of a number of minute pigment granules aggregated into small spherical masses. The pigment remains quite unaltered after the application of ether, alkalies, or the strongest acids. The crystalline form of blood pigment (hæmatoidine) is not met with, since this seems to be produced only in places where an actual extravasation of blood has taken place, whilst the pigment in the granular and amorphous condition seems to result from stasis of blood, and more or less transudation of coloring matter, or hæmatine, through the walls of the vessels into the surrounding lymphatic canals. It seems impossible otherwise to account for what I have seen. I have found, for instance, this matter in great abundance around almost all the small vessels and capillaries that were examined belonging to the brain and spinal cord of two individuals. Both were lunatics; the one an epileptic and chronic maniac, subject to paroxysms of great excitement, and the other a chronic maniac of the most violent and excitable disposition, whose fits of passion were both frequent and long-continued. It was during the examination of the brains of these individuals that this granular blood pigment, surrounding the vessels, first attracted my attention. I have since found that a similar condition had been noticed and described by Robin, and I can endorse his statement that a few such masses of pigment are usually to be met with, here and there, on the cerebral vessels of even young and healthy subjects. It is, therefore, the abundance of this matter only which is to be looked upon as an index of disease; and the duration of past congestions may be roughly guessed at by the more or less excessive accumulation of pigment around the vessels.

Occasionally, however, an actual rupture of one of the minute vessels may take place under the increased strain upon its walls in cerebral congestion. This is all the more likely to occur in elderly people whose vessels have been weakened by fibroid or atheromatous degenerations. In such cases I have not unfrequently found, after careful preparation, evidences of past capillary hemorrhages on several of the smallest vessels of the same brain. After the brain substance has been washed away, and when the vessels are floated in water in a shallow dish, one or more little orange-colored specks may be seen, even smaller than a pin's head. On examination with the microscope these are found to be accumulations of altered blood pigment in the form of amorphous canary-yellow colored flakes, interspersed with distinct crystals of hæmatoidine, situated around one of the minute vessels, and distending its sheath in a more or less obtuse fusiform manner. In these cases the presence of the perivascular sheath seems to have limited the amount of blood effused. As soon as the sheath became distended in the immediate neighborhood of the rupture, the pressure so produced would tend to close the aperture in the ruptured vessel.

Lastly, there is to be mentioned that condition of certain parts of the brain which was spoken of by Durand-Fardel[1] as "l'état criblé," and which he and others regard as an evidence of previous dilatation of the vessels from long-continued congestions. This condition is occasionally well seen, more especially in old people, in the white substance immediately beneath the gray matter of the convolutions. On section a number of round or oval apertures appear—some large enough to admit a pin's head—and within each may be seen the cut extremity of a vessel. In these situations the canals in the nerve substance have become enlarged by pressure, and the lymphatic sheaths have been dilated to a similar extent, whilst in the space between the sheath and the much smaller bloodvessel a large quantity of pigment granules is generally met with. This dilatation of the vascular canals sometimes reaches an extreme degree in the corpora striata and

[1] "Le Ramolliss. et la Congest. du Cerveau," Paris, 1866. These irregular aneurismal dilatations of thin-walled capillaries must not be confounded with the distinct though microscopic aneurisms, occurring on some of the smallest arteries after they have undergone a process of fibroid thickening in different parts of the brain in old people. We have previously hinted at the occasional connections between cerebral congestion and cerebral hemorrhage, and now we may state that the links which bind the two together are frequently the aneurisms just mentioned. Congestion may have something to do with their formation, as it certainly has to do with their final rupture, leading to effusion of blood. (Trans. of Path. Soc. vol. xviii. 1867.)

[1] Loc. cit. p. 57.

in the optic thalami, and the same condition may be encountered, though to a less extent and less frequently, in the substance of the pons Varolii. Whenever the granules are met with, however, the structural conditions and the mode of origin seem to be the same. Durand-Fardel says:—"Tantôt l'état criblé du cerveau se trouve répandu dans une grande étendue des hémisphères, tantôt on ne l'observe que dans un espace circonscrit." This condition may be met with at all ages, though it is found more particularly in old people: and in them, the same writer tells us, the canals are sometimes so large and numerous in the corpora striata, that these bodies may seem to have lost nearly half their substance. It seems most probable that these canals have been produced by the dilatation and pressure exercised by congested vessels, though their method of pathogenesis cannot be said to have been ascertained in a thoroughly satisfactory manner.

Prognosis.—In cases of cerebral congestion regard must be paid to the age of the patient, the form of his attack, the severity of the symptoms, and the frequency with which the symptoms or the attack of symptoms may have occurred.

Age cannot be fairly estimated by the mere duration of life, for some men are "older" at fifty-five than others are at seventy years, and are so without any necessary coexistence of exhausting or definite disease. The apparent age is a truer guide than the real age in the matter of prognosis. Baldness, gray hair, rigid vessels, a weakened heart, arcus senilis, and enfeebled powers, must be taken into more serious account than the date of birth; and judged by such tests, the prognosis is unfavorable in proportion to the oldness or agedness of the individual.

The form of attack is worthy of most grave consideration. That which is of the worst omen is the apoplectic; next to this is that characterized by delirium; after it the convulsive form; and least serious of all is the febrile, or quasi-febrile.

The severity of symptoms is of much value in relation to the apoplectic form; the danger being in direct proportion to the profundity of coma, and its duration. It is of but little moment when delirium is the most prominent symptom, and the value that it possesses is in inverse rather than direct ratio to the force of the disturbance; the prognosis is worse when the delirium is mild, muttering, and continuous, than when it is noisy, or even violent, and—as is usually the case—of short duration. When convulsions occur, it is not safe to base any prognosis on the mere fact of their severity; for often patients recover after the most frightful seizures, whereas others succumb to much milder paroxysms. The degree to which, in the intervals of seizure, the mind is restored to its normal state, is a fairer criterion of the amount of danger than is the violence of the convulsion. In the febrile form the prognosis is bad in direct proportion to the intensity of the symptoms. When the disturbance is slight, confident hopes of recovery may be entertained; when it is severe, there is room for the apprehension of ulterior and "inflammatory" changes.

Congestion of the Brain is rarely fatal at its first attack; it becomes dangerous in proportion to the frequency and readiness of its induction; and this is true with regard to each form in which the symptoms may be developed.

The other conditions by which the prognosis must be determined are those of organic disease or degeneration in any of the important vital organs. It is obvious that the heart, the vessels, the kidneys, and the liver should be examined with care, and that the opinion formed as to the future should be guided by the kind and amount of disease that may be found in them. The prognosis, however, when such diseases are discovered, is not that of cerebral congestion only, but of those complicated morbid conditions of which it is but one form of expression. It may be that Congestion of the Brain is likely to prove the cause of death, but the nature of the disease which leads to such congestion furnishes the material, by a consideration of which the probabilities may be estimated.

Treatment.—As there are two distinct, practically opposite, conditions of the body under which cerebral congestion may occur, so there are two different lines of treatment to be adopted. If the brain congestion be but one of many symptoms of a general plethora, much may be gained by either general or local blood-letting; if it be but the outcome of weakness and vascular obstruction, then such measures may increase the evil. The previous habits and health of the patient, the present state of his integuments,—their warmth, vascularity, and color,—the state of the pulse, of the heart and vessels, will furnish the guides in this important matter. A man in middle age who has overstrained himself, or placed his head in some dependent position, and who is attacked by violent convulsions, characterized by great turgescence of the skin, bloodshot eyes, and a full but labored pulse, may be relieved, and greatly relieved, by venesection to the amount of six or ten ounces. But such cases occur rarely, and in the majority of instances no man would at the present day think of bleeding from the arm. When, however,

there is distinct general weakness, and, with this, heat of head, oppression, continuous headache, and a tendency to drowsiness, much relief may be obtained by the application of leeches to the temple, or by cupping to three or four ounces at the back of the neck. When there is no such heat of head, and no flushing of the face, but when the diagnosis of cerebral congestion may still be made—per viam exclusionis—and when the vital powers are low, the pulse small, feeble, irregular, or intermittent, even a small abstraction of blood locally may be followed by the worst results. It is when attacks of congestion are frequently repeated, and other measures have failed to relieve them, that local depletion may be found of signal service.

It is well to raise the head, to apply cold water or ice to the forehead, and to place the feet and hands in hot baths. If the stomach be overloaded, an emetic of mustard or ipecacuanha may be given; and often with the discharge of the stomach the symptoms pass away. This is especially useful when the attack has followed a full but hastily taken meal. It is of great importance to empty the rectum, and the most efficient means for doing this is the administration of an injection of warm water. Should there be any suspicion of the existence of hardened masses of feces, the injection of a large quantity of warm olive oil will prove more useful than that of water.

When the tendency to cerebral congestion is noted, rather than any marked symptoms of its presence to a high degree, the secretions must be carefully regulated; and among these one of the most important is the urinary. Many cases of threatening aspect are to be relieved by saline diuretics; and I have known a copious flow of urine to be followed by the removal of symptoms which had existed in spite of free purgation and other treatment.

There are many cases occurring in advanced life in which the congestion is of only momentary duration; and the patient, when seen by the physician, is simply bewildered, pale, and with a cool, moist skin, and feeble pulse. Under such circumstances the cautious administration of stimulants is called for; and of these sal-volatile and wine are the most useful. It is well to combine with them carbonate of potash, or of soda, as there is often considerable "acidity of stomach," and the discharge of flatus by the mouth, which results from such administration, is often followed by a complete remission of the symptoms.

As precautionary measures, quiet of mind, and gentle exercise of body, with the careful avoidance of either fatigue, sudden change of posture, or strain, should be enjoined; and much relief may be obtained by insuring a position during sleep which shall prevent not only the head, but the head and shoulders, from sinking down to the level of the body. This may be easily obtained by a simple contrivance placed under the bed or mattress upon which the patient lies; such an arrangement being much better than a mass of pillows, which shift their places, and often maintain the head in a condition of undue heat.

[In patients who have been subjected to the influence of malaria, special care is sometimes needful in diagnosis, in order for the proper adaptation of treatment. A lady was placed under my care who, without any distinct chill, became comatose, and continued so for about twelve hours. Her age, over sixty years, made apoplexy not improbable. Her pulse, however, was feeble, as well as moderately slow; and her respiration was not stertorous. She was known to have just visited a malarious region. Ordinary derivative measures were used, and, as soon as she was able to swallow, quinine was given, a grain every hour, watching its effects. Under this treatment she recovered.—H.]

CEREBRITIS.

By J. Russell Reynolds, M.D., F.R.S.,[1] and H. Charlton Bastian, M.D., F.R.S.

It is probable that general inflammation of the brain never exists alone, but that it is invariably associated with meningitis. The terms encephalitis, meningo-cerebritis, and phrenitis, which have been employed to denote the condition now referred to, are sufficient of themselves to

[1] The section on Pathology is written by Dr. Bastian.

point out this constant association. Nevertheless, in some cases there is to be found, during life, the predominance of a class of symptoms which simple meningitis will not account for; and, after death, the presence of such changes in the cerebral tissue, as do not necessarily accompany the meningeal inflammation. It would seem, therefore, that the brain substance is not only susceptible of morbid change of an inflammatory type, but that the presence of such change may determine the clinical history of the case. We may, in particular instances, refer some of the symptoms of a complex encephalitis to inflammation of the membranes, and others to an implication, in like change, of the cerebral tissue.

Meningitis has already been described, and it remains for us, in this place, to describe only those symptoms which mark the extension of the malady to the brain itself. All that relates to that which has been described as local Cerebritis, or limited softening of the brain, will be found under the articles on Abscess of the Brain and Softening of the Brain.

Causes.—The most common causes are injuries to the head; such as violent contusions, wounds, diseases of the bone, and insolation. It would appear, however, that sometimes prolonged mental exertion or moral excitement have led to the development of this disease. In rare cases there has been no distinctly recognizable cause, the symptoms having appeared in the absence of any one of the conditions above mentioned.

Symptoms.—These are, of necessity, associated with those of meningitis, but sometimes they are the earliest to appear, and are predominant throughout the case. Thus, some mental change may be the first evidence of disease; it may be very slight, and may be mistaken for "hysteria," "stomach disturbance," or some such vague malady. In one case, which I saw several years ago, there was a mere confusion of ideas, and a worried manner, with misuse of words, and this for two or three days before other phenomena appeared. Usually the patient is sullen, and the faculties are obscured; there is a confused, "muddled" state of the intellect, sometimes merging into mild delirium, sometimes, when meningitis is present, alternating with, or superseded by, violent excitement.

There is deep-seated, oppressive pain in the head, described as sometimes shooting from the centre to the vertex, the temples, eyes, or ears; and this pain is persistent, and is out of all proportion to the pyrexia, which is often very slight. Except in dependence upon meningitis, there is no intolerance of light or sound, but there may be obscurity of vision, diplopia, and failure of sight, together with ringing noises in the ears, and some difficulty in hearing.

There is general muscular lassitude, but neither definite paralysis nor spasm; the limbs are weak and aching, but they may all be moved.

Such symptoms may continue for two, three, or four days, and then a violent convulsion may occur, followed by coma, from which the patient never thoroughly recovers. There is, however, partial recovery sometimes, and then more or less general paralysis is discovered. The patient is stupid, sleepy, comatose, and lingers for a shorter or longer time, in proportion to the amount of nourishment that can be given and retained by either stomach or rectum. Convulsions, somewhat epileptic in character, usually recur, and in their intervals there is to be observed a gradual dying out of the various functions of the brain. Mind, sensation, and voluntary power are lost, and the patient lives a mere vegetative life, disturbed occasionally by slight spasmodic movements, or rigid contraction of the muscles. The convulsions are often of long duration, involve the limbs especially, and are not marked by notable asphyxia.

The general symptoms are, as a rule, so slight that they attract no notice. There is no fever, little or no vomiting, and no obstinate constipation of the bowels. Very often the sphincters are relaxed quite early in the history of the case, and nothing abnormal can be discovered in the evacuations.

Diagnosis.—That which gives to the diagnosis of meningitis its gravest element is the recognition of coexisting Cerebritis, and hence the diagnosis is valuable as an aid to prognosis. Cerebritis may be inferred when there is a rapid transition from the excitement of meningeal inflammation to the marked loss of function which is characteristic of cerebral change. When the signs of meningitis are unusually severe, the pain deep-seated, and followed after twelve or twenty-four hours by convulsions, coma, and paralysis, there is commonly Cerebritis of considerable extent.

Pathology.—Of uncomplicated Cerebritis we have no knowledge. When inflammation of the brain substance exists, it is either associated with a more marked change of the same kind in other parts, such as the meninges, in which case it is treated of as a concomitant condition, and not as a primary morbid affection; or else it speedily lapses into other distinct pathological states, such as abscess or softening, which, on account of their importance, are usually described as independent affections of the brain.

Two kinds of Cerebritis are usually de-

scribed, namely, the diffuse or general form, and local Cerebritis, which by most recent writers has been held to be synonymous with "red softening" or "acute ramollissement" of the brain.

The diffuse form, or general Cerebritis, is a more or less wide-spread affection of the cortical substance, or gray matter of the convolutions, and is always associated with inflammation of the meninges. It may be met with in surgical cases, from injury to the skull; when, conjoined with it, there is inflammation of the dura mater and arachnoid, together with the formation of purulent lymph within the arachnoid cavity, and also beneath the visceral layer, into the meshes of the pia mater. Cerebritis may also be met with in the more limited meningitis, such as occurs when the disease is not of traumatic origin, and which, affecting the pia mater principally, is not accompanied by any purulent effusion in the sac of the arachnoid. In these cases there is extreme vascularity of the cortical gray substance, which is also more soft and pulpy than natural; and it is frequently adherent to the meninges, so as to be torn when these are stripped off. For further particulars we must refer to the articles "Meningitis" and "Tubercular Meningitis," under which heads these morbid conditions are more fully described. It should be stated, however, that many pathologists of the French school look upon general paralysis of the insane as a disease due in part to a species of chronic Cerebritis. The same adhesion between the gray matter and meninges is frequently met with in this disease; but for further information we must refer to the article on this subject.

With regard to local Cerebritis, we think with Lebert and other pathologists that this may be the antecedent condition and proximate cause of abscess in the brain; and we do not deny, also, that some acute softening of the brain may have an inflammatory origin. We do, however, strongly object to the view that all "red softenings," or "acute ramollissements," have to acknowledge this method of pathogenesis. We believe that most of the softenings hitherto placed in this category have been brought about by embolism or thrombosis, owing to the interference with the crebral circulation thus induced; and that the characters usually considered as diagnostic of their inflammatory nature are capable of receiving a totally different interpretation, as may be seen on reference to the article "Softening of the Brain." In this view we are supported by many recent writers on the subject. With regard to the occasional existence of softening of the brain of inflammatory origin, we do not altogether disbelieve in its occurrence, only we plead ignorance as to the characters by which such softenings are to be distinguished from others of a degenerative nature, due to arterial or venous obstruction. We certainly think it is a pathological condition which occurs very much more rarely than the statements of some pathologists would lead us to imagine. It may, perhaps, be looked for most confidently in cases of wounds or injuries to the brain, or around adventitious products, as centres of irritation.

Prognosis.—The prognosis is as bad as it is possible to be. There is no probability of recovery when symptoms such as those above described have been developed.

Treatment.—Only palliative measures can be used with any advantage. We have never seen any good result from mercury given by the mouth or by inunction; nor from blisters, cupping, or other modes of blood-letting. Pain may be relieved by the application of ice; and spasmodic movements may be limited by sedatives, such as belladonna and Indian hemp; but beyond such relief of symptoms therapeutic art has failed.

SOFTENING OF THE BRAIN.

By J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S.[1]

Definition.—A disease characterized during life by impairment of mind, sensibility, and motility, and after death by diminished consistence and degeneration of the cerebral substance.

The disease now to be described is that which has been known as white or non-

[1] The sections on Pathology and Pathological Anatomy are written by Dr. H. C. Bastian.

inflammatory softening: ramollissement blanc, or ramollissement non-inflammatoire.

Causes.—There is little that is satisfactory which can be said with regard to the remote etiology of Softening of the Brain. Among the conditions which predispose to its occurrence the most important is age, or agedness. Softening of the Brain is essentially a manifestation of decay, and this may be either the natural result of the wear and tear of a long life's work, or it may be the early outcome of excessive strain. The real cause is that waste of tissue which is unbalanced by repair, and this may come from the long continuance of work,—old age,—or the unhealthy severity of work, and its undue relation to rest. The proximate causes may be resolved,—as will appear in the section on "pathology," — into morbid conditions of the vascular system. Neither sex, constitution, nor season of the year has been shown to exert any marked predisposing influence, nor has any distinct relation been made out between any one particular condition of the heart and cerebral softening. Degeneration of the kidneys and impaired nutrition of the heart and vessels are among the conditions which frequently accompany ramollissement; but these ought to be regarded as certain parts of a general change of which the cerebral softening is but another or counterpart, rather than as predisposing causes of its existence. Vegetations on the valves of the heart may become detached and may block up one of the cerebral arteries; and thus their presence on the valves might be regarded as predisposing to Softening of the Brain. But it must be remembered that such vegetations when *in situ*, i. e. undetached, do not specially predispose to Softening of the Brain, that they may lodge in other vessels than those of the cerebrum, and that when they are carried from the heart to the cerebral arteries they become determining and not predisposing causes. If we may employ the term "predisposing cause" under these circumstances, we should do so to the general or constitutional state that has led to the production of vegetations rather than to the vegetations themselves.

There is nothing definite to be said with regard to exciting causes. Attacks have sometimes followed violent mental or moral excitement, anger, abuse of alcohol, over-fatigue, or local injuries; but in the majority of cases no such conditions have been present, and in very many there has been a singular immunity from all apparent causes of disturbance. Exposure to cold has been followed by an apoplectic seizure, and one of the more frequent determining causes of an attack has been too free a purgation of the bowels.

Symptoms. — Softening of the Brain may occur as either an acute or a chronic disease. It will be well, therefore, to describe the affection under two general headings, and first:—

Acute Softening of the Brain.

1. *Premonitory Symptoms.*—These may be absent altogether, but such complete immunity is rare; for Softening of the Brain most frequently occurs in those whose health has been for some time below the average, and very frequently in others who are the subjects of some distinct chronic and exhausting diseases. There is nothing so special in the character of the general condition which may precede Softening of the Brain as to render it of much value in the forecast of a patient's chances. There is often an enfeebled condition, with impaired nutritive power, shown in the general bearing of the patient, and more distinctly in the weakness of cardiac impulse, rigidity of arterial vessels, and local inequalities of temperature. These facts may be noticed for months or even years, but there is nothing in them that points specially to the brain as the organ which is likely to give way. In combination with symptoms of cerebral failure they are, however, of great significance.

The symptoms which, when thus combined, are premonitory of softening, are often those already described as characteristic of impending or actually developed congestion of the brain. (See p. 845, article "Congestion of the Brain.") They are—headache, more or less constant in duration, and usually "dull" in character, dulness of sight or hearing, numbness, obscure pain, weight, or an indescribable sensation of "something wrong" in the extremities, slight confusion of thought, sleepiness, weakness of purpose, hesitation in judgment, irritability of temper, diminished control of emotion, deficiency of muscular power, a stooping gait, and tendency to cramp in the limbs. Sometimes the face assumes a dull, expressionless aspect when the patient is at rest, and he may pass hours in a state of apparent indifference to all around him; but when called upon to exert himself is able to resume his habitual manner, and do his accustomed work, although with some heaviness of manner and apparent effort. There may be occasional and slight symptoms of faintness, the face becoming pale, and the limbs cool; and such occurrences are of much significance. It sometimes happens, moreover, that the altered sensations above described are noticed more on one side of the body than on the other; and the fact of this limited distribution is highly indi-

cative of impending evil. There may be a little dragging of one leg, or only a tendency to lean to one side when either walking or sitting; and this, when constant in its locality, is of much graver meaning than is a much greater amount of weakness when variable in its seat.

Such symptoms may continue for a shorter or longer time, and may precede either acute or chronic softening, and there is nothing in their nature, when existing only to the degree described, and which can only be regarded as "premonitory," that furnishes any clue to the form which the developed symptoms are likely to assume.

2. *The developed symptoms* may occur in one of three distinct forms. The patient may either have an *apoplectic* seizure, be taken with *convulsions*, or may pass into a state of *delirium;* and it will be convenient to describe these forms separately, premising that sometimes they pass into one another, and that occasionally mixed cases are observed, in which stupor, delirium, end convulsions alternate.

(*a*) *The apoplectic form* may be very gradual or very sudden in its onset. When the former, there is an increase of the premonitory symptoms for days or weeks; when the latter, there may have been no special premonition, but the patient suddenly falls down in what is termed an "apoplectic fit," and he is said to have had "a fit," or "a stroke."

Very commonly the attack occurs after too long an abstinence from food, or when the patient is fatigued by too long a walk, or too protracted an effort; sometimes when, as in congestion of the brain (see page 845), he is making an excessive exertion. It is not common, so far as my experience extends, for patients to wake up in the morning and find themselves paralyzed on one side of the body, a mode of attack by no means uncommon in the case of cerebral hemorrhage.

The condition of the *mind* is highly significant. Transient excitement, talkativeness, irritability, or wandering of thought, amounting sometimes to mild delirium, may occur for a few minutes. The patient says or does something quite out of relation to his surrounding circumstances or previous conversation; speaks as if to some person he may not have seen for years, asks a question which refers to events long since passed, or in some other manner shows that he is "not quite himself;" is bewildered and "queer," vexed or pathetic; he makes some effort to get up and do some extraordinary thing which no one can understand, is impatient of attempts at dissuasion or control, looks faint, and becomes more or less insensible, sometimes falling to the ground, sometimes voluntarily sitting or lying down, as if merely fatigued, or disgusted with the stupidity of those around him, who do not understand what it is he wants to do.

The patient may for a few minutes be completely insensible, and when he is so, it is probably due to sudden congestion of brain, or to equally sudden anæmia of brain, either of which may be recovered from in a few seconds or minutes. When, however, the physician sees the case, he rarely finds absolute loss of consciousness. The patient lies quietly, in apparently profound sleep, snoring, and taking no notice whatever of the questions that friends ask in anxious and beseeching tones; but if spoken to sharply, told to put out the tongue, open the eyes, give the hand, or do any other simple thing, he responds at once, usually makes an awkward failure, and then relapses into his former state; if asked a question, he makes some inarticulate or unintelligible sound in reply, and again falls back into his heavy sleep, sometimes muttering to himself, but more commonly snoring continuously, or occasionally interrupting the rhythm of his snore by a long-drawn sigh.

In such a state the patient may continue for hours, days, or even weeks. There is often sufficient intelligence remaining for him, when roused, to swallow food, to recognize friends, to make efforts to say something; but so much dulness of apprehension, and so much difficulty of expression, that the real life is "hidden," and it is impossible to know that we are on such common terms with it that we can understand its meaning.

In some cases there is after a longer or shorter period marked improvement, the faculty of articulation returns, and a certain amount of conversation is possible to and with those who will give pains to learn the language that is spoken. The names of common objects are forgotten, or are confounded with those of others; and this, sometimes with such constancy that friends may understand what is intended, sometimes with such thorough want of uniformity that the meaning is unintelligible. Notwithstanding this great obscurity of expression, it may be perfectly clear that the patient himself knows distinctly what he means, is aware that he is wrong in his use of words, is vexed at his blunders, and ingenious in contriving means to counteract or avoid them. For example, he may know so well the words which he wants, and which he supplies by others in ordinary conversation, as to write down lists of words, and point to one or more of them in order to make up his sentences, or correct erroneous expressions. (See page 855.)

Occasionally, after being even profoundly affected, the mental condition may undergo great improvement, and the patient, although not perfectly restored, be carried back again to the point de-

scribed in the notice of premonitory symptoms. But far more commonly there is no real restoration; some confusion may clear away, the stupor may be lessened; but when these improvements have occurred the mind is found to be dulled and incompetent, and in a state of gradually increasing deterioration; week by week, and month by month, the patient is further and further removed beyond the reach of intercourse, until the attempts to talk with him are given up so gradually by the friends that they are scarcely aware of the change, and so imperceptibly to the weakening mind of the patient that he takes no notice. In this way a sudden attack of softening may pass into what is termed "chronic softening," the patient becoming imbecile of mind, and powerless in body.

In a certain number of cases, the course is rapid, the apoplectiform attack is repeated, and at the end of two or three days there is profound coma, passing into the sleep of death; in a much smaller number, the symptoms are very transient, and the recovery may be complete. For example, a young lady, æt. twenty-two, in her first attack of acute rheumatism, marked by considerable swelling and redness of knees, wrists, and ankles, and a recently developed systolic murmur at the base of the heart, received a visit from some friends, was excited in conversation, and had palpitation of the heart. A few minutes after her friends had left her she grew faint, looked pale, became unconscious, and remained so for two or three hours. At the end of that time she was confused, unable to utter any intelligible sentence, clipped her words, made some sounds that were quite inarticulate, and had marked right hemiplegia, the features being drawn to one side, the right arm being completely, the right leg incompletely, paralyzed. At the end of three days speech was perfect; and at the end of a fortnight the hemiplegia had disappeared. The most rational interpretation of such case is, that an embolus blocked up the left middle cerebral artery, and led to impaired nutrition of the brain, which equals the first stage of softening; but that, owing to either the re-establishment of the circulation by the breaking up or removal of the embolus, or to the perfect establishment of the circulation in the collateral vessels, the nutrition was restored to its ordinary condition.

Sensibility is sometimes quite destroyed at the time of attack, and for some few minutes afterwards; but in the greater number of cases it is only dull or impaired, and subsequently changed. While the patient is lying apparently unconscious, or only half conscious, it is often obvious that some sensibility is present, for he moans, moves about uneasily, puts his hand to his head, and starts or draws away some one or other of his limbs if the skin be scratched or pinched. It is probable, from the frequency with which the hand is put to the head, that there is headache, or an uneasy sensation in the head; and a general feeling of distress, with, very frequently, some distressing sensation in one or more of the limbs. Often before the patient is able to speak, he is evidently uneasy in some of the extremities, and these are usually on one side of the body, and are paralyzed; he looks inquiringly at them, or rubs them, moans at them, and cries out if they be either moved or touched. When sufficiently conscious to make himself intelligible, the patient often complains of coldness, or numbness, or "queer feelings" in the arm or leg of one side; of headache, or discomfort in the head not amounting to pain; of a bewildered feeling, and some vertigo. There is occasionally hyperæsthesia, and its occurrence in the limbs affected by a stroke of hemiplegia is thought to be highly characteristic of acute Softening of the Brain. It would appear, however, that, instead of true hyperæsthesia, there is a modified sensibility, such as that described above, and that it renders ordinary impressions painful. Sometimes these modifications present very curious features; the patient feels distinctly and painfully any impression on the skin, but is unable to refer it to its proper locality. For example, a pinch on the sole of the foot is referred to the inguinal region, while similar irritation above the knee may be felt in the shoulder, or side of the neck; and sometimes the sensation may be referred to the wrong side of the body.

In the majority of cases the sensibility of the limbs is, after an apoplectic attack of softening, speedily restored to its normal condition.

The special senses are, as a rule, unaffected except in the earliest stage of the attack, when all of them may be in abeyance. There is not rarely some complaint of tinnitus, and of muscæ, or of dulness in hearing, or mistiness of sight, but there is no one change which is characteristic of softening. The optic disk is often paler than natural. The vessels are extremely small, and either white or gray atrophy may be apparent. The outline of the disk is sometimes very sharply defined, and its shape distorted; but there may be integrity in the appearances presented by the eye when there are unequivocal indications of Softening of the Brain. There is often to be observed some marked peculiarity in the eyeballs and in the pupils, which being, however, illustrations of altered motility rather than of sensibility will be described under the following heading:—

The symptoms due to changes in motility. — It has been often observed that the eyeballs are directed to one side, and that the head is turned in the same direction, so as to give the idea that the patient is making an effort to look at something by the side of him, and usually on the opposite side to that of the paralysis in limbs. If carefully examined it may sometimes be shown that the patient does not see at all, and that the retina is quite insensible to light; while in other cases the patient sees distinctly, and may, by an effort of the will, bring the eyeballs to the middle line or even beyond it and to the opposite side. In one curious care, under my care in University College Hospital, this synergic condition of the eyeballs was observed for nearly a fortnight, at the end of which time the patient died. The patient was, when roused, sufficiently sensible to give a coherent account of himself, his sight and hearing were good, he could distinguish not only objects but colors, and seemed rather amused at being put through an examination on such points; yet while talking to me the eyeballs were constantly turned towards the right side, and so much so as to hide a considerable portion of each iris beneath the lids at the left inner and right outer angles. The patient often fell asleep, and began to snore while the students were standing round his bed; and what was very interesting to observe was this, that at the moment of doing so the eyeballs returned to the middle line. Upon touching him, or speaking to him so as to rouse him a little, the synergic movement again instantly appeared. Owing to the ease with which the transition from sleeping to waking could be effected in this case, the above observation was repeated many dozens of times, and always with a similar result. The eyeballs are usually, except at or soon after the occurrence of the attack, unaffected. It is the rare exception, and not the rule, to meet with strabismus even to a slight degree. There is nothing characteristic in the condition of the pupils; they may be found in almost every degree of either dilatation or contraction, and they are usually equal on the two sides in case of acute softening. Their relation to light is determined by the general sensorial condition rather than by any special involvement of their own motor centres. It has been said that occasionally the pupils dilate upon exposure to light and contract upon its withdrawal, but there has been, I believe, a fallacy in such observations which it is very easy to correct. The facts as they have been witnessed by myself in many cases are these: that the patient is found asleep, or in a state of half-unconsciousness, with contracted pupils; a strong light is brought before him, or the eyelids are suddenly raised, and then immediately there is dilatation; left to himself, again the drowsiness comes on, and the pupils pass into the state of contraction. The pupils dilate because the patient is roused, not because they are exposed to light; the contraction and dilatation that have been observed have had no relation to light or darkness, but simply to the facts of sleeping and of waking. This I have shown again and again by gently raising the lids of such a patient, and exposing the contracted pupils to the light without arousing him; there is then no dilatation nor change of any kind: but if he be addressed loudly by name, or if his toe be pinched so that he is awakened, the pupils instantly dilate.

The features are sometimes quite symmetrical, both when at rest and when in motion; but commonly there is some deviation, noticed most distinctly in the lower part of the face at the angle of the mouth when the patient speaks or laughs; and sometimes it is so trifling that it may escape observation unless the patient smile, or make a forced effort to exhibit either the upper or the lower teeth. In other cases there is marked paralysis of the face on one side, and dragging of the features towards the other, with deviation of the tongue; but let it be remembered that this paralysis does not, as a rule to which there are very few exceptions, involve the muscles of the forehead, eyeballs, eyelids, or pupils. The patients can equally raise the eyelids, open or close the eyes, and there is neither ptosis nor strabismus.

The speech is commonly interfered with, not only at the moment of attack but for a long time afterwards, and sometimes persistently. It may be so thoroughly abolished that no intelligible sound is uttered, although it is obvious that ideas of some kind are passing through the patient's mind; it may, on the other hand, be so slightly affected that alteration is observed only in the articulation of certain sounds, such as those of the letters *l* or *r*. Between these two extremes there is almost every variety of degree in the impairment of speech as a mechanical act, and there is also every shade of difference in the precision with which it expresses mental processes. Some patients can read with ease and correctness, articulating every sound distinctly: and yet they cannot construct for themselves a sentence of half a dozen words, so as to answer intelligibly the simplest question. Such patients, although able to hold a pen and copy sentences, or sometimes to write a few words from dictation, cannot compose anything for themselves. In such instances language is interfered with on its intellectual side. Other patients can write well, when not flurried, can talk

for a little time so as to be understood, can help to convey their meaning by signs and gestures; but when "excited," or sometimes even when not disturbed in such manner, they can make no such succession of articulate sounds as shall be intelligible. Here speech is interfered with on its mechanical side. In the former group of cases there is usually paralysis on the right side of the body: in the latter there is not any constancy in such association. It is to the former class that the terms "aphasia" and "aphemia" have been applied; and it is not rare to meet with cases which illustrate either it or the opposite condition: it is exceedingly easy to recognize intellectually the difference between the two extremes of symptoms, or between them as conjoint elements in a particular case; but by far the most common event is to meet with such combinations of the two that it is by no means so easy to say how much is due to the one failure and how much to the other.

As the words aphasia and aphemia have now passed into frequent use, and the conditions described by them have become not unfrequently the topics for medico-legal investigation, it is desirable that some further attention should be directed to them, or to what they mean. Aphemia was the word constructed by M. Broca;[1] and aphasia, an old Greek word, signifying the dumbness occasioned by strong emotion, was that used by M. Trousseau[2] to denote the same thing, viz., the loss of speech or of articulate language, when occurring as a symptom of disease.

The condition now well known as aphasia was observed by the older writers on medicine, some of whom appear to have recognized the distinction, and others to have failed to do so, between it and a more general condition of injury to the nervous centres. But the special pathological significance of the loss of language has been demonstrated within a recent period. Dr. Gall was the first who sought to discover the locality or seat of what he, in accordance with a certain school of philosophy, was led to regard as the separate faculty of language, and he arrived at the conclusion that this faculty had its place in those portions of the anterior lobes of the brain which lie upon the supra-orbital plates.

The idea of Dr. Gall was taken up and strongly advocated by M. Bouillaud,[3] who distinguished, with care, between the recollection of words and the power of producing distinct sounds for their expression. M. Bouillaud's great point, however, was to show that lesions of the anterior lobes of the brain occasioned loss of the faculty of speech, whereas diseases of other portions of the nervous centres were not so accompanied. Exceptions to Bouillaud's law were somewhat frequently pointed out both in this country and in France, and until a few years ago the general doctrine had fallen into almost complete disregard, although M. Bouillaud had repeatedly brought forward fresh facts in confirmation of his dogma.

The next step of great importance was taken by M. Dax, who, in 1836, pointed out, as the result of his analysis of numerous observations, that disturbances in the faculty of speech were always related to lesions of the left hemisphere, and never to those of the right. This work, to which my attention has been called by the able author of the article on Aphasia, in the new "Dictionnaire encyclopédique des Sciences médicales," was entitled "Lésions de la Moitié gauche de l'Encéphale coincidant avec l'Oubli des Signes de la Pensée."[1] The paper of M. Dax appears to have attracted little notice, and it was not until nearly thirty years had elapsed that M. Broca produced his celebrated paper,[2] in which he announced his conclusion that the seat of the faculty of articulate language was in the second, and especially in the third frontal convolution of the left anterior lobe of the brain. M. Broca used the word aphemia to denote the condition of patients thus affected; M. Trousseau the word aphasia. The last step in this history, and one almost equal in importance to either that had preceded it, was taken by Dr. Hughlings Jackson, who arrived independently at a conclusion similar to that of MM. Dax and Broca, but who went still further than either, and showed the anatomical nature of the lesion which most frequently caused aphasia, viz., plugging of the middle cerebral artery on the left side by an embolus derived from valvular disease of the heart.[3]

Aphasia may be produced by numerous diseases of the brain, such, for example, as congestion, hemorrhage, or tumor—but the most frequent cause is that to which Dr. Jackson directed especial attention. Certain aphasic patients can write, while others fail to do so; those who are capable of the act occasionally write sense, frequently nonsense, but more frequently either unintelligible characters or distinct

[1] Sur le Siége de la Faculté du Langage articulé; Bullet. de la Soc. Anat. 1861.

[2] Gaz. des Hôpitaux, 1864.

[3] Traité de l'Encéphalite.

[1] Gaz. Hebdomad. 28 Avril, 1865.

[2] Sur le Siége de la Faculté du Langage articulé, ant. cit.

[3] Loss of Speech, its Association with Valvular Disease of the Heart, &c. &c.: Clinical Lectures and Reports, London Hospital, vol. i. p. 388.

but unconnected words. Those who cannot write at all are usually, it must be remembered, paralyzed on the right side. Certain patients are able to make intelligible signs, others fail to do so; some have the power to calculate, to draw figures, and to perform on musical instruments, while others lose these faculties together with that of articulate language.

The mental state of the patient varies greatly, from a condition of almost perfect intelligence to that of almost complete fatuity. On several occasions the question has been raised whether an individual in a condition of aphasia should be regarded as competent to make a will. No general principle can be laid down with regard to so complicated a question, but each case must be determined on its own merits; still it must be remembered, —1st, That while intelligence may remain intact, the power of expression may be so damaged that it is impossible for any one to be certain that he has correctly interpreted the patient's meaning. 2d, That the patient may be distinctly capable of intellectual decision on matters up to a certain point of complexity, and quite incapable of dealing with those which are beyond that point; that he may be able to decide some simple questions, whereas he becomes bewildered when attempting to unravel those which require sustained thought for their comprehension. 3d, That the facts which are most valuable as bearing on this question are very rare, and contradictory, viz. those which are supplied by patients who have been aphasic, have recovered, have remembered their previous mental condition, and have been able to give an account of it.—On the one hand, there has been obvious mental obscurity; on the other, perfect clearness of intelligence, although, as in the case of Lordat, the memory for words was lost as well as the faculty for their expression.[1] 4th, That the balance of evidence is to the effect that the mind is usually somewhat damaged, although its degree of impairment may vary between wide ranges; that the loss of speech in aphasia may coexist with loss of mental power, but that it does not depend upon it. A patient may be dumb because he has no idea to convey, but such a person is not aphasic; he may have much to say, but be unable to find the words in which to express his thoughts,—such a patient is aphasic; he may be quite clear in his thought, quite certain of his words, and able to write them with facility, but he cannot speak articulately, because he cannot make the sounds he wants,—such patient is not aphasic in the true sense of the word, but is paralyzed in either the tongue, lips, or palate, or in all of them together.

The condition of aphasia has its analogues in locomotor ataxy, writer's cramp, and allied affections; and similar physiological considerations will carry the explanation of these curious states to about the same level of precision. Talking, walking, and writing are each of them very complicated processes, and are, in man at least, the result of education. Artificial, as well as natural, associations of nerve-action are involved in each of them,[1] and the result which ought to come from such co-ordination may be stopped at any point. We cannot yet assert what is the primary loss in the condition of ataxy, or in that of writer's cramp, neither can we do so with any accuracy in regard of aphasia. In each of them desire, volition, and intelligence may coexist with entire muscular capacity; and yet the patient who wishes, tries, and knows how to walk, to talk, or to write, and who, moreover, has full power in his legs, his arms, and his apparatus for articulation, is unable to accomplish his purpose: he staggers and falls in the one instance, he makes illegible scrawlings in the second, and meaningless sounds or unintelligible jumbles of words in the last.

The limbs are almost invariably paralyzed when there is Softening of the Brain, sometimes on both sides of the body, but with far greater frequency on one side. The paralysis is usually not absolute: it is more marked in the upper than in the lower extremity; it is seen to its highest degree in the fingers or toes, to its lowest degree in the shoulder or hip, and with intermediate severity in the forearm and leg. There is commonly some spasmodic contraction of the muscles which are paralyzed, and this may take the form of either tonic rigidity, or of occasional clonic, or even choreic movements. Sometimes the muscles are rigid at the very moment of the apoplectic seizure, but more commonly the reverse is observed—there is complete flaccidity —and not until after three or four hours is the stiffness of limb to be recognized. At first it is noticed only after repeated flexion and extension of a joint; subsequently it is persistent, and is not developed but simply exaggerated by attempts at movement. This rigidity is distributed with irregularity, and is noticed principally in the shoulder, elbow, and knee-joints; and in this respect it differs notably from what has been termed "late rigidity,"[2] viz. that stiffness of limb which comes on

[1] Analyse de la Parole, &c., pour servir à l'Histoire de l'Alalie et de la Paralalie. Par le Prof. Lordat. Dictionnaire, ant. cit. p. 632.

[1] See article, "Writer's Cramp."

[2] See Dr. Todd's Clinical Lectures.

after paralysis has lasted for many weeks or months, and which is distributed like paralysis itself with prevailing frequency in the distal extremities of limbs, and undergoes a gradual diminution as the joints are tested, one by one, from below upwards.

The electric irritability of muscles I have often found to be absolutely normal, even when paralysis is complete, and it has remained so for a considerable time; its persistence or the reverse has not been determined by either the degree, duration, or locality of the paralysis, by the presence or absence of rigidity or of wasting. The irritability of the paralyzed muscles, as tested by percussion, has been found often in considerable excess, and this when the electric contractility has been normal. Let it be distinctly understood that I am not now speaking of the force of muscular contraction, which is invariably, or almost invariably, diminished in a palsied limb, but of the readiness of response to electricity, which is tested—not by the vehemence of contraction in a muscle, but—by the weakness of the electric power which will bring muscles into play. In this sense the palsied limb exhibits every condition of irritability, viz., the normal, excess, or diminution. It does not appear to be of any moment in the diagnosis of cerebral softening from any other lesion in the brain, but it is well to bear in mind the facts that have been mentioned, inasmuch as mistakes have sometimes arisen owing to a confusion of terms when the distinction is being made between cerebral and spinal lesion or disease.

There may be no *general* symptoms in cases of acute softening; at the time of seizure the patient may have been in apparently good health. As already stated, however, this is not common. Usually the individual is "older than his years," as shown by aspect, manner, gait, premature baldness or grayness of head; he has been weak for some time, has been threatened with a "break-down somewhere;" has suffered from disease of heart or kidney; exhibits arcus senilis, and has a rigid pulse.[1]

Beyond these general states there is nothing to be noted; the patient may, and often does, vomit when the attack is beginning to clear away and some slight consciousness returns, but it is rare to meet with a repetition of the vomiting. The appetite often is quite good; the digestion, secretions, and evacuations, natural; the temperature, usually quite normal, is sometimes raised to a very high point, and in many cases is notably depressed in the affected limbs. When the temperature has been raised there has been considerable jactitation of the limbs, or convulsive movements, more or less general in their distribution.

(*b*) The *convulsive form* of acute Softening of the Brain is sometimes so distinct in its features that the case is more likely to be confounded with epilepsy than with hemorrhage or congestive apoplexy. After a few of the premonitory symptoms already described (p. 857) the patient is taken in a fit, which passes through the ordinary phases of an epileptic attack; but the patient does not become profoundly stupid or sleepy afterwards. He is, perhaps, restless, and a little loquacious, or he may be simply quiet, but "not quite himself," for a few minutes, an hour, or even longer, when a second seizure follows, in its turn to be succeeded by a third, and so on; each convulsion being followed by increasing stupor, and almost invariably by paralysis.

Regarded closely, these facts are to be observed with respect to the history of the disease. The premonitory symptoms, although so slight as to have attracted little notice at the time of their occurrence, are found, when attention has been directed to them by the onset of convulsions, to have been highly significant of impending evil. They consist generally in some marked change in the mental condition of the patient, such as peculiar drowsiness, listlessness, weariness, impatience, or some flaw in memory, with distinct but

[1] In an interesting case recently under my care in University College Hospital, a man became suddenly hemiplegic and aphasic, and continued so until he died. There was distinct evidence of old valvular disease of the heart, the radial arteries were hard and visible, the brachial arteries when he bent his elbow stood out like twisted cords, the pulsation of which was not only excessive and visible, but distinctly locomotive; there was highly-marked arcus senilis; the man was old, bald, and gray. The inference drawn from these symptoms was that there was softening in and below the left corpus striatum, and that the softening was due to disease of the cerebral vessels. On post-mortem examination the diagnosis was verified exactly as to the nature and locality of the disease; but, strange to say, the brachial arteries, which were inspected carefully, presented no unhealthy appearance, and exhibited their ordinary amount of elasticity when stretched between the fingers. The inference from this is important, viz.: that vessels may lose their elasticity during life, and that to such a degree as to form valuable guides in the diagnosis of disease, but yet there may be no atheromatous deposit, or other change in their physical appearance, which can be detected after death. The mode in which the function is performed during life is a better test of the physical capacity and condition of the organ than is the physical state of the dead artery when examined directly by the hand and by the eye.

momentary incapacity to understand what is said. There may be, however, some little hesitancy in speech, the mispronunciation of a few words, a little weakness of one side, or some vague feeling of *malaise*, with numbness of extremities, vertigo, or faintness, thought to be either hysteric or dyspeptic in their origin, until the fit occurs and demonstrates the gravity of their meaning. The convulsions, although resembling epileptic convulsions in the main, differ from them in certain particulars, viz. :—The tonic stage is but feebly marked, and there is not much asphyxia. Clonic spasms are more violent on one side of the body than on the other, and they continue for a long time. The patient does not "come out of the fits," but passes from one into another with no distinct intervening period of quiescence. The fits occur in increasingly rapid succession, and at length the patient can scarcely be said to be either "in the fit" or "out of it." He lies in a semi-comatose condition, occasionally muttering, and making movements which appear to be voluntary, but which are interrupted by spasmodic jerkings, by suspension of the breathing, or by momentary tetanic rigidity. Gradually the limbs on one side cease to exhibit voluntary movement — they fall heavily when allowed to do so; the eyeballs are often directed to one side, and the features lose their symmetry; and thus the case goes on until it passes from a convulsive into an "apoplectic" or paralytic form. The patient who was "taken in a fit," thought to be epileptic or dyspeptic, becomes distinctly hemiplegic, and the true nature of the case is recognized.

From this condition there may be partial restoration; after a few days of marked disturbance, intelligence returns up to a point varying through wide ranges, and the state of the patient may be that described in the previous section (see page 858). The convulsions, however, sooner or later, recur; either to be again recovered from, or to carry the patient beyond the reach of hope. The intervals between the attacks of convulsions may be either weeks or months; but in many cases the course is much more rapid, and the patient dies within twenty-four or forty-eight hours from the onset of the fits.

(*c*) The form marked by *delirium* has usually been preceded by distinct premonitory symptoms, and it is most commonly observed at advanced age. The patient suddenly "wanders" in his talk, becomes loquacious or restless, is busy in manner, exerts himself, seems tired, and falls asleep. He wakes up, somewhat confused, but appears "to be himself again" for a few days, or even weeks, when the confusion and delirium reappear, and are more persistent. There is no complete restoration, but gradually one side is found to be paralyzed, or to be slightly weaker than the other. The delirium alternates with coma, more or less profound; and the patient passes into a state like that following either the apoplectic or the convulsive form.

The delirium, usually mild, is sometimes violent; but when it is so there is generally some distinct meningitis, and the case runs a rapid course, reaching its termination in a few days.

3. The *final symptoms* of softening, like the prodromata, are similar in their character whatever the form in which the attack takes place. The patient becomes more and more comatose, the paralysis extends to the sphincters, the respiration becomes embarrassed, and death follows, usually "without a struggle." Nothing is more gradual or more tranquil than the mode in which the sleep of such patients often deepens, almost imperceptibly, into the sleep of death.

A peculiar form of softening has been described by M. Duparcque, as occurring in children of precocious intelligence, the symptoms of this condition being the following :—headache with drowsiness, perfect integrity of the mind, exaltation of the special senses and of general sensibility, without fever, delirium, or convulsions. After death the only change which has been discovered has been Softening of the Brain. M. Duparcque denominates this disease "ramollissement blanc aigu essentiel chez les enfants."[1]

Chronic Softening of the Brain.

The symptoms of this condition may follow an "apoplectic seizure," whether the latter has had for its anatomical basis congestion, hemorrhage, or acute ramollissement. They may, on the other hand, be developed very slowly and insidiously, and may, or may not, be preceded by those phenomena already described as "premonitory" of acute softening.

There is diminution of intelligence. The patient is unable to pay attention, and consequently fails to receive new ideas. Subsequently memory is impaired, past ideas are not recalled with readiness, and there is general confusion and incoherence. Sometimes there is mild delirium, or merely a restless and excited manner, towards the evening of the day, or in the night; occasionally there is the monotonous repetition of a particular word or act, which may continue for hours, days, or even weeks.

In regard of emotion, the majority ex-

[1] Archives générales de Médecine, Fév. 1852; quoted by M. Valleix, Guide de la Méd. prat., tom. 2me, p. 176.

hibit dulness, or some degree of melancholy; and it is not uncommon to find that the expression of feeling is very little under control, and the sufferer is said to have become "hysterical;" and this is often the earliest indication of failing power. (See article on "Hysteria.") In other cases laughing and crying are very common; but they occur without assignable cause, and without the apparent existence of any correspondent emotion. The intellectual weakness increases, and the patient becomes drowsy. At first he may be aroused, but subsequently there is profound coma, and the patient dies comatose. In rare cases, however, the intellect may be preserved throughout. The gradual failure, one by one, of the intellectual faculties is, *per se*, one of the most characteristic symptoms; and the peculiar monotony (of word or action) has led Durand-Fardel to a diagnosis in some obscure cases.

The most common alteration of sensibility is cephalalgia. It exists in about half the cases, and is felt generally among the earlier symptoms; but sometimes it does not commence until an advanced period, and it generally disappears towards the close of life. Its intensity is highly variable, rarely so great as that of meningitis, or *à fortiori* of tumor; its locality is frontal in the majority, and it is not often confined to one side of the head. When pain is not present there is generally a sense of weight and confusion of head; and, as I have observed in many cases, such a peculiar sensation that the patient says he fears his "mind is going." Painful sensations are often present in the limbs; and they are sometimes referred to the surface, sometimes to the muscles, and in other cases to the articulations. These modifications assume the form of so-called hyperæsthesia, cutaneous and muscular; or of numbness, formication, &c. They are commonly limited in extent to the parts presenting motorial changes; and when this is the case, they are highly characteristic.

Diminution of sensibility is common, but anæsthesia is rare; and, in respect of the former, it usually exists in conjunction with paralysis. These changes are gradual and imperfect in their development; and it is uncommon to find complete anæsthesia of the special senses.

Unless an apoplectiform attack has taken place, the muscles rarely exhibit any sudden changes; but when such a seizure has occurred, there may be complete hemiplegia; the face, articulation, the tongue, and the limbs of one side being involved in paralysis.

Paralysis in the typical form of chronic softening is distributed generally, and developed gradually; weakness of the muscles preceding their complete removal from volitional control. Hemiplegia is the most common form that is observed when chronic softening has an abrupt commencement, but it is followed, in many cases, by general paralysis, incomplete in degree; and this is important as a distinction from the persistent paralysis of hemorrhage. At first, one leg drags in walking, or one hand feels less strong than the other, and grasps less firmly. The diminution progresses in an intermittent course; complete paralysis lasting sometimes for a few minutes or hours, and then the power returning to a certain degree, and for a longer or shorter time; but general weakness is found outside the range of limited paralysis. The motorial changes may, however, be limited to particular groups of muscles; for example, those of the face, of speech, of one arm, &c. Spasm, of tonic character, exists with great frequency, and may be found in the paralyzed or non-paralyzed side, though much more commonly in the former. The rigidity increases gradually, and persists till within a few days of death, when it usually disappears altogether. Tremors or epileptoid convulsions may alternate with, or take the place of, tonic spasm; or there may be local clonic contractions, and the muscles may be unduly sensitive to percussion. General paralysis usually occurs for some days or hours before death; and then stertor, involuntary micturition, and universal flaccidity are present.

PATHOLOGY.—Since the year 1820 Softening of the Brain has received considerable attention from pathologists, and more especially from those of the French school. Previous to this time, it is true, such a pathological condition had been recognized and reported by several observers, and among them by Morgagni;[1] but their accounts are meagre and unimportant, so that for the first real description of the disease we have to refer to the works of Lallemand[2] and Rostan.[3] The first of these writers looked upon all softenings of the brain as of an inflammatory nature, and there can be little doubt that many of the cases he described were really instances of cerebritis of traumatic origin, associated with inflammation of the meninges; whilst Rostan, whose observations were made upon people of an advanced age at the Salpêtrière, thought these affections were sometimes inflammatory, and sometimes not. The latter, also, first called attention to the fact of the frequent association of softenings of the brain with

[1] De Sedibus et Caus. Morb. t. v., Epist. v. ix. lvii.

[2] Rech. Anat. Path. sur l'Encéph. 1re lettre, 1820.

[3] Rech. sur le Ramolliss. du Cerveau. 1820.

calcification of the arteries in old people. Since this time the opinions entertained by different writers as to the nature of softenings of the brain have been various, though for the most part they may be ranged under two principal categories, since the subject which has always been most in dispute (and which cannot now be said to be entirely settled) has been, whether we are to regard these affections as inflammatory or non-inflammatory in their origin. The great though pardonable error of the earlier pathologists was, that they looked upon Softening of the Brain as a single substantive disease, instead of regarding it, as we now do, as the pathological sequence of various more or less different conditions. And, as we shall hope to show, much of the difference of opinion amongst later pathologists has been engendered and propagated because they have looked at various kinds of Softening of the Brain too much from the mere point of view of morbid anatomy. Thus some have attached an undue importance to certain appearances the real nature and value of which could only be rightly estimated by a consideration of the pathology and mode of origin of the lesions in question.

Before dwelling upon this point further, I will briefly indicate the nature of the principal fluctuations of opinion amongst successive writers on this subject, whose works have followed those of Lallemand and Rostan.

Cruveilhier[1] held that certain forms of softening were of an inflammatory nature, but that certain others were not, these latter being supposed to be more allied to softenings of the stomach and intestines. His "ramollissement apoplectique," or "apoplexie capillaire," as he afterwards termed it (answering to some of the forms of red softening), he did not regard as inflammatory in nature, but he thought that this condition passed by almost insensible gradations into one of ordinary apoplexy. Bouillaud[2] at first proclaimed the inflammatory nature of softenings of the brain, though afterwards[3] he acknowledged the difficulty of the question and the desirability of further investigation on the subject. Andral[4] rejected the inflammatory doctrine. He spoke of obliteration of arteries and poverty of the blood as probable causes, but he also regarded the state of softening as due to a special alteration of nutrition which might supervene under the influence of the most different conditions. Abercrombie[1] looked upon softening as a species of gangrene, but spoke of two forms, one of which was essentially inflammatory in its origin, whilst the other—principally met with in old people—was due to disease and obstruction of the cerebral arteries. Much the same views were entertained by Carswell[2] and Copland ;[3] and others afterwards insisted, as Abercrombie had done, upon the importance of obliteration of the arteries in connection with Softening of the Brain. Amongst the earlier of these may be mentioned Bright,[4] Crisp,[5] Piorry,[6] Gely,[7] Gueneau de Mussy,[8] and Bouchut.[9] But there appeared in France, almost at the same time, two of the most decided advocates of the inflammatory nature of Softening of the Brain, namely, Gluge,[10] who founded his theory upon the supposed nature of certain granular corpuscles or cells, to which we shall subsequently have to refer, and Durand-Fardel.[11] Those who had already called attention to arterial obstruction as a cause of cerebral softening referred to coagulations occurring in the vessels themselves, but Virchow, in his first memoir upon embolism,[12] opened up a fruitful and entirely new field for inquiries into the pathogeny of Softening of the Brain, which has since attracted the attention of many investigators, whose labors have yielded the most important results. Amongst others who have contributed to elucidate this aspect of the question, I may mention Kirkes,[13] Fritz,[14] Schutzenberger,[15] Oppolzer,[16] Cohn,[17] Lancereaux,[18]

[1] Introd. à l'Etude de la Médecine pratique (1821) ler cahier, p. 112.—Anat. Patholog.—Dict. de Méd. et de Chir. prat., Art. "Apoplexie."

[2] Traité de l'Encéphalite, 1825.

[3] Dict. de Méd. et de Chir. prat. t. xv. p. 793.

[4] Précis d'Anat. Path. 1829, and Clinique Médicale, transl. by Spillan, 1836, p. 160.

[1] Path. and Prac. Research on Diseases of the Brain and Spinal Cord. 3d ed. 1836, p. 22.

[2] Path. Anat., Art. "Softening," and Cyclop. of Pract. Med. vol. iv.

[3] Dict. of Pract. Med.

[4] Guy's Hosp. Rep. No. 1.

[5] Lancet, 1840. Cases of Cerebral Disease.

[6] Bulletin Clinique.

[7] Gazette Médicale, 1838.

[8] Archiv. gén. de Méd. 1re Série, t. xxvi. p. 559.

[9] Actes de la Soc. des Hôpitaux, 1850, t. i. p. 43.

[10] Comptes Rendus, 1837, et Archiv. de Méd. Belges, 1840.

[11] Traité du Ramollissement, 1843, et Malad. des Vieillards. Paris, 1854.

[12] Archiv für Anat. und Physiolog. 1847.

[13] Med. Chir. Transact. vol. xxxv. p. 281.

[14] Gazette Hebdom. 1857.

[15] Gaz. Méd. de Strasbourg, 1857, p. 50.

[16] Wien. Med. Wochensch. 1859 and 1860

[17] Klinik der Embol. Gefasskrank. Berlin, 1860.

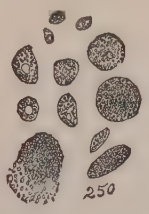
[18] De la Throb. et de l'Emb. Céréb. Paris, 1862.

and Hughlings Jackson :[1] of special importance also are the experimental investigations of MM. Prevost and Cotard.[2] Reference may, moreover, be made to the work of Lancereaux for further historical information, and to the recent volumes of the "Transactions of the Pathological Society of London" for numerous cases recorded by English pathologists.

In spite, however, of the light thrown upon the pathology of the disease by the recognition of the frequency of its association with embolism of the cerebral arteries, Durand-Fardel,[3] in 1854, again described the various softenings of the brain as inflammatory affections; and a few years later this view received the support of Calmeil.[4] Rokitansky[5]—as the representative of the Vienna school—also pronounced in favor of the inflammatory nature of red softenings; and amongst British Pathologists there are some who still look upon various forms of Softening of the Brain as inflammatory, principally from the fact of the occurrence, in the softened tissue, of Gluge's granule cells, which are erroneously supposed to be produced only by a process of inflammation. The latest French writers, however, seem almost entirely agreed as to the non-inflammatory nature of the great majority of cerebral softenings, as may be seen by reference to the works of Lancereaux,[6] Laborde,[7] and Proust,[8] and to the memoirs of Bouchard,[9] and of MM. Prevost and Cotard.[10]

The acute course run by many softenings of the brain, and the red and swollen appearance of the part after death, appear at first sight to lend support to the doctrine of their inflammatory origin, though, after recent inquiries into the effects of obstruction to the circulation in different parts of the encephalon, it will be found that we shall have no difficulty in otherwise accounting for these phenomena. It seems impossible now, moreover, to look upon the large granule corpuscles, or so-called "compound inflammation globules" of Gluge, as products of inflammation only. These bodies are constantly present in all but the most recent patches of softening, and it seems to have been the tolerably wide acceptance of Gluge's opinion as to their origin, that has kept up the doctrine of the inflammatory nature of cerebral ramollissement. In their most topical form these bodies present themselves as large, spherical, or somewhat elongated aggregations of minute granules, generally about $\frac{1}{1000}''$ in diameter, though they may vary from $\frac{1}{2000}''$ to $\frac{1}{300}''$ in diameter. They may be surrounded

Fig. 50.



by a delicate cell-wall, or this may be no longer visible. Occasionally a clear space, indicative of a nucleus, may be detected in their interior. From the fact that bodies in every way similar have been found by Turck,[1] Bouchard,[2] myself,[3] and others in secondary atrophic degenerations of the spinal cord, in which there is a simple process of wasting and not the slightest suspicion of the existence of an inflammatory process, there is the strongest evidence in favor of the opinion of Virchow, Robin, and other histologists, that these bodies result from the degenerations of pre-existing cells by the accumulations of fat and protein granules in their interior. They are, according to Virchow, produced from the cells of the neuroglia,[4] or connective tissue of the brain: and on this supposition we may easily account for the presence of granule corpuscles in the midst of the white substance of the brain, where formerly no cells were thought to exist. When these connective-tissue elements undergo the fatty and granular degeneration, they appear greatly to enlarge in size, the cell-wall becomes progressively thinner till it at last disappears, and ultimately the spherical aggregation breaks down into a mass of granular débris. It is thought also, by some, that granule corpuscles may originate in part by the aggregation of molecules originally separate, such as are always plentiful in tissues undergoing degeneration; and by others that they may arise from the granular and fatty degene-

[1] Lond. Hosp. Reports, vol. i.
[2] Recherches phys. et path. sur le Ramolliss. cérébrale. Gaz. Méd. de Paris, 1866.
[3] Loc. cit.
[4] Malad. Inflammat. du Cerveau. Paris, 1859.
[5] Path. Anat. (Syd. Soc.) vol. iii. 1850.
[6] Loc. cit.
[7] Le Ramolliss. et la Congest. du Cerveau. Paris, 1866.
[8] Des diff. Formes de Ramolliss. du Cerveau. Paris, 1866.
[9] Archiv. génér. de Méd., Mars, 1866.
[10] Loc. cit.

[1] Compt. Rend. Acad. des Sc. de Vienne, Mars, 1857.
[2] Archiv. gén. de Méd., 1866, p. 281.
[3] "On a Case of Concussion-lesion of the Spinal Cord, with extensive ascending and descending secondary degenerations." Med.-Chirurg. Trans. 1867.
[4] "Myélocytes" of Robin.

ration of the drops of myeline set free from the nerve fibres in softening nerve tissue. It must, however, be very difficult to substantiate either of these modes of origin.

The extreme vascularity of the brain, and its naturally soft consistence, must be taken into consideration if we wish to understand how it is that diminution in the nutrition of any of its parts, and the degeneration which is its accompaniment, should lead to such marked alterations in consistence as are met with in cerebral softenings. Bearing these peculiarities in mind, however, the lowering of nutrition from vascular obstruction, with its consequent effusion of serum, together with the degeneration subsequently taking place, seems adequate to explain all the degrees of ramollissement which exist during life. Theoretically we should be compelled to admit that Softening of the Brain might be brought about by (1) an improper state of the nutritive fluid or blood; (2) by a want of due activity in the elements of the tissues themselves; and (3) by an impediment to the proper circulation of the blood. As matter of fact, however, we can say nothing positive concerning the first cause, as to whether it is capable alone of producing a condition of cerebral ramollissement. It can probably be looked upon only as a predisposing cause, under the influence of which softenings might occur, in cases where there was a concurrent action of even slight determining causes. Where the vitality of the tissues has been lowered in anæmic and cachectic states of the system, such as we meet with in patients suffering from cancer, we can easily imagine that this poverty of blood would be a powerful predisposing cause, though we must also take into account the fact that these states of the system also tend to produce some of those changes in the vessels which are so frequently instrumental in bringing about Softening of the Brain. We can say little, also, that is definite as regards the influence of the second cause, though a primary fatty degeneration of the nerve elements leading to one form of softening has been described by Dr. Hughes Bennett.[1] A proper activity of the elements of the tissues themselves is certainly one essential in healthy nutrition, and to its gradual failure we may perhaps attribute the occurrence of many of the pathological changes characteristic of old age. Doubtless, both alterations in quality of blood, and diminished nutritive activity of tissue elements, may be looked upon as accessory causes of no unfrequent occurrence in the production of cerebral softening, especially in old people. But undoubtedly the most frequent causes of Softening of the Brain, at all ages, are to be looked for under the third head, which includes all the varieties of impediment to the circulation of the blood. These may be classified in the following manner:—

- Morbid conditions of cerebral vessels
  - Obstructing circulation
    - Arteries . . . Embolism. / Thrombosis.
    - Capillaries . . Embolism.
    - Veins and sinuses Thrombosis.
  - Preventing osmosis and nutritive exudation
    - Diseases of coats of capillaries and small arteries.

1. *Obstruction of Arteries.* *a. Embolism.*—The fibrinous masses of which emboli are composed have their origin for the most part in the left cavities of the heart and in the arch of the aorta, though more rarely they may proceed from the pulmonary veins. They may consist of portions of the fibrinous vegetations which are often met with on the mitral or aortic valves, as the result of endocarditis or atheroma, or of portions of those fibrinous depositions that are apt to form on the rough edges of atheromatous and calcareous patches of the arch of the aorta when these are not situated beyond the origin of the cerebral vessels; whilst at other times they may be constituted by detached portions of old clots which have been formed in the left auricle—or even in the left ventricle in cases where there has been a retardation of the force of the blood current, either owing to fatty degeneration alone or in combination with extreme dilatation of the heart. Virchow believes that cerebral emboli may proceed also from clots formed in the pulmonary veins; and it seems possible that small cancerous masses, swept away by the pulmonary veins in cases of carcinoma of the lungs, may occasionally go to form cerebral emboli, since in two instances small cancerous fragments have been found in the healthy heart and aorta—once by Lancereaux,[2] and once by Vidal.[3]

b. Thrombosis.—The various causes of thrombosis have been so well put by MM. Prevost and Cotard,[4] that we cannot do better than follow their arrangement.

(1) Pathological changes taking place in the walls of the arteries, by which their calibre is often much narrowed, and

[1] Clinical Lectures. Fourth ed. p. 355.
[2] Bulletin de la Soc. Anatomique, 1858.
[3] Comp. rendu de la Soc. de Biologie, 1861.
[4] Gaz. Méd. de Paris, Mai 19, 1866, p. 336.

their lining membrane roughened, are conditions most favorable for the occurrence of thrombosis. Changes of this kind, whether atheromatous or other, directly favor local coagulation.

(2) Retardation of the rapidity of the circulation also predisposes to coagulation of the blood in situations where other conditions favorable to its occurrence are present. This retardation may be brought about by: *a*. Diseases which weaken the force of the heart, such as dilatation without proportionate hypertrophy; and also, more especially, fatty degeneration of this organ. *b*. Narrowing of the calibre of arteries from atheromatous and other degenerations diminishes the rapidity of the circulation in these parts, and so predisposes to local coagulation, independently of the roughened surface with which the narrowing is usually associated. *c*. The loss of elasticity in the arterial walls, as a result of their degeneration, also assists in bringing about a retardation, since M. Marey has shown[1] that the elasticity of the arteries increases the rapidity of the blood current.

(3) And lastly, there are certain special states of the blood which seem to predispose towards the formation of arterial and venous thrombosis — sometimes so strongly as to bring this about even without an actual diseased condition of the vessels. This tendency is most marked in the cachectic states of the system before alluded to, and it is said to exist more especially in those cases in which the cancerous diathesis is well marked. Here, however, it seems probable, that the feebly acting, and perhaps degenerated heart, may be almost as instrumental in bringing about the coagulation, as any special alterations in the nature of the blood itself. There would be a conjoint action of these two predisposing causes.

All the conditions predisposing to thrombosis not unfrequently coexist in many old people, and this fact harmonizes well with the extreme frequency with which softenings of the brain, not due to emboli, are also met with in the same subjects. Any of the cerebral arteries may become the seat of degeneration in old age, so that thrombosis, and softenings due to this cause, may be met with in the most various regions of the brain. But the middle cerebral arteries are those which seem more especially liable to embolic occlusion, and, according to some observers, that on the left side is more frequently occluded than its fellow on the right. There still seems to be some doubt, however, as regards this latter point. Thus, in forty-four cases collected by Lancereaux,[2] although the left internal carotid and its branches were occluded fourteen times, and the right only twice, the left middle cerebral artery was affected twelve times, and the right twelve times; some arteries of the pia mater near the left cerebral peduncle once; and the basilar artery and its branches three times. In eighteen cases reported by MM. Prevost and Cotard,[1] the same frequency of occlusion of the middle cerebral arteries was found, though the numbers are higher for the right than for the left side. Thus, the right middle cerebral was occluded seven times, and the left three times; the right anterior cerebral twice, and the left twice; the basilar artery once; the right internal carotid twice, and the left once.

It should be mentioned also, that in the experiments of MM. Prevost and Cotard, in which they injected tobacco seeds into the carotid arteries of dogs, it was almost always found that the Silvian or middle cerebral arteries were more especially occluded. In the statistics of thirty-two cases reported by Meissner,[2] the situations are found to be somewhat different. Thus, the most frequent seats of obliteration were ascertained to be at the termination of the carotids. The obstruction was met with in this situation seven times in one of these arteries only, and twice in both at the same time. Next in order of frequency stood the posterior cerebral artery, in which the obstruction was met with eight times; then came the Silvian artery, seven times; the basilar artery four times; whilst in the vertebral it occurred once in one artery, and once in both; and in the artery of the corpus callosum twice.

In almost every case where Softening of the Brain is associated with thrombosis or embolism of the cerebral arteries, it is found that the obliteration exists in one of the branches beyond the circle of Willis,[3] even though obliteration of the parent

[1] Physiolog. Méd. de la Circ. Paris, 1863.
[2] Loc. cit. Mai 19, 1866, p. 337.

[1] Loc. cit. p. 19.
[2] Zur Lehre von der Thrombose und Embolie. Schmidt's Jahrbuch, 1861, No. 1, p. 89.
[3] On this subject Dr. Kirkes wrote: "Although by the arrangement of the vessels composing the circle of Willis ample provision is made against obstruction ensuing in any of the main arterial channels on either side *previous* to their arrival at the circle, there is comparatively little provision for an obstruction ensuing in any of the main branches into which this arterial circle breaks up. This remark applies chiefly to the middle cerebral artery, which, if plugged at its origin, becomes at once altogether useless as a bloodvessel, for nearly all its divisions, especially those for the central parts of the brain, proceed to their several destinations without receiving any anastomosing branch from the other divisions of the circle of Willis."— *Med.-Chir. Trans.*, 1852.

trunk also exists at some point before it gives off the branches for this anastomosis. Obliteration of the trunk of the carotid alone is not sufficient, under ordinary circumstances, to produce cerebral softening, as may be seen from a *résumé* by M. Ehrmann,[1] of cases in which, the carotid arteries having been tied, the operation was followed by cerebral disturbance. The symptoms of cerebral mischief at first set up gradually disappeared when the circulation was re-established by means of the circle of Willis; and where softening did actually occur, this was due either to the extension of a clot upwards, beyond the circle, into one of the cerebral arteries, or perhaps, as M. Ehrmann suggests, to some unusual distribution of the arteries themselves at the base of the brain, preventing the establishment of a collateral circulation, such as ordinarily takes place.

The seat of the softening also corresponds with the anatomical distribution of the branch occluded, though the two are never coextensive. Usually the brain in the peripheral portions of the vascular department is healthy, owing to this portion of its tissue being nourished by the collateral capillary circulation, whilst the central portions of the vascular region are principally affected: thus, as Lancereaux points out, in cases of obliteration of the Sylvian artery, softening of part of the corpus striatum and of the neighboring white substance is generally observed, whilst the gray matter of the convolutions as well as the walls of the ventricle are often intact. It has been suggested by Durand-Fardel that the obliteration of the arteries is secondary to the softening, and not the cause of it; but, in reply to this, it is only necessary to state that the actual seat of arterial occlusion is almost always outside the softened tissue, and in these cases, as well as in those in which there is obliteration of the arteries within the softened patch itself, an examination of the vessels will either show degenerated and roughened walls together with the presence of an adherent clot within, or else it will establish the existence of a small obstructing mass, differing from recent fibrine in composition and appearance, and unattached to the walls of the vessels.

2. *Obstruction of Capillaries.*—In certain cases, by the rupture of old clots of the heart having softened centres, or by rupture of the inner coat of the aorta over large softened atheromatous patches, a mass of granular débris is carried into the cerebral arteries, whilst, from the minute size of the particles of which it is composed, these penetrate to and block up the minute arteries and capillaries of the part. If the quantity of matter thus carried to the brain be considerable and widely dispersed, death may rapidly follow before there is time for definite alterations of the cerebral tissue to take place, and owing to the extent of the capillary obliteration the brain, it is said, may present an anæmic appearance. Such was frequently found to be the case by MM. Prevost and Cotard, when they injected fine lycopodium powder into the carotid arteries of dogs. When a smaller number of capillaries are obliterated, either by atheromatous matter, by small particles of fibrine, or by pigment granules,[1] local patches of softening may be produced, having the usual characters of Softening of the Brain due to arterial obstruction.

3. *Obstruction of the Veins and Sinuses.*—The general causes favorable to the production of thrombosis have already been mentioned. The cases of obliteration of the cerebral veins and sinuses are in part due to some of these, though, just as frequently, they are the sequences of blows on the head, or of inflammatory conditions of the scalp and cranial bones. Indeed out of the seventy-four instances of thrombosis in the cerebral sinuses which have been recorded by Lancereaux,[2] and other observers, such as Tonnelé,[3] Rilliet and Barthez,[4] Lebert,[5] Gerhard,[6] and Von Dusch,[7] thirty-nine are found to belong to this latter category. Amongst these, in no less than thirty cases it was due to caries of the bones of the skull; in so large a proportion as twenty-four of these cases it was the temporal bone that was affected as a result of otitis. [Dr. Lidell has given the history[8] of 130 cases of cerebral thrombosis. Of these, 86 were inflammatory in origin, 38 marasmic or due to debilitative causes, and 6 traumatic. Among the inflammatory cases, facial carbuncle was the most frequent cause. Otitis and erysipelas preceded thrombosis in a few cases. —H.] Both the lateral sinuses are seldom implicated at the same time in these secondary thromboses, and the longitudinal sinuses are even more rarely affected from such a cause; whereas in those cases in which the thrombosis proceeds from more general causes, such as alterations in the quality of the blood or slowness of circulation, its almost habitual seat is found

[1] Thèses de Strasbourg, 1859.

[1] Lancereaux, loc. cit. p. 106; Frerichs, Traité des Maladies du Foie, p. 264; and Charcot, Gaz. Hebdom. 1857, p. 659.

[2] Loc. cit. p. 116.

[3] Journ. Hebd. de Méd. 1829, p. 337.

[4] Malad. des Enfants, t. i. p. 161, 1853.

[5] Virch. Archiv, Bd. ix. p. 381.

[6] Deutsche Klinik, 1857, No. 45.

[7] New Syd. Soc. vol. xi. p. 81.

[8] American Journal of Med. Sciences, Jan. and July, 1874.]

to be the superior longitudinal sinus, from which the thrombus frequently prolongs itself down to the torcular Herophili and then on each side into the lateral sinus. It is in this latter class of cases, moreover, that cerebral softenings are associated with the thrombosis. These are of a peculiar kind, consisting principally of a number of small patches of red softening, occupying chiefly the gray matter on the upper surface of the brain; and they are often distributed symmetrically over both hemispheres. Occasionally, softening of a portion of brain tissue of considerable extent has been noted. Besides such peculiarities in the seat and distribution of the softened patches, we usually meet, in these cases, with serous effusion into the ventricles and beneath the arachnoid, or more rarely with an actual effusion of blood in these situations or into the substance of the brain itself, together with many minute patches of hemorrhage in the gray matter, such as have been described by Cruveilhier under the name of "apoplexie capillaire" The actual combination of these conditions met with in individual cases depends upon the seat of the obstruction, the rapidity with which it is brought about, and the condition of the vessels themselves. In the secondary thromboses, on the other hand, there is often evidence of more or less circumscribed inflammation of the meninges, although the cerebral softenings and extravasations of blood very rarely occur. This, according to Von Dusch, is owing to the fact that in these cases the thrombosis starts from the veins in communication with the inflamed spot, and reaches the sinus only after the collateral circulation has had time to establish itself, instead of forming at once in the sinus, and before a collateral circulation has been set up.

4. *Alterations in the walls of the Capillaries.*—Fatty degeneration of the walls of the capillaries has been described by Hughes Bennett,[1] Paget,[2] Todd,[3] Moosherr,[4] and Charles Robin.[5] This alteration is most frequent in old age, and is said to be especially common in individuals suffering from Bright's disease, or from other maladies producing a low cachectic state of the system. In some of these cases such changes may supervene at a much earlier period than is usual. It is thought that such changes may not only favor the occurrence of cerebral hemorrhage, but that they may also lead to softening when the changes are universal and well marked in the capillaries of a certain area. Such degenerations of the walls of the capillaries must not however be confounded with the accumulation of fat granules and of granule corpuscles on the walls of capillaries[1] which are situated in the midst of softened brain substance. The first state may possibly be a cause of softening, but the second condition is always a consequence of it.[2] The observations of Moosherr and Robin, more particularly, have shown that a certain number of fat particles may almost invariably be found within the sheaths of many of the small arteries and capillaries of the brain when this is quite healthy, and that, too, even in children. In many cases it is extremely difficult to discriminate between small fat particles and calcareous granules[3] in the walls of the capillaries, without submitting them to the action of dilute hydrochloric acid. This calcareous degeneration of the capillaries is more rare than the ordinary fatty degeneration, though when it exists in an extreme degree, it is also capable of giving rise to Softening of the Brain, as may be seen by the perusal of a remarkable case reported M. Delacour,[4] in which the small arteries and capillaries were completely calcified. In these cases, as well as in those of fatty degeneration, the softening is brought about by a gradual diminution in the nutrition of a portion of the brain, the capillaries of which have been altered in structure so as no longer to permit the osmosis of a quantity of blood plasma sufficient to maintain the ordinary balance of nutrition in the surrounding tissue, and to prevent it from undergoing processes of degeneration.

In addition to the various softenings of the brain, which may be produced by the influence of some of the conditions already mentioned, and others of traumatic origin, which are mostly "red," owing to effusion and dissemination of blood, there are also *secondary* or consecutive forms of softening, which may be classified under two heads, viz.: 1. Softenings set up around tumors and adventitious products generally, in the brain. 2. Atrophic softening due to the separation of nerve fibres from their ganglionic communications. The first variety of secondary softenings will be referred to elsewhere (Art. "Adventitious Products"). Those coming under the second head are by no

[1] Edin. Med. and Surg. Journ. 1842.
[2] Medical Gaz. 1849, and Surg. Pathol. (revised by Turner) 1863, p. 106.
[3] Clinical Lectures on Paralysis, &c. 1854.
[4] Ueber das Patholog. Verhalt. der Klein. Hirngef. Wurzburg, 1854.
[5] Compt. rend de la Soc. de Biolog. Paris, 1855, p. 142.

[1] Wedl, Patholog. Histol. (Syd. Soc.) p. 291, fig. 64.
[2] Billroth, Archiv der Heilkunde, Drit. Jahrgang, p. 47.
[3] Jenner, Med. Times and Gaz. January 31, 1862.
[4] Gaz. des Hôpitaux, 1850, p. 107; also Wilks, Journ. of Ment. Sc. vol. xi. p. 191.

means frequent; the lesion resulting from the separation of a tract of nerve-fibres from their central ganglionic connections being usually a simple atrophy or slow wasting. Although the method of degeneration, in this condition and in softening, has been proved to be identical, nevertheless actual cerebral softening does not usually occur, apparently because the atrophic change is brought about rather more slowly and without the occurrence of obstructions in the vessels of the part capable of producing œdema. Still, softenings from this cause have been met with. This kind of atrophic change was pointed out by Cruveilhier[1] in the cerebral peduncles, the pons, and the medulla oblongata, and since his time our knowledge of the process has been greatly advanced by the investigations of Turck,[2] Waller,[3] Van der Kolk,[4] Phillipeaux and Vulpian,[5] Gubler,[6] and Bouchard.[7] Laborde[8] has, moreover, quite recently stated that in cases where there is softening of the corpus striatum or of the optic thalamus, a similar process is also set up on the surface of the hemispheres in some related portion of the superficial gray matter of the convolutions. These softenings of the convolutional gray matter are stated to be always on the same side of the brain as the lesions in the central ganglia, and Laborde says he has also ascertained that a relationship exists between the particular convolutions affected and the particular portions of the central ganglia which have been destroyed, so that where softening of the anterior portion of the corpus striatum or optic thalamus exists, the same process occurs on some portion of the anterior convolutions; with destructions of the central portions, the middle convolutions are affected; and with destructions of the posterior portions of either of the central ganglia, a corresponding change is set up in some of the posterior convolutions. Should future observations confirm the opinions of Laborde, these changes would seem to be related to the secondary atrophic degenerations, and would be most interesting in a physiological as well as a pathological point of view: it is well to mention, however, that MM. Vulpian and Charcot maintain[9] that the coexistence of these peripheral and central lesions is a mere coincidence, and that there is no necessary association between them.

Seat of Cerebral Softening.—Foci of softening may be found in all parts of the brain, but they exist most frequently in the convolutions and most rarely in the cerebellum and in the pons Varolii. Durand-Fardel[1] found, as the result of an analysis of his own observations combined with those of Rostan, Andral, Raikem, and Lallemand—yielding altogether eighty-six cases of "acute" softening—that the convolutions were affected fifty-nine times; the corpus striatum and optic thalamus, either singly or combined, twenty-eight times; and the white substance of the hemispheres alone, only nine times. Although the combined statistics yield this result, however, it does not agree with the experience of Rostan[2] alone, who says expressly that the corpora striata and the optic thalami are the parts most frequently affected with softening, and that after them comes the central white substance of the hemispheres. Laborde says[3] that the corpus striatum is affected nearly twice as frequently as the optic thalamus, since he has found the former softened forty-six times and the latter only twenty-four times. He states also, that the change most frequently exists only on one side, and that it is rare for the two corpora striata to be affected at the same time without a corresponding change in one or other of the optic thalami. The two hemispheres are about equally liable to undergo such a process, though, as before indicated, the softenings of embolic origin seem to occur rather more frequently in the left hemisphere.

The extent of the softened patch is extremely variable: it may be found from the size of a pea up to such an extent that the whole of one lobe, or the greater part of one hemisphere even, may be so affected.

Periods of Life at which Softening of the Brain is most frequent.—Durand-Fardel has attempted to show by a combination of his own cases with those of Rostan, Lallemand, Bouillaud, and Andral, that Softening of the Brain may be met with at all periods of life, and that it is by no means a malady peculiar to old age. Laborde,[4] however, has subjected the same statistics to a more rigid scrutiny, and by eliminating various cases of inflammatory softening of traumatic origin, and others which have been improperly included from various causes, he has found that the very statistics made use of by Durand-

[1] Anat. Patholog.
[2] Comp. rend. Acad. des Sciences de Vienne, Mars, 1857.
[3] Nouv. Méth. Anat. pour l'Investig. du Syst. nerv. (Lett. à l'Acad. des Sc. 1852.)
[4] New Syd. Soc. vol. xi. p. 129.
[5] Mém. de la Soc. de Biolog. 1859, p. 343.
[6] Archiv. gén. de Méd. 1859, p. 31.
[7] Ibid. Mars, 1866.
[8] Le Ramolliss. et la Congest. du Cerveau, Paris, 1866.
[9] Physiolog. génér. et comp. du Syst. nerveux, Paris, 1866, p. 653.

[1] Malad. des Vieillards, Paris, 1854, p. 68.
[2] Ramolliss. du Cerveau, Paris, 1823, p. 161.
[3] Loc. cit. p. 72.
[4] Loc. cit. pp. 144–185.

Fardel do show, if properly sifted, that the most undoubted relationships exist between non-inflammatory Softening of the Brain and old age. In a total of eighty-one cases, seventy-five of the individuals were from sixty to seventy-five years of age, four from fifty to sixty, whilst only two individuals were between thirty and fifty years of age. It must not, however, be supposed that Softening of the Brain is so exclusively a disease of old age as these figures would represent, since softenings of embolic origin are now known frequently to occur in young adults and even in children; softenings from thrombosis, also, may and do occur occasionally in certain adults (at any age), who may be suffering from cachectic diseases. Still, degeneration of the cerebral arteries and the various other causes of thrombosis increase in frequency with increasing age; and so is it with non-inflammatory softenings of the brain, to which these conditions are bound in such a close pathological relationship.

MORBID ANATOMY.——Softenings of the brain present themselves under different forms according to their variations in seat, their age or duration, and other conditions; and the common character of diminished consistence, which first attracted the attention of pathologists, is curiously enough the one least indicative of a morbid change that has taken place during life, since marked alterations of this kind may be induced *post mortem*. The tests employed by earlier pathologists, therefore, which were intended to establish this one fact of diminished consistence, namely, the effect of a gentle stream of water in washing away the softened tissue, the impressions derived from the sense of touch, and the speedy rounding of the angles which takes place in a portion of brain cut out from the softened portion, are useless for the purpose of demonstrating that we have to do with a pathological process established during life. To make sure of this we must take into account other characters afforded by the softened tissue, and have recourse to more delicate means of investigation, such as are afforded by the microscope and the specific gravity apparatus.

The most notable differences of appearance in softenings of the brain are due to variations in color. The greatest variety of tints may be met with. In some cases the softened tissue has a dead white color; or it may be of a reddish hue, and may present all shades from a uniform pinkish tint, through different grades of red, up to a claret color; whilst, in other instances, various shades of yellow and even brown discoloration are met with. These colors may exist alone, or variously intermixed. But in spite of this apparent complexity, there are three principal kinds of "ramollissement"—known as the *white*, the *red*, and the *yellow*—under which heads it will be convenient for us to describe the various forms of Softening of the Brain. But in making this division it should be understood that we do not necessarily look upon these varieties as different stages of the same condition, as has been done by Durand-Fardel[1] and Lancereaux[2]—both of whom describe "ramollissement blanc" as the last or chronic condition of red softening. Lallemand also looked upon white softening as due to a purulent infiltration, though the absence of pus has been established again and again by later observations. On the other hand, MM. Hardy and Béhier,[3] as well as Rostan and Abercrombie, have pointed out that this kind of softening is often met with in old people, and more particularly in those who are weakened and feeble, which agrees well also with the observations of M. Charcot, who has often met with white softening in old people afflicted with cancer at the Salpêtrière.[4] MM. Prevost and Cotard, moreover, admit the existence of a Softening of the Brain, white from the first; and this has also seemed to be the case in several instances which have fallen under our own notice. It would appear probable that white softening is mostly due to a rather more chronic process than that which gives rise to the red, and that it is such as would exist if this change had been brought about by disease of the coats of the capillaries, supplemented by general mal-nutrition from poverty of blood. Prevost and Cotard[5] suggest, also, that in some of those cases, where the softening seems to be associated with obliteration of vessels, the absence of redness may be due to the extent and nature of the arterial obliteration being such as to prevent the collateral fluxion of blood into the vessels of the diseased part. In other cases they admit that the cause seems inexplicable. Red softening seems to be more particularly limited to cases in which there is arterial obstruction, or impediment on the venous side of the circulation; whilst there can be little doubt that many of the shades of yellow in softenings of the brain are due to alterations that have taken place in what was previously red softening, although Rokitansky has described a special kind of yellow softening having intrinsic characters of its own.

1. *White Softening.*—In this species of ramolissement, which occurs only in the white substance of the hemispheres, a

[1] Malad. des Vieillards, Paris, 1854, p. 72.
[2] Loc. cit. p. 20.
[3] Traité de Pathol. int., Paris.
[4] Vide Proust, loc. cit. p. 49.
[5] Gaz. Méd. de Paris, 1866, p. 207.

varying amount of diminution of consistence is met with, without alteration of color. In some cases the amount of softening is so slight as to escape detection by the unaided senses, whilst in others the portion of brain is reduced to a thin diffluent pulp, which may be poured out so as to leave a distinct cavity with irregular though softened walls—for the degeneration is never strictly limited, it shades off imperceptibly into the healthy tissues. Thus, in what appear to be minute circumscribed patches of white softening, granular corpuscles are diffused for some distance in the firmer tissue surrounding the softened patch. Sometimes nearly the whole of the white substance of one hemisphere may be found in a more or less softened condition. The specific gravity of softened white matter usually falls to 1·032, but when in its natural condition it is about 1·040; and as a general rule it may be said that any portion of the brain when softened has a specific gravity lower by six to eight degrees of the hydrometer scale than the specific gravity of the same part when in its normal condition.[1]

2. *Red Softening.*—This variety may be met with either in the gray matter or in the white substance of the brain, though in the former the coloration is generally more distinct and of a darker hue, on account of the naturally greater vascularity of the brain substance in this situation. When affecting the gray matter, the patches may be small and distinctly circumscribed, so as to present the appearance of superficial ulcerations; or they may be more diffuse and extensive, presenting less of the appearance of ulcerations, but existing as dark, somewhat swollen patches, which from their color were formerly spoken of as "plaques ecchymotiques," and "taches scorbutiques des convolutions." These have also been described by Cruveilhier[2] under the name of "ramollissement *hortensia* ou *lilas*." This softening of the convolutional gray matter may exist alone, it may be associated with a similar change in the subjacent white matter, or, as Laborde has pointed out, it may coexist with softening of one of the central ganglia on the same side. In these latter cases the softening is often slight, and takes place without much alteration in the natural color of the part, so that it is liable to be overlooked. Where the convolutions are softened, the gray matter is frequently torn in attempting to strip off the membranes. This may be due, however, to the mere fact of diminished consistence, rather than to any increase in the natural adhesions existing between the parts. Red softening of the white matter often exhibits a general rosy hue, intermixed with darker-colored patches from effused blood and minute points of redness, which have been described as "capillary apoplexy." It may be diversified also with patches of simple white softening here and there, and after a certain time shades of buff, yellow, and even brown coloration supplant or become mixed up with the red. The brown color is most frequently met with in old patches of softening in the corpus striatum. The red color being due to the increased quantity of blood in the part, owing to extreme congestion of the vessels (combined with staining from transudation of coloring matter), we have, in this fact, an explanation of the circumstance that when a recent red softening is cut into, the surface often rises up above the level of surrounding parts, and presents a slightly swollen appearance. Also, when red softening of the white matter exists combined with a similar condition of several of the contiguous convolutions, owing to the swelling and consequent pressure thus produced, these become flattened on the surface, whilst the sulci are rendered indistinct. The process of degeneration of the nerve tissue is the same in this form as in the simple white softening, and it may advance to the same condition of diffluence. It is only that the color and composition of the softened part in a case of red softening are altered by the great admixture of blood together with the products of its retrograde metamorphosis.

Red softening, if not of traumatic origin, as before stated, is almost invariably connected with obliteration of the vessels or other impediment to the circulation, and the "redness" or "swelling," which were formerly considered so indicative of its inflammatory origin, are capable of receiving an altogether different explanation, tending to show that these characteristics are dependent upon mechanical rather than upon vital influences.

It has been known for some time through the writings of Rokitansky and Cohn,[1] that infarctus of the abdominal viscera, owing to arterial obstruction, commences as a deep red spot in the territory of the affected artery, this appearance being due to the engorged condition of its capillaries, and the occurrence of minute extravasations of blood. And, quite recently, MM. Prevost and Cotard having injected into the arterial system of a dog (whose abdominal walls had been opened so as to expose its contained viscera) some water holding tobacco-seeds in suspension, in a few moments after the injection there appeared, at the inferior extremity of the

[1] "On the Specific Gravity of different parts of the Human Brain:" Journ. of Ment. Sc., Jan. 1866.

[2] Anat. Patholog. vol. i. Livraison.

[1] Klinik der Embolisch. Gefässkrankh., Berlin, 1860.

spleen, a red prominent spot of definite outline, which rapidly increased in size till it equalled that of a two-franc piece. The corresponding branch of the splenic artery was afterwards found to be obliterated by some of the seeds. Ecchymotic-looking spots were produced upon the kidneys at the same time. What was here actually seen to take place accords perfectly with what is observed in cerebral softenings brought about by the injection of foreign bodies into the arterial system. Their impaction in the cerebral arteries constantly results in the production of red softenings with great fulness of the vessels, as has been abundantly proved by the experiments of Cohn, Vulpian, and by those of MM. Prevost and Cotard. Here, then, we have the two appearances, redness and swelling, produced so rapidly as to make it quite out of the question for us to regard them, in spite of the opinion of Oppolzer, as of inflammatory origin; so that we must seek for some mechanical cause of the phenomena. Cohn and Rokitansky attribute the results to a collateral fluxion of blood through the contiguous capillaries, whilst Virchow regards them as the effects of a venous reflux, in consequence of the suppression of the *vis à tergo* on the side of the artery. In support of the former view, Weber[1] has since pointed out that when an artery is obliterated, the pressure at the point of obliteration increases, and becomes equal to that at the origin of the artery, and that when a certain number of capillaries are obliterated, the pressure augments in the artery belonging to them, and in those remaining pervious. Prevost and Cotard also support this view, and they call attention to the theorem of hydrodynamics upon which it depends, to the effect, that when a tube receives at one of its extremities a fluid at a certain pressure, and allows it to escape freely at the other extremity, the pressure diminishes from one end to the other of the tube according to an arithmetical progression. Marey[2] has shown that this theorem is applicable to the circulation of blood, owing to the resistance in the veins being so slight as practically to make it appear as though there were a free flow into the capillaries.[3] This increased tension of blood in the collateral capillaries would, therefore, seem to account in great part, not only for the surrounding congestion, but for the flux of blood into the territory of the obliterated artery, where it would stagnate; and, owing to the increased tension of the blood (combined with the progressive weakening of the capillary walls, and the diminution of their usual support from the softening of the surrounding brain tissue), we can easily conceive the mode of origin of those effusions of blood and those dilatations of capillaries such as are actually encountered.

3. *Yellow Softening.*—A special form of degeneration of brain substance has been described under this head by Rokitansky.[1] It usually occurs in sharply circumscribed spots—varying in size, but rarely exceeding that of a hen's egg—in which the cerebral substance is converted into a very moist, tremulous, and occasionally gelatinous pulp, retaining none of the characters of proper brain tissue. The altered portion rises considerably above the level of the section, and is of a straw or sulphur-yellow color. In slighter degrees of the disease, the color is merely dull white, inclining to yellow, though the tissue is still much softer and moister than usual. The expressed fluid has a distinctly acid reaction. The transition to the healthy brain tissue is usually abrupt, and there are no signs of inflammation in or around the softened patch. Its usual seat is the white matter of the hemispheres, though it may affect the central ganglia, or, much more rarely, the convolutional gray matter. With regard to the pathogeny of this form of softening we are almost entirely in ignorance. Rokitansky looks upon it as a peculiar chemico-pathological transformation of brain substance, in which "the liberation of an acid—the phosphoric, and especially one or more of the fatty acids—may be conjectured to be one of the most important phenomena." Besides occurring as an independent condition such as above described, this form of yellow cerebral softening is said by Rokitansky to exist frequently around old clots, tumors, or other adventitious products in the brain. Cruveilhier[2] has also frequently met with it around old adventitious products, though he has never seen it existing alone.

Besides this special form of yellow softening, as before stated, we meet with a yellow color of the brain substance in the secondary stage of red softening, in which case the tint may be partly due to the presence of altered blood pigment, and partly, according to Lancereaux and Virchow, to the presence of what were the red globules of the blood, but which have now, in part, been deprived of their hæmatine.

Histological Alterations and Microscopical Appearances in different Stages.—The

[1] Handbuch der Allgem. und Spec. Chirur. 1865.

[2] Physiolog. Méd. de la Circ., Paris, 1863.

[3] This is an important point, because Poiseuille (Recher sur les Causes du Mouv. du Sang) maintained that the pressure was the same in all parts of the arterial system.

[1] Path. Anat. (Syd. Soc.) vol. iii. p. 419.

[2] Anat. Path. vol. i. Livraison viii.

changes which take place in the nerve elements of a part, whether from the separation of these from their proper ganglionic connections, or from the cutting off of the supply of blood to the part, have been shown to be absolutely identical in nature, and to differ only as regards the rapidity with which the change is brought about. This has been proved by the experiments and observations of Phillipeaux and Vulpian on the changes taking place in the peripheral extremities of divided nerves, and by the observations of Turck, Gubler, and Bouchard on the progress of secondary degenerations of the spinal cord, as compared with the ordinary histological changes which take place in softening nerve substance. In the one case we have a simple destruction, or necrobiosis, taking place amongst the elements of the tissue, in which, from the comparatively slow way that they are brought about, all the steps of the process may be easily traced, whilst in the other we have a more rapid and tumultuous form of necrobiosis, which, on account of its rapidity, is associated with diminished consistence. The investigations of MM. Phillipeaux and Vulpian[1] have yielded the following results. At the end of the first day there is found to be a diminution of the proper excitability in the peripheral extremity of the cut nerve, whilst this is lost altogether at the end of the fourth, and the filaments which had previously shown no change begin to alter in appearance. From the fifth day the medullary substance of the tubes seems to be coagulated, and at the same time fissures establish themselves in its thickness and divide it into unequal blocks or divisions. This is called the segmentation of the nerve tubes. Soon after this the fragments of the medullary substance undergo a further alteration, fatty granules[2] form in their interior, which go on increasing in quantity, and at last entirely replace the fragments of myeline. Bouchard[3] has recognized precisely the same changes in cases of secondary atrophic degeneration, and, speaking of one of these cases, he says: "Independently of the fatty granules contained in the altered tubes, a great number were free between the tissue elements, and, at certain points, aggregated together into masses, so as to constitute what are known as the 'corps granuleux' of Gluge." In the same case, the vessels presented on their surface heaps of molecular fat particles, or even a complete envelope of these, so as to render the vessels black and opaque under the microscope. Here, then, are produced, without the intervention of inflammation, all the appearances which have been supposed to be characteristic of inflammatory Softening of the Brain. Dr. Hughes Bennett says:[1] "*Exudative or inflammatory softening* always contains granules and granule cells, which are numerous, according to the degree of softening. The granules are for the most part seen coating the vessels, and the cells also may occasionally be seen there in various stages of development. In the demonstrations that are made under the microscope, they are frequently seen diffused among the tubes, which, according to the severity and extent of the lesion, are easily separated from one another, or broken up in a variety of ways."

MM. Prevost and Cotard have found from their experiments on dogs, that at the end of the first twenty-four hours after the obstruction of an artery there was red pulpy softening, with slight diminution of consistence, and, on examination with the microscope, there were seen broken-up fragments of nerve tubes, drops of myeline, blood corpuscles, and peculiarities of the capillaries, though no granules or granular corpuscles were at that time visible. As early as the third day, however, they have found granule corpuscles formed, and an abundance of granular matter lying amongst the tissue elements, as well as more especially aggregated along the walls of the vessels. These results are quite in accordance with our own observations, since we lately met with an instance of traumatic softening in which a few fully developed granule corpuscles, and very many in a less mature state, were seen, which must have been produced in rather less than two and a half days.[2] Bouchard believes that these granule corpuscles may result from the "granulo-graisseuse" degeneration of drops of myeline, and Prevost and Cotard

[1] Mém. de la Soc. de Biologie, 1859, p. 343.

[2] At the end of a certain time the fatty granules are absorbed, and nothing is left but the sheath of Schwann folded on itself and on the axis cylinder. Little is known as to the actual condition of this last, though the researches of Schiff and MM. Phillipeaux and Vulpian go to prove that the return of function in a divided nerve proceeds not from the production of new tubes amongst the débris of the old, as formerly supposed, but from the reformation of myeline within the wasted sheaths themselves, and around the old axis cylinder, which is capable of persisting for a long time without undergoing much appreciable alteration.

[3] Arch. Gén. de Méd. 1863, p. 281.

[1] Clinical Lectures. Fourth ed. 1865, p. 354.

[2] The man on whom this observation was made fell down an area and fractured his skull. He was admitted into St. Mary's Hospital on September 7, 1866, at 4 P. M., immediately after the accident, and died on the 10th of the same month at 3.10 A. M. The exact interval was, therefore, 2 days, 11 hours, 10 min.

also think they may result from the aggregation of granules originally separate. We have, ourselves, never been able to substantiate either of these modes of origin, and we agree with Virchow[1] in the opinion that they mostly originate from the fatty degeneration of the cells of the neuroglia, since granule corpuscles are commonly met with in the midst of the white matter of the hemispheres, having a more or less distinct cell wall, and which show a large nucleus in their interior after staining with carmine.[2] The cells of the neuroglia are the only elements existing in this situation capable of giving rise to such bodies.[3] They are also to be seen in the gray matter lying between the ganglion cells, which, in old age, undergo more or less of the pigmentary degeneration, and always present quite a different appearance. Robin formerly held that these granular corpuscles were produced by the degeneration of pus cells; but pus cells in their natural state are never met with in simple Softening of the Brain, and it seems scarcely fair or reasonable to assume that they should be seen only in a state of degeneration. In cases of softening of the convolutional gray matter or of the central ganglia, a degeneration of the proper nerve cells takes place, which become filled with dark-colored granules. These are generally at once distinguishable from ordinary granule corpuscles by their irregular, angular shape, and by the presence of the stumps of one or more cell prolongations. At the same time that these bodies are forming in the degenerating tissue, granules collect along the walls of the capillaries, partly in an altogether irregular manner, and partly in the form of more or less spherical aggregations. Some of the capillaries become completely covered in this way; but the collection of granules is *on* the walls of the capillaries, and is a consequence, not a cause of the softening. It must not be confounded with fatty degeneration of these vessels, in which the granules are imbedded *in* the walls of the capillaries. There are other alterations of the capillaries met with, especially in red softenings, which have been particularly dwelt upon by Laborde.[4] At first, partial dilatations of the walls of the capillaries are seen, like minute aneurismal swellings, or, in other places, little ampulliform dilatations including the whole circumference of the vessel, and constituting what he describes as the *moniliform* condition of the capillaries. At a later stage, complete as well as partial dilatations of the capillaries are to be seen, together with actual ruptures here and there, and minute extravasations of blood. Still later, the capillaries become enormously dilated, and their walls thin and granular from degeneration. The punctiform hemorrhage, to which Cruveilhier gave the name of "apoplexie capillaire," is sometimes due to minute extravasations from rupture of the capillaries, sometimes to the extreme dilatation of capillaries gorged with blood, and often to the production of what has been wrongly called "dissecting aneurism," occasioned by rupture of the proper wall of a minute artery and an effusion of blood into the lymphatic sheath which surrounds it. The blood remaining in the capillaries, and also that effused externally amongst the nerve elements, shows, for certain time, traces of the individual blood corpuscles, more or less decolorized and yellow, as well as flattened and pressed together; whilst mixed up with them are reddish or reddish-yellow flakes of tissue, stained by the transuded hæmatine. In those minute patches of extravasated blood, in which the coloring matter exists in some quantity, we afterwards find it in the form of amorphous, yellow or orange-colored granules or flakes, intermixed with the characteristic orange or ruby-colored crystals of hæmatoidine. These are very minute, and of an oblique rhomboido-prismatic form. It is not known exactly in how short a time these crystals may appear in extravasations of blood in the human brain. Dr. Wilks[1] has, however, met them as soon as three weeks after such an occurrence, and Cruveilhier found the "coloration jaune orange" developed after twenty-five days in the seat of an hemorrhagic effusion into the brain. Once formed, the hæmatoidine crystals remain as indelible evidences of past extravasation of blood.

In the extreme stage of softening, the fluid matter occupying its site no longer presents the slightest trace of nerve structure—the degeneration is complete, and nothing can be recognized by the microscope save granules and granule cells, mixed up with the various kinds of blood pigments, amorphous fragments of tissue, and the débris of degenerated vessels. When we have to do with the last stage of red softening, and especially when this is situated in the corpus striatum or optic thalamus, the contents of the softened centre may present a brownish or even chocolate hue.

[1] Wiener Medicin. Wochenschr. January 19, 1861.

[2] Case of Concussion Lesion, Med.-Chir. Trans. 1867.

[3] Corpuscles almost precisely similar are met with in other organs, whose tissues are in a state of degeneration, which undoubtedly originate from the fatty and granular degeneration of pre-existing cells.

[4] Loc. cit. p. 114.

[1] Lect. on Path. Anat. 1859, p. 133.

Fatty degeneration of the tissues being complete, the process of repair begins at a variable period—probably in from one to two months after the commencement of the degeneration. These alterations have been fully described by Durand-Fardel,[1] and differ according as they are situated at the surface of the brain, or in its central parts. In the former situation the process results in the formation of the so-called "plaques jaunes," and in the latter it is accomplished by what Durand-Fardel calls "infiltration celluleuse."

These so-called "plaques jaunes," which have been well represented by Cruveilhier,[2] exist in the form of yellow or ochre-colored, rounded patches. They may be confined to a single convolution, or may extend over several, at the same time dipping down into the sulci. The pia mater over them may sometimes be easily stripped off, whilst at other times it is closely adherent to the tissues beneath. The substance of the patch, though pliable, is tough and resists the knife; it usually implicates the cortical gray matter only, and its circumference is pretty sharply defined from the surrounding healthy tissue. More rarely, however, it is separated, as well circumferentially as beneath, from the healthy brain substance, by a layer of softened tissue. Histologically, these patches are composed of connective tissue containing an abundance of nuclei; also of intermingled hæmatine granules and crystals of hæmatodine, together with fatty particles, a few granule corpuscles, and some degenerated vessels. Rokitansky[3] denies that these yellow patches are the sequelæ of softening of the convolutions, and looks upon them as changes resulting from superficial hemorrhage. But the result of a recent experiment by Prevost and Cotard goes strongly to support the view of Durand-Fardel. They found a well-marked yellow patch on the middle lobe of one of the hemispheres of a dog, which is in every way similar to those met with in man; and the corresponding middle cerebral artery of this dog had been obliterated, thirty-five days before the death of the animal, by the injection of tobacco seeds into the carotid artery. From what we ourselves have seen, however, we are inclined to think that superficial extravasations of blood into the pia mater may also, as Rokitansky says, give rise to yellow patches, though of a different kind from the "plaques jaunes" described by Durand-Fardel. In cases where a superficial hemorrhage has been the antecedent condition, the coloration is almost entirely due to an accumulation of blood pigment in the meshes of the pia mater, with atrophy of the subjacent convolution, rather than to a fibro-cellular conversion of the substance of the gray matter itself.

When a focus of softening in the midst of the white substance of one of the hemisheres begins to undergo the process of repair, the walls of the softened cavity become bounded by a pulpy tissue of a white or grayish color, which, on microscopical examination, is found to be continuous with the neuroglia of the contiguous healthy portion of the hemispheres. Tissue of the same kind also extends across the cavity in different directions, breaking it up into divisions or compartments, in the meshes of which may be found a whitish liquid containing fragments of nerve substance which have not yet completely undergone the fatty metamorphosis. This fluid holds in suspension, also, fat particles, and a number of corpora amalacea. The formation of the vascular and nucleated connective tissue constitutes the "infiltration celluleuse" of Durand-Fardel. Though met with principally in the white substance of the hemispheres, it is also seen more rarely in the central ganglia. When situated in the corpora striata, the walls, instead of being white, are often of a yellowish or ochre color, which makes the identity of this process with that which gives rise to the "plaques jaunes" of the convolutions all the more evident. The fluid contents of the cavity gradually become absorbed, and its walls close in and contract in the same way as do those of an apoplectic fit. Indeed, in these last stages there may be some difficulty in discriminating between the two. In the remains of the apoplectic cyst, however, more coloring matter is usually found; its walls are also generally more dense and contractile, and a more complete obliteration of the old cavity is said to follow.

Lastly, there is a condition of the central ganglia of the brain, more particularly of the corpora striata, which has been described by Durand-Fardel[1] under the name of "état criblé." On making a section of these central ganglia, small pisiform cavities or lacunæ are occasionally seen, which sometimes seem bounded by a distinct membrane. Similar cavities may also be seen, though more rarely, in the pons Varolii. These are regarded by Laborde[2] and others as minute apoplectic cysts, resulting from slight effusions of blood, whilst others again look upon them only as dilatations of the lymphatic canals,

[1] Malad. des Vieillards, Paris, 1854, p. 72.
[2] Anat. Path., Livraison 33, pl. 2.
[3] Patholog. Anat. (Syd. Soc.), pp. 394 and 416.

[1] Who, however, attaches little importance to this condition, and looks upon the little cavities as the results of dilatations of the vessels, owing to long-continued congestion.
[2] Loc. cit. p. 94.

in which, as pointed out by His,[1] the cerebral vessels are contained. Laborde, however, thinks there is another and more important modification of this condition, in which no lining membrane is to be met with, but in which the little cavities are somewhat larger, so as to be even capable of containing a good-sized pea. These he looks upon as the result of "une désorganisation partielle et progressive," and as true, though minute and circumscribed, softenings of the parts in which they are found.[2] [In regard to localization of cerebral lesions, see the article on Cerebral Hemorrhage and Apoplexy, in this volume.—H.]

It only remains for us now to notice the softenings which have a post-mortem origin, and to point out how these may be distinguished from those having a real pathological significance which we have hitherto been considering.

Ordinary post-mortem softening of the brain is due to the combined influence of two causes; namely, putrefactive changes, and the maceration of the cerebral tissue from absorption of fluid.[3] This is commonly met with on the surface of the thalami and in the parts bounding the posterior portions of the lateral ventricles, in all those cases where an interval has existed between the death and the autopsy, and more particularly when the atmospheric temperature has been high and the ventricles have contained an excess of fluid. In these cases the surface of the parts affected is broken up, and presents an irregular appearance, whilst the tissue itself is in a more or less diffluent condition. The fornix also frequently shares in this change. It has been a subject of dispute as to what is the nature of the process which gives rise to the softening of the central parts of the brain in acute hydrocephalus—whether, in fact, it has been produced by inflammation, or is merely the result of maceration; and in the event of the latter method of pathogenesis being the real one, whether this maceration has occurred during life or after death. Doubts have been expressed by many pathologists as to whether such a process of maceration ever occurs during life.[1] When merely macerated nerve tissue is examined by the microscope, broken up and dissociated nerve elements only are met with, and none of the granule corpuscles or other appearances characteristic of real softenings that have been produced during life. Examined by the specific gravity apparatus also, we have several times found the actual density of the altered tissue the same as that of contiguous unaltered portions. This is somewhat remarkable and becomes very characteristic; since if a portion of brain tissue having a similarly diminished consistence, brought about by a pathological softening rather than by a post-mortem maceration, had been examined, the specific gravity would have been found lower than that of similar healthy tissue in the same brain by from eight to ten degrees of the hydrometer scale. The specific gravity test thus becomes a most important auxiliary to the microscope; and we have several times found it most useful in examinations of the spinal cord. Thus, a short time since, on making sections of a cord through the cervical, dorsal, and lumbar regions respectively, the surfaces exposed were quite pulpy and irregular in the two former regions, whilst in the lumbar portion the surfaces were firm and smooth. Yet the specific gravity of portions of the cord from the dorsal region was the same as that of other portions from the lumbar region, whilst in the cervical region the specific gravity was even slightly higher. Microscopical examination, moreover, yielded no evidence of a pathological change in any portion of the cord. We have found much the same state of things also in other cases. Diminished consistence or diffluence, therefore, must not be confounded with diminished density or specific gravity; and it

[1] Zeitsch. für Wissen. Zoolog. 1865. Bd. xii.

[2] Laborde says: "Nous possédons plusieurs observations de ces curieuses désorganisations partielles siégeant au centre de la *protubérance annulaire* et paraissant répondre au point de vue symptomatique, à certains cas de paralysie diffuse, généralisée, dans laquelle s'éteignent progressivement un grand nombre de vieillards."—P. 95.

[3] Dr. Bennett calls attention, in his "Clinical Lectures," to the softenings which may be produced by mechanical means, owing to the clumsy use of instruments in removing the brain and spinal cord from the body. This mode of origin should also be borne in mind.

[1] The impediment to the return of blood through the venæ magnæ Galeni, owing to thrombosis in these vessels, to which the collection of fluid in the ventricles is in part due, also gives rise to a condition of œdema in the walls of the ventricles themselves, and is followed by a true degenerative softening of the brain tissue. (Pathology of Tuberc. Mening., Edinb. Med. Journ., April, 1867.) Respecting this condition of œdema of the brain, however, which it may be presumed occurs occasionally in heart disease and other conditions impeding the return of blood from the head, we have no very definite knowledge. It seems doubtful whether any amount of serous infiltration would be capable of producing actual softening during life, or do more than make the brain appear flabby—a little moister and softer than usual—and, at the same time, slightly lower its specific gravity. The brain is usually said to be "wet" when in this condition.

should be remembered that it is the combination of the two, associated with certain microscopical changes, which are the characteristics of real pathological softening of the brain.

Diagnosis.—1. *Acute softening* may, in its *apoplectic form*, be confounded with congestion of the brain, with hemorrhage, or with urinæmia; but by regard to the mode of onset of the symptoms, and to their proportion *inter se*, a diagnosis may be established in the majority of cases. At the onset of attack it may be impossible to distinguish the nature of the malady, but after a few minutes, or perhaps only after two or three hours, it is possible to aim at something like certainty.

From *congestive apoplexy* softening may be distinguished by the longer duration of mental obtuseness; and by the distinct limitation of intellect in one or two directions, when the general obscuration of the "fit," or "stroke," has passed away. At the onset of attack in congestive apoplexy there may be complete loss of consciousness, and the same thing may occur at the commencement of acute ramollissement, and for precisely the same reason, viz. the presence of congestion. In the former case, however, the mind rapidly recovers, and is restored to its previous condition; in the latter all that was due to mere congestion is speedily removed, but there remains the impairment due to softened tissue. This may be aphasia, or some other special alteration in the mental powers, such as have been described in the section upon symptoms. Attacks of softening differ still more distinctly from those of congestive apoplexy, when they are unattended by any of the phenomena of hyperæmia, and occur after middle life, and especially in advancing years. The patient becomes more or less suddenly confused, but does not lose his consciousness; he may wander in his talk, utter some exclamation of alarm, or may simply look distressed and as if about to cry; he knows what is said, and makes signs to those about him; is obviously aware that something very wrong has happened, and continues in this state of mental impairment for hours, weeks, or months. The difference from congestion is seen in the primary absence of general mental change —loss of consciousness—and in the persistence of limited intellectual failure.

The two classes of change in function, now described with regard to mind, are to be observed also in respect of sensation and motility. There may be, at the moment of attack, general anæsthesia and general paralysis; but if so, they are due to congestion or to shock, and they, with either of those conditions, soon pass away; leaving behind them, however, impaired sensation in one or two limbs, and with this, localized paralysis. On the other hand, there may be no general change in the power of feeling or of motion—there may be neither congestion, nor sufficient shock—the patient feels some numbness, coldness, or deadness, together with weakness of one or two limbs, and these conditions pass on into hemiplegia, *i. e.*, loss of power, and loss or diminution of sensibility in the arm and leg of the side opposite to the lesion. Beyond these facts there is not unfrequently some hyperæsthesia or morbid sensibility of the paralyzed limb, and this is much more common in cases of softening than in those of congestion; and, again, there is twitching of the limbs, or rigidity of those that are paralyzed.

The general aspect of the patient differs from that of the person struck with congestive apoplexy. There is, unless congestion be present as a transient condition, pallor instead of dusky redness, coldness of the head instead of heat, and a faint look in the place of bloated suffocation; there is often some sweat upon the brow; the patient is spare in habit, and the attack occurs when sitting quietly at the desk or when making no such exertion as could tax the physical powers. Beyond these general conditions there may be observed rigidity of arteries, an irregular, weak, and often intermitting pulse, a feeble heart, arcus senilis, and irregular distribution of bodily warmth.

From *hemorrhagic apoplexy* acute softening may be sometimes distinguished by the following considerations:—In hemorrhage there is often some evidence of either congestion or of shock; in softening there may be an entire absence of both. In hemorrhage the attack frequently occurs at night; the patient goes to bed apparently well, and wakes in the morning feeling as usual, but on attempting to move finds that one side is paralyzed. In hemorrhagic apoplexy the attack is often absolutely instantaneous, in softening it is gradual. In the former there may be not the least—even momentary—confusion of mind, whereas in the latter there is distinct mental perturbation and insufficiency. In hemorrhage, when the intellect is profoundly affected, as it sometimes is at the onset of attack, there is often a rapid restoration, and in the course of a quarter of an hour the patient's mind is as clear as it was before; in softening *per se* there is less distinct mental obscuration at the commencement, and little or no subsequent recovery. In hemorrhage there is sometimes alarm, and not unfrequently anxiety and depression, whereas in softening there is more commonly too much confusion of thought for any definite apprehension to be entertained, and sometimes there is transient excitement or mild delirium.

Sensibility is often unaffected in hemor-

rhage, it rarely escapes altogether in an attack of softening. In the former there is, as a rule, unilateral anæsthesia, which rapidly diminishes or disappears; in the latter there is dulness of sensation, with morbid feelings of coldness, numbness, tingling, &c., which persist.

The paralysis in hemorrhage is, typically, hemiplegic; in softening it is more irregular, and sometimes more closely limited. In the former there is neither rigidity nor convulsion unless the coma be profound, and the paralysis extensive; in the latter there is often either twitching or tonic spasm even when the paralysis is slight, and the mental perturbation comparatively trivial.

In hemorrhage there is very frequently hypertrophy of heart, with granular degeneration of the kidneys; in softening there is very commonly a weakened heart, with valvular disease.[1] Hemorrhage may occur in a person of strong limb and general good health, whereas softening is more common in the aged, the enfeebled, and those who have suffered from exhausting diseases, or still more exhausting cares.

From *urinæmia* as a cause, *per se*, of an apoplectic seizure, softening may be distinguished by the fact that in the former, convulsions of some kind and to some degree almost invariably precede the coma; and that these have followed premonitory symptoms of drowsiness, oppression, and headache. The coma exhibits in a marked manner the features sometimes observed in acute softening of the brain, viz., an apparent profundity, with susceptibility of being roused. Urinæmic patients lie in apparently profound torpor, but they may be roused by a touch or a word, and may appear in possession of all their faculties; left to themselves they relapse almost instantly into the state of stupor. In softening, if the coma be highly marked the awakening is less distinct, and the answers are less rational. In urinæmia there is often amaurosis, and a generally obtuse condition of sensibility, neither of which is frequent in ramollissement. There is much twitching of the limbs, and often marked rigidity in the former case, but the locality of these symptoms changes from side to side, and is not accompanied by fixed paralysis; there may be twitching or rigidity in the case of softening, but these are found in the same limbs day after day, and are attended by distinct and persistent loss of power. The stertor in urinæmia is unlike that of softening, being oral rather than guttural;[1] the pulse-respiration ratio is much changed, being sometimes 5 : 1;[2] sensori-motility and irritability of muscles on percussion are often notably increased. Beyond these features there are the signs of characteristic debility and cachexia, the pale waxy skin, with vomiting and diarrhœa; and above all the œdematous condition of the eyelids and ankles, together with albuminous urine. In cases of acute cerebral softening, all these symptoms may be absent. The two diseases are frequently combined, but when they are so it is not impossible to determine how much is due to the one, and how much to the other condition.

When Softening of the Brain occurs in a *convulsive* form, its diagnosis is to be established by regard to the symptoms already described, p. 863. It is by a consideration of the prodromata and of the after-phenomena that the distinction may be made from epilepsy; it is by a similar process that tumor of the brain or of meninges may be diagnosticated.[3] That which is characteristic of softening is not the fact of the convulsion, nor the form which the convulsion takes, but the gradual development of intellectual, sensorial, and motor failure, such as has been described in the section upon symptoms; and the absence of those general and special changes which are characteristic of tubercular, carcinomatous, or other morbid growths.

The form of softening which is marked by *delirium* is not likely to be confounded with any other malady. It is essentially an affection of old age, and may be distinguished from simple senile congestion by the persistence of its symptoms, and by the speedy development of those signs of failure in nerve-power, to which so much attention has already been directed.

2. *Chronic softening*, when its course has been chronic throughout, has to be distinguished from tumor and from meningitis; and although the distinction is not possible in all cases, approximation to certainty may be attained in the majority by regard to the following considerations.

In *tumor* there is pain, intense in degree, subject to violent exacerbations, limited to and fixed in one locality; the special senses are affected, so that there is blindness or deafness, or the two combined, on one side generally, but sometimes on both; there are local paralyses and epileptoid convulsions; but, apart from the convulsions, unimpaired intelli-

[1] Dr. Kirkes found the heart hypertrophous in thirteen of twenty-two cases of cerebral hemorrhage, and in all of these the kidneys were diseased, being for the most part granular and atrophous. (Medical Times and Gazette, Nov. 24, 1855.)

[1] Addison, Guy's Hospital Reports, 1839, vi.

[2] Marcé, Schmidt's Jahrb., Nov. 1855.

[3] See articles Epilepsy and Adventitious Products in the Brain.

gence. There is often marked disturbance of the stomach, obstinate vomiting, and constipation; and there may be the signs of the tubercular, carcinomatous, aneurismal, or syphilitic dyscrasiæ. In *chronic meningitis*, there is pain, generally distributed over the whole head, not very severe in degree, and, although varying in intensity, not subject to the paroxysmal exacerbations observed in tumors; there may be local paralyses, and these are especially observed in the muscles of the eyeball; there is much but intermittent mental excitement and irritability of temper alternating with marked depression; there are disorderly spasms and paralyses of the limbs together with frequent but irregular accessions of fever; there is often a syphilitic taint, but there may be an entire absence of that dyscrasia, and the symptoms may have dated from a blow or fall.

In *chronic softening* there is dull headache, and gradual impairment of intelligence, motility, and sensibility, together with advancing years or a prematurely aged appearance, a feeble heart, rigid vessels, and most commonly some disease of old standing in such important viscera as the kidneys, heart, or liver.

Thus, to resume, the characteristic feature of tumor is pain, that of softening is failure of power, that of chronic meningitis is the mixture of excited with depressed functional activity. It is the progressive deterioration of cerebral faculty which marks out the disease we are considering; the patient begins to die, as it were, before his time, and his death begins in the highest element of his organism.

Prognosis.—Occasionally there is complete recovery after an attack of acute softening; for example, apoplectic seizures have occurred, which have been followed by hemiplegia and mental dulness of many months' duration; these symptoms have passed away entirely, and upon post-mortem examination, after a number of years have elapsed, the signs of old softening have been distinctly discernible. It is possible, of course, that the softening may have originated in or around a "clot," but that possibility does not affect the general question of prognosis. Again, there may be improvement, but not complete recovery; a patient may suffer a sudden apoplectic seizure, and may lie for days in a state of profound danger, the mind almost a blank, and the limbs hemiplegic; but after a time he may improve, and his improvement may continue for months, slowly going on from week to week; he becomes able to understand what is said, to speak or to make intelligible gestures, he may walk, or may even regain the use of his hands, and may remain more or less aphasic for an indefinite period. He may have a second attack, and one so characterized as to show that the other side of the brain has been affected, and he may be partially restored from this. At length a third or a fourth seizure comes, from which there is no recovery. Upon post-mortem examination in such cases distinct softenings may be found in such situations as to relate them to the first or second attack, and the cause of such softenings may be discovered in the obstructed arteries. The prognosis of softening, therefore, although unfavorable, is not necessarily fatal.

The prognosis is relatively favorable when the patient is young, and has been previously healthy; as, for example, when an apoplectic attack occurs during the course of rheumatic endocarditis in a young subject. It is unfavorable when the patient is old, or is affected by chronic disease of the kidneys, liver, or heart, when the arteries are rigid and the circulation low. It is favorable when the softening is, as it were, the result of accidental interference with the supply of blood; it is unfavorable when that interruption of the arterial circulation is but part of a general organic change.

The severity of an attack is to be judged of by regard to its mode of onset. If accompanied by either much congestion or by profound collapse, the symptoms, although very highly marked and widely distributed, may pass away; whereas the same amount of symptoms occurring without evidence of congestion or collapse would indicate, in direct proportion to their severity and extent, the gravity of the lesion. *Cæteris paribus*, the lesion is in proportion to the *extent* of the symptoms; and the prognosis is worse when the mind, sensation, and motion are all slightly impaired, than it is when either one of them alone is profoundly affected.

If the patient be young, and if there be no signs of general impairment of nutrition, hopes may be entertained that there will be some recovery of mental and motor power: but if the patient be old, or if there be weak circulation, and rigid vessels; and if the attack has had many forewarning symptoms, such as occasional forgetfulness, numbness of the extremities, and the like, the probability of restoration is very small, while the likelihood of increasing mischief or of renewed seizure is very great.

The prognosis of approaching death after an apoplectic or convulsive seizure is based upon the increasing rapidity and feebleness of pulse, the involuntary passage of the urine and feces, and the general flaccidity of the limbs.

Treatment. — Attacks of softening may be postponed by attention to the

following points: 1. The maintenance of an even temperature in the body: the feet and hands when chilly and blue should be put in hot water, or wrapped in and rubbed with warm flannels, and the patient should be placed in the recumbent posture, with the head only slightly raised. 2. The avoidance of long intervals between meals; food, easy of digestion should be given frequently, and the patient, if old, should not be allowed to pass the night without nourishment. 3. The ready administration of some gentle stimulant when there is any tendency to occasional pallor or faintness. A glass of wine, or some sal-volatile and water, should always be at hand, and should be given, not recklessly, but fearlessly if the premonitory symptoms become threatening. 4. Direction of the mental habits; easy and pleasant occupation of the mind, with careful abstinence from lazy inaction on the one hand, or violent excitement on the other. 5. Careful attention to the excretions, the skin, the kidneys, and the bowels. Exposure to cold is very prejudicial, and, although constipation and straining at stool are to be strenuously avoided, nothing is much more mischievous than the relaxation of close and too warm rooms, and the production, by medicines, of anything approximating purgation of the bowels.

When the premonitory symptoms are those of much headache and drowsiness, obvious relief may be gained by warmth to the extremities and by the use of such diuretics as the liquor ammoniæ acetatis, with infusum scoparii, nitrate of potass, and spirits of nitric ether, or of juniper.

When there is the tendency to nocturnal delirium, a judicious administration of liquid nourishment, with very small quantities of wine, may suffice to give relief. Should this fail, the most useful medicine that I know of is the Indian hemp, in doses of a quarter to half a grain of the extract; and next in value is the chloral hydrate in doses of five or ten grains.

If there are marked symptoms of spasmodic or convulsive character, bromide of potassium in doses of from five to fifteen grains may be given three times daily with a bitter infusion and some diffusible stimulant, such as chloric or nitric ether.

On the occurrence of an attack, either apoplectic or convulsive, there is but little that can be done beyond the regulation of temperature and of secretion that has been already described; but when the attack has passed away something may be gained by the administration of cod-liver oil, hypophosphite of soda, and vegetable tonics. In many cases of chronic softening marked improvement follows the exhibition of cod-liver oil, and I am disposed to regard this as the most valuable agent in the treatment of the malady. It should be administered in conjunction with a fully nutritious but easily digestible diet, and with free exposure, without fatigue, to fresh air. It appears to me highly doubtful whether under any circumstances of softening of the brain the smallest good has followed either general or local blood-letting, the application of blisters, the administration of mercury or of iodide of potassium.

When recovery has advanced to a considerable degree, and some limbs remain paralyzed, good has distinctly followed the exhibition of iron, and of strychnia in exceedingly small doses, and the cautious application of galvanism to the weakened muscles.

ADVENTITIOUS PRODUCTS IN THE BRAIN.

By J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D.,[1] F.R.S.

In this chapter is included a description of many diseases differing widely from one another in their pathological character, but agreeing in this, that they lead to the development within the cranium of some abnormal physical conditions, the nervous symptoms of which may bear close resemblance, *inter se*, during life.

Symptoms. — It has sometimes happened that, on post-mortem examination, tumors have been found in the brain, the existence of which had never been suspected during life. The patient may have

[1] The section on Morbid Anatomy is written by Dr. Bastian.

presented no sign of cerebral disease, and may have died from an affection of the lungs or other organs; and, with the help conferred by post-mortem discovery upon the direction of questions as to past history, no evidence can be obtained of any symptom which can be referred to the brain. Such a case occurred to myself some years ago; several large hydatid growths being found in the brain of a young girl who had never suffered in any such way as to lead to the suspicion of cerebral mischief. These facts should be borne in mind when dealing with certain cases where signs of cerebral disease are present, but the diagnosis is obscure. When it is said that "a tumor cannot exist because of the absence of this or that symptom," it should be replied, that a tumor may exist without any symptoms at all.

There may be very highly marked symptoms, and yet these may be of such a character as to mislead. Dr. Abercrombie[1] has related examples of disease within the cranium, the locality and nature of which had been unsuspected during life, the patient's history having been such as to lead to a diagnosis of some affection of the stomach. Cases of the kind described by Dr. Abercrombie are rare, and it is probable that they will be rendered more so, as clinical examination becomes more minute. The fact, however, remains, that the complaint of a patient may be so marked with regard to dyspepsia, vomiting, constipation, and the like, and so trivial in respect of headache, giddiness, or other morbid sensations, that, although he has a tumor in his brain, no suspicion may be entertained of its existence, and the diagnosis that is arrived at may be malignant disease of the pylorus, or the liver.

In other cases the symptoms of intra-cranial tumors are highly characteristic, and the diagnosis of their existence, their exact situation, and anatomical nature may be sometimes made with a precision and minuteness such as is scarcely attainable with regard to any other diseases.

The *intellectual* faculties may be quite unaffected; indeed, they very often are retained in their integrity when other functions of the brain are seriously impaired. The changes they present are of two kinds: there may be on the one hand great irritability of temper, a condition totally different from the previous habit of the individual, and with this, but very rarely, some mild delirium, or confusion of thought; on the other hand, there may be loss of memory, and general impairment of intelligence, with depression of spirits and listlessness. When convulsions occur, as they frequently do, during the few days that precede death, there may be profound coma as their sequel; but the convulsions which exist in the earlier period of the growth of tumors are often accompanied by only partial loss of consciousness, and are followed by none of the stupor which is so commonly observed in epilepsy.

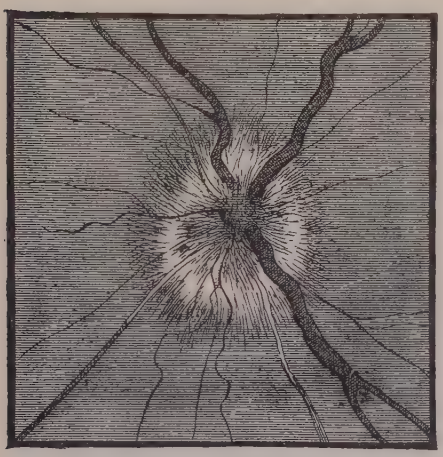
Sensation is altered in various manners, but by far the most characteristic change is that of pain. Headache is often slight at the commencement, but afterwards it arrives at great severity; it is usually confined to a definite point or region of the head, and persists in that locality; it undergoes occasional exacerbation, and sometimes the suffering seems almost intolerable, and elicits from the patient agonizing cries; it is, however, rarely absent altogether during the intervals of paroxysm; it is increased by intellectual and physical exertion, by emotional disturbance, by sensational impressions, and by forced respiratory movements. It is sometimes almost the only symptom, but in rare cases it is absent altogether. The aggravation of pain which is occasioned by light, or noise, or movement, is such as to make a patient hold his head steadily between his hands, or bury it in the bed-clothes; and this intolerance has sometimes been regarded as hyperæsthesia, from which it differs widely. The sight and the hearing may be dull, but yet sensorial impressions intensify the headache. The sense of sight is often lost in one eye or in both; or there may be simply some mistiness or imperfection of vision, with dark or bright spots before the eyes. The iris does not often lose its irritability, but it is often found diminished; whereas, in some cases of complete blindness, it contracts readily on the admission of even a feeble light. Various changes may be observed in the ophthalmoscopic appearances, but that which is, perhaps, the most characteristic of tumor —although by no means constantly present—is enlargement of the veins, an enlargement sometimes to be observed in one eye only, sometimes in both. [The importance of this appearance, of *choked disk*, has been differently estimated of late years by leading authors; but statistics show[1] that in about ninety-five per cent. of cases of intra-cranial tumor, either this appearance or that of *descending optic neuritis* is found when sought for with the ophthalmoscope. Türck first called attention to retinal signs of brain disease, in 1853. Von Graefe, in 1860, pointed out the existence, in different cases, of two conditions; the one, of great engorgement

[1] Practical Researches, p. 337.

[1] Annuske, Reich, Longstreth, &c. See a paper by Dr. W. F. Norris, and a discussion in Pathological Society of Philadelphia, in Philada. Med. Times, Aug. 30, 1879.]

of the intra-ocular end of the optic nerve (stasis papillæ or choked disk), and the other, of interstitial inflammation of the optic nerve, descending from meningeal inflammation (descending optic neuritis). Both of these conditions are followed by atrophy; and the difficulty of discrimination between them, and of separating them from simple atrophy of the optic nerve, becomes greatest in their later stages. Since 1860, Bouchut, Allbutt, Hughlings Jackson, and others have given much attention to this aid in diagnosis. Annuske found that, in 920 cases examined, nearly all brain-tumors were attended either by optic neuritis or choked disk. The location of

Fig. 51.



Choked Disk. (After Liebreich.)

the tumor does not seem to be at all constant in its relation to these results. Neither do large tumors more constantly produce them than small ones. Some marked cases of brain-tumor, moreover, have been proved to be entirely without ophthalmoscopic indication of their presence. Hence Becker (1868) and Schweigger (1871)[1] have denied the practical importance of the coincidence when it occurs. We must conclude, however, that at least an important *negative* value belongs to it; viz., that, when tumor of the brain is suspected, but not proven, the total absence of ophthalmoscopic changes makes such a diagnosis much less probable than if they are ascertained to exist. The ophthalmoscope should be used, therefore, in every doubtful case, even if there be no observed defect in the patient's vision; since it has been found[2] that sight may be apparently good, and yet examination will show decided alterations to have commenced in the optic disk.—H.] Hearing is less commonly impaired, but it may be lost completely on one side without having attracted the attention of the patient; noises in the ears are common, either with or without any diminution of the faculty. Numbness, tingling, creeping feelings, sensations of heat or cold, may exist in the limbs, or in certain tracts of skin over the trunk, and sometimes there may be distinct anæsthesia of parts. Vertigo is frequent, and often most distressing; usually it is relieved by closing the eyes and maintaining perfect rest; but sometimes it is aggravated by darkness, and the patient has to maintain a fixed gaze in order to ward off the feeling. Commonly the vertigo is of such a kind that the patient feels as if rolling over, or swimming along in space; and it is comparatively rare to hear complaint of the apparent rotation or motion of surrounding objects. Affections of the sight have been found most frequently when the tumor has occupied the anterior lobes of the brain, and least frequently when in the posterior lobes or cerebellum.

In the cases which have been placed on record, *convulsions* have occurred more frequently than *paralysis*, and among those which have presented the latter, one-half have exhibited the former. Convulsions, of epileptic form, often occur during the few days that precede death; but in certain kinds of tumor or of adventitious product in the brain, epileptoid convulsions may exist for years, and the cases presenting them may be termed "epileptic." When no general paroxysms occur, there are, very frequently, clonic spasms or tonic contractions of the muscles. As the result of an examination of a large number of cases, it may be stated that convulsions are most common when the disease is situated in the posterior lobes of the brain, or in the cerebellum, and least frequent when the anterior lobes are affected; the distribution being exactly the reverse of that which pertains in regard of amaurosis.[1]

Paralysis is sometimes observed in one muscle of one eyeball, such, for example, as the external rectus, leading to convergent strabismus; or in all the muscles supplied by the third nerve on one side, so that ptosis and divergent strabismus, with dilated pupil, are the results. The speech may be also affected, the patient being unable to articulate certain sounds; the facial muscles may be so paralyzed as to produce every degree of deformity, or the paralysis may be more widely distributed, and be hemiplegic, or, but more rarely, paraplegic in its form. Sometimes the lesion may be of such kind, and in such degree and locality, as to affect the

[1 Norris, loc. citat.]
[2 Shakespeare, Phila. Med. Times, loc. citat.]

1 Vide Auct., Diagnosis of Diseases of the Brain, &c., p. 186.

nutrition of muscular and other textures. For example, in a case under my own care, where a tubercular mass involved the seventh nerve, and also the deep origin of the fifth, not only was there loss of sensibility on one side of the face, but the temporal muscle was much wasted, the conjunctiva and cornea became sloughy, and the mucous membrane of the mouth was aphthous and studded with vegetable parasites. The paralysis of cerebral tumor is developed—as a rule—slowly and insidiously; and when it occurs in one of the limbs, is sometimes preceded by pain or some other alteration of sensibility; but in a few cases it is produced as an "apoplectic" phenomenon, and may be the first symptom to attract the notice of either patient or physician.

The *general* symptoms of tumor of the brain vary almost indefinitely, for they may be simply those of reflex or direct disturbance of the stomach and other viscera, when hydatid or fibrous growths are their producing cause; or they may be the special features of carcinoma, tubercle, or syphilis, when any one of those dyscrasiæ is the primary fact in the formation of the adventitious product. Thus they may, on the one hand, be all-important, and may partially conceal the cerebral disease; on the other they may be so trivial as to awaken no attention; while in an intermediate group they may have such character and relation as to render it possible to make an accurate diagnosis of the nature of the lesion. Thus there may be tumors on the scalp, or, in other regions, glandular swellings of strumous character; or there may be distinct evidence of syphilitic deposit, of carcinoma, or of aneurismal dilatation of the vessels. Tuberculosis gives rise to the most common form of tumor in the child or young adult, and syphilis is the next in frequency in early life or middle age; while carcinoma is prevalent in direct proportion to advancing years. Sometimes there is obvious alteration in the general contour of the head, but such change is almost confined to the period of growth; whereas in later years there may be gradual prominence of one eyeball, or the distinct pressure of a growth through the bones. Under such circumstances the diagnosis is tolerably easy.

As a rule the commencement of symptoms is insidious, and their progress slow; but sometimes after a few premonitory phenomena there is a sudden attack of convulsions, or an apoplectic seizure. Under such circumstances a diagnosis is possible only by consideration of the subsequent history.

Diagnosis.—In some cases it is impossible to gain even a hint of the nature of the malady, while in others the diagnosis is as certain as that of any disease with which we are acquainted. It will be convenient to speak first of the diagnosis of tumor generally, secondly of the diagnosis of the locality of growths, and thirdly of the recognition of particular forms of morbid product.

1. If convulsions be the prominent feature of the case the diagnosis is to be made from *epilepsy*, and here the distinction depends upon the recognition of symptoms over and above those of the latter disease. The convulsions are commonly epileptiform in type, but very often they present these differences—they are irregular in development, there is not absolute loss of consciousness, there is little or no asphyxia, and no subsequent stupor, while the spasmodic movements are more marked on one side than on the other, they last for a longer time than is observed in epilepsy, and frequently terminate without being followed by anything like epileptic coma. In many cases the age at which they commence is so far advanced as to make epilepsy improbable, and there are symptoms—such as pain, affections of the senses, and paralysis—which do not occur in simple epilepsy. Again, the mental state in a person the subject of tumor may remain quite intact, and may fail to present the peculiar sluggishness which is often, although by no means invariably, the concomitant of repeated epileptic seizures. Epilepsy is essentially a chronic disease, the commencement of which dates in a large proportion of instances from or soon after the period of puberty; it is characterized by fits of a peculiar type; and when uncomplicated it exists for years, and in the majority of cases without entailing any ulterior change in the functions of either brain or spinal cord. Tumor in the brain is of comparatively rapid development; it begins, as a rule,—to which there is the one exception of tubercular growth,—after adult age has been reached, and most commonly when the period of middle life is passed; and when convulsions are present they are by no means the prominent symptoms of the malady, for, although often severe, they may be cast into the shade by the violence of pain and the loss of special senses.

It is possible that the early and even some of the advanced symptoms of tumor in the brain should be confounded with, or passed over as, *hysteria*. Such mistake can only arise through carelessness, or through a prejudiced mode of dealing with the obscure affections of women. Not long since a lady consulted me, who was supposed to be hysterical, and who had been treated upon that supposition. Yet her symptoms had not commenced until after thirty years of age; she had violent paroxysmal headache, was blind of one

eye, and deaf of one ear, and the amaurosis and the deafness had crept on slowly. The distinction from hysteria may be made by regard to age, affections of the special senses, the absence of the peculiar mental condition of the hysteric patient, and the nature of the paroxysms.

Chronic meningitis may be of such character that its physical conditions become identical with those of morbid growths, and its symptoms pass then into those of tumor. This is the case, for example, when there is syphilitic thickening of the membranes, which may at any time become so complicated by nodular thickening of either of the membranes or the bones, as to give rise to the special conditions and symptoms of a tumor. Again, a tumor, of fibrous or carcinomatous character, the growth of which is habitually slow, may sometimes set up, in its neighborhood, chronic meningitis, so that the symptoms of the two morbid processes may be found in association. Under either set of circumstances the diagnosis is possible by a regard to the mode of development of, and relative proportion between, the symptoms. As already stated (see p. 881), the distinction between tumor and chronic meningitis lies here—that in the former the characteristic features are violent pain, marked diminution, or loss, of one or more of the special senses, limited paralysis, integrity of mind, and occasional epileptoid convulsions; whereas in the latter the pain is slight, the special senses are perverted but not lost, the mind is damaged, and the convulsions are less distinctly epileptoid. In the former there is the predominance of pain, in the latter there is no such predominance, but a mixed condition of excited and diminished action in mind, sensation and motility. In the case of chronic syphilitic meningitis the diagnosis may be assisted by the history of syphilis, or by the presence of its symptoms in a tertiary form. It must be remembered, however, that the diagnosis may often be carried to this point,—the obvious presence of chronic meningitis, and the probability of tumor; but this latter cannot be affirmed to exist because of the absence of its special symptoms (see page 884). On the other hand, in certain cases, there may be no doubt of the existence of a growth, when paroxysmal pain, &c., occur in a patient exhibiting the features of the carcinomatous cachexia.

From *chronic softening of the brain*, the diagnosis is to be made by recognizing the absence of the characteristic features of that malady, viz. loss of mental power, of sensation, of motility. Certain cases of tumor may be taken for examples of chronic softening, and they are such as have been marked by a small amount of pain, by repeated convulsions, and gradual failure of intelligence. It would be impossible under such circumstances to make an accurate diagnosis.

2. The diagnosis of *the particular locality of a tumor* may sometimes be very minute and accurate, but in other cases vague, and not unfrequently erroneous. The side of the brain affected is usually, but not invariably, the same as that upon which the pain is felt, and on which the special senses and the muscles of the eyeball are affected; it is the opposite to that upon which spasm or paralysis occurs in the limbs. Again, the locality of pain may be taken for a guide as to the situation of a tumor in the anterior, middle, or posterior lobes. Such guide, however, is not always trustworthy, for frontal pain may be the result of a cerebellar tumor. Upon analyzing a large number of cases, I find that convulsions are most frequent in tumors of the cerebellum, and that they diminish in frequency as the seat of lesion advances forwards, *i. e.* through the posterior and middle to the anterior lobes of the cerebrum; and that amaurosis, impaired articulation, and intelligence observe a contrary relation to those lobes, being most common when the tumor is in the anterior cerebral lobes, and relatively less frequent as the seat of tumor retrogrades. Romberg has suggested that the position of a tumor on the upper surface or at the base of the brain might be determined by an observation of the effect produced on pain by forced inspiration or expiration: the pain of tumors, when seated at the base, being aggravated by inspiration, that of those on the upper surface by expiration, sneezing, or coughing. This Romberg explains by the rising and falling of the brain during the respiratory movements, and the consequent pressure of the mass against the upper or lower bony walls.[1] I have met with several cases which confirm Romberg's statement, but several others in which no reliance could be placed upon the test. The particular portion of the brain involved in a tumor may be sometimes determined by a careful consideration of the distribution of all the symptoms; such diagnosis, however, requires merely the application of anatomical and physiological knowledge, and needs no further notice here.

3. A diagnosis of *the nature of a tumor* is always of great importance both in regard of prognosis and of treatment. Sometimes all that can be accomplished is a guess, but sometimes tolerable certainty may be attained. Under certain circumstances we may distinguish between tubercle, syphilis, lead-poisoning, aneurism, hydatids, carcinoma, and hypertrophy.

Tubercle is the most common form of tumor in the child or young adult, and

[1] Manual of the Nervous Diseases. Syd. Soc. Trans. vol. i. p. 159.

we might infer its presence if, in addition to a tubercular family history, there were the obvious features of the tubercular constitution, marked wasting of the body, together with an elevated temperature, and the presence of tubercular disease in the lungs or bronchial glands. It is somewhat curious to observe that the temperature of cases of cerebral tuberculosis is not raised to the degree, nor with the persistency, that is to be noticed in regard of tuberculosis elsewhere, but of all cerebral growths that which exhibits the greatest amount of elevation of temperature is tubercle. After puberty an examination of the chest is of great importance in the diagnosis, since it rarely happens that a healthy state of the lungs is found coexistent with tubercular disease of the cranium.

Syphilis may occur in such locality as to produce any of the varied symptoms which have been enumerated; but there are certain features which help to distinguish it from other forms of adventitious products in the nervous centres. Headache is rarely intense, but is prone to undergo nightly increase; actual loss of sight or hearing is not common; but implication of the third or sixth nerve is very frequently observed, so that patients exhibit ptosis, dilated pupil, and divergent strabismus, or more commonly inversion of the eyeball; there is often much depression of spirits, and further a wide extent of symptoms, the spinal cord as well as the encephalon being involved in the mischief. The characteristic features of this disease are, however, to be sought elsewhere, in the presence of periosteal thickening, eruptions on the skin, and such other phenomena as have been described in the article on Constitutional Syphilis.[1]

The *intoxication of lead* may be followed by such induration of brain substance as shall produce the symptoms of tumor; but the diagnosis turns upon a consideration of the previous history of the case, and the discovery of exposure to lead by trade, accident, or medicine; the previous occurrence of symptoms of lead colic; the presence of general cachæmia, of a blue line on the gums, and of lead in the secretions. The extensors of the hands and fingers are the most frequently paralyzed; the extensors of the toes, the tibialis anticus, and peronæi are not rarely affected; the paralyzed muscles become much impaired in their nutrition, and exhibit marked diminution, even extinction, of both contractility and sensibility on the application of powerful faradization; but yet they may respond readily to a slowly interrupted battery-current of moderate power. It has been further observed that this loss of contractility to faradization sometimes persists after there has been restoration of voluntary power.

Induration of the brain may occur in scorbutus, in rickets, or in epilepsy, but the diagnosis of its presence in either condition would be attended with much difficulty during life. Dr. Cohn observes that in one case there was, in the instance of rickets, an entire want of intellectual disturbance;[1] whereas in epilepsy—as described by Bouchet and Cazauvielh, and as observed by myself—the presence of induration might be inferred from the progress of general intellectual decay; attention, apprehension, memory, and judgment failing; the patient becoming gradually incoherent, and general paralysis creeping on, while the fits increased in frequency but diminished in violence.

The *aneurismal* nature of a tumor could be guessed at only *per viam exclusionis*, or rendered probable by the observation of disease of similar kind in other portions of the arterial system; for it has been only in very rare cases that an aneurismal bruit has been discovered in the head during life; similar remarks may be made with regard to hydatid growths, and it must be remembered that hydatids in the brain may exist without producing any symptoms whatever.

The presence of *carcinoma* would be inferred only upon the recognition of the cancerous cachexia; and here age would be an important element in the consideration. Lancinating pains in the limbs are not, as Rostan supposed,[2] of any diagnostic value; but the diagnosis must rest upon the discovery of the carcinomatous dyscrasia, and the coexistence of tumors elsewhere, and especially of such as affect the integument and bones of the skull.

Hypertrophy of the brain, although not an adventitious product, is best described in this place. It may be met with in young children; but the only characteristic feature of the disease is enlargement of the cranium. Dr. West observes that this is "first apparent at the occiput, and the bulging of the hind-head continues throughout especially striking. The forehead may, in the course of time, become prominent and overhanging, but the eye remains deep sunk in the socket, for no changes take place in the orbitar plates, such as are produced by the pressure of fluid within the brain."[3] There is no prominence, but actual depression, of the fontanelles and sutures; the general nutrition of the child is imperfect, but there is

[1] Part i. p. 423.

[1] See Cohn, in Gunsb. Zeitschr. v. 35, 1854; Schmidt's Jahrb. Bd. 86, 1855, No. 6, p. 322; and Bouchet and Cazauvielh, De l'Epilepsie dans ses Relations, &c.

[2] Recherches sur le Ramollissement, p. 404.

[3] Lectures on Diseases of Infancy and Childhood, p. 9.

nothing special in the cerebral symptoms which would lead to the diagnosis of this particular form of malady. Partial hypertrophy of the brain may be attended by no disturbance of the cerebral functions; the morbid condition, under such circumstances, can be recognized only by post-mortem examination.[1]

Morbid Anatomy.—In this place we do not profess to give an account of intracranial adventitious products as a whole, but shall strictly confine ourselves to such products or growths as have their seat in some part of the encephalon, and we must refer to another article for a description of the morbid growths which take origin from the meninges. Whilst it is indispensable, from a pathological point of view, to refer to these growths under different heads, it must not be supposed that we are able clinically to exercise the same precision. And neither, during the life of the individual, is it possible to determine whether a new formation, imagined to exist, has originated in the midst of the brain substance, or, having sprung up from one or other of the meninges, has merely grown into or pressed upon this secondarily. In both cases the symptoms produced may be almost identical. Similar effects are also, in rare instances, produced by the extension of an external morbid growth inwards through the orbit.[2]

In addition to the adventitious products more strictly so called, such as blood-clots, hydatids, &c., many varieties of tumors are met with in the encephalon. As in other parts of the body, these often present unmistakable characters, though occasionally their histological composition is so indefinite as to make it extremely difficult to classify them. They exist either as distinct growths, with sharply defined outlines, or they may be, as it were, infiltrations passing insensibly, at their circumference, into the surrounding brain tissue. In the former case they are often inclosed in a fibrous envelope, of more or less thickness, which now and then may be found in a calcified condition; whilst the brain tissue around may be quite firm and healthy, or it may be softened to a variable extent. The softening may be simply white, or, if there have been much antecedent congestion, it may exhibit various shades of red; whilst in other cases the yellow gelatinous softening is met with, such as Rokitansky[3] and Cruveilhier[4] have described. This seems to occur most frequently around cancerous growths. At other times the brain tissue surrounding adventitious products is condensed and indurated.

Such adventitious bodies as silver and lead are met with only in minute quantities, and are for the most part diffused through the substance of the brain. Not being collected into distinct aggregations, we require the aid of the chemist to recognize their existence. Saline matters, also, either abnormal in kind or in quantity, may be diffused through the tissue of the nervous centres, when the nutrition of these fails and the brain matter is undergoing certain modes of degeneration. This is a subject upon which, at present, our knowledge is very defective.

The various new formations and foreign bodies which are, from time to time, met with in the substance of the brain, may be thus arranged, though they will not all be treated of in the present article:—

1. Tubercle.
2. Syphilitic growths.
3. Cancer.
4. Melanotic tumors.
5. Gliomata.
6. Fibro-plastic tumors.
7. Fibrous do.
8. Osseous do.
9. Tumors of the pituitary and pineal bodies.
10. Nodules of gray matter on ependyma of ventricles.
11. Vascular erectile tumors.
12. Aneurisms.
13. Blood-clots.
14. Abscesses.
15. Plastic lymph on the walls of ventricles.
16. Cysts.
17. Calcareous and other concretions.
18. Entozoa { *a.* Cysticerci. *b.* Hydatids.
19. Silver and lead.

It will be convenient to place also in the last section the remarks that we have to make upon hypertrophy of the brain.

1. *Tubercle.*—Tubercle in the brain is much more frequently encountered in children than in adults, but, as pointed out by Andral, it is not commonly met with in children under two years of age. The period of maximum frequency is thought to be from the third to the seventh year inclusive. It rarely occurs in the brain in children without at the same time existing in some other organ of the body. In 117 examinations of adults who had died of phthisis, Louis met with tubercle in the brain only in one case, whilst MM. Rilliet and Barthez discovered masses of tubercle in the brains of 37 out of 312 children in whom this morbid deposit existed in one or other of the remaining organs of the body.

[1] See cases reported by Giacomo Sangalli, Gaz. Lom. 1858, quoted in Schmidt's Jahrb. Bd. 102. 1859, p. 22.

[2] Ch. Robin, Gaz. Méd. 1855, 6 et 13 Oct.

[3] Path. Anat. (Syd. Soc.), vol. iii. p. 419.

[4] Anat. Path., 8me Livr. p. 5.

Sometimes a single mass of tubercle exists in the brain, sometimes two or three, and in others a large number of smaller masses. In size the separate masses vary between that of a millet seed and a large hen's egg — those most commonly met with, however, being about equal to a filbert or a small walnut. They are mostly spherical in form, but are occasionally more or less lobulated. All parts of the brain, from the surface to the centre, are occasionally the seat of this deposit. Perhaps, having regard to its size, the cerebellum is the most favorite seat of tubercle. It is so regarded by Dr. Wilks and by Sir William Jenner; and Andral was, doubtless, of the same opinion, since, without regard to size, he placed the cerebellum second, after the cerebral hemisphere, in the order of frequency of site. After these, in order of frequency, Andral names the pons Variolii, the medulla oblongata, the spinal cord, the peduncles of the cerebrum and cerebellum, the optic thalamus, and the corpus striatum. Very frequently, when masses of tubercle are situated in or upon the cerebellum, they impede by their pressure the return of blood through the venæ magnæ Galeni, or the straight sinus, and so cause effusion of serum into the ventricles, and all the symptoms of chronic hydrocephalus.[1] Very rarely, almost the whole of the cerebellum, or one of its lobes, may be, as it were, replaced by tubercular matter.[2]

The usual condition in which tubercle is met with in the brain is, according to Rokitansky, in the form of masses in the size and shape already mentioned, "of a yellow or yellowish-green color, of the consistence of lard or cheese, and firm, but easily lacerable." It is remarkable that the gray, translucent form of tubercle is rarely, if ever, met with in the brain. Lebert says he has seen it rarely, and Rokitansky believes "that there are some rare cases which prove that tubercle in the brain does, in part at least, commence in the gray translucent form, for portions of a tubercular mass are sometimes found in that state." He believes that it rapidly passes over from this form into that of the yellow cheesy tubercle. So far he is very much in accord with Virchow,[3] who holds that each mass of cerebral tubercle is in reality made up by the aggregation of a multitude of small miliary tubercles. Each mass is formed, not by the growth of one original focus, but "by the continual formation and adjunction of new foci at its circumference." He adds :—"If we examine one of these perfectly yellow, or white, dry, cheesy tubera, we find immediately surrounding it a soft, vascular layer which marks it off from the adjoining cerebral substance—a closely investing areola of connective tissues and vessels." In this layer the young granules[1] are formed. They are continually produced at the circumference, "and the large tuber grows by the continual apposition of new granules (tubercles), of which every one singly becomes cheesy."[2] But though this is the condition in which growing masses of tubercle are met with in the brain, at a later stage the process of increase stops, and, owing to an irritative process, a fibrous envelope gradually forms round the mass, so as completely to isolate it from the surrounding brain tissue. This condition is so general that many pathologists have held that all tubercles occur in an encysted condition in the brain.[3] The thickness of the fibrous envelope varies with age—it may be an almost imperceptible layer of fibrous tissue, or it may attain a remarkable thickness and almost cartilaginous consistence. Occasionally, even, it becomes completely calcified.[4] Softening is met with, at times, in the centre of the tubercular masses; and, very rarely, in encysted tubercle the whole contents may undergo this change. Such a change is reported by Dr. Ogle,[5] in which a cyst the size of a pigeon's egg, with thin and friable parietes, was found in the pons Varolii, containing a "yellow glairy fluid in which a number of light-colored soft particles of albuminous matter existed." More rarely still, in the brain, the tubercular mass has been found to have undergone a process of cretification.

The brain tissue around masses of tubercle is often perfectly natural, at other times it may be congested, more or less softened, or even indurated.

2. *Syphilitic Growths.*—These are very rarely met with in the substance of the brain. Instances have, however, been

[1] Wilks' Path. Anat. 1859, p. 158. This fact is strongly insisted upon, also, by Sir W. Jenner in his clinical teaching.

[2] *Vide* Hooper's Morb. Anat. of the Human Brain, p. 60, pl. xi.

[3] Cell. Pathol., transl. by Chance, p. 477.

[1] Gray granulations.

[2] Although differing so widely in their views as to the nature of tubercle, Rokitansky's description of the circumference of these masses in the brain is almost identical. He says: "An extremely moist and jelly-like cellular structure connects the tubercle with the surrounding cerebral tissue. . . . . This stratum further contains, scattered mostly through its inner part, some small gray or grayish-yellow tubercles, which occasionally unite with the great central mass." —Loc. cit. vol. iii. p. 429.

[3] Vide Andral's Précis d'Anat. Pathol. t. iii. p. 841.

[4] Dr. Ogle, Brit. and For. Rev., Oct. 1864, p. 463.

[5] Trans. of Path. Soc., vol. v. p. 26.

recorded. Dr. Aitken saw a "gummatous tumor" occupying the left optic thalamus, in a patient of Dr. Goodfellow's, who had suffered from syphilis, and some of whose children had died from inherited secondary syphilitic lesions.

Dr. Wilks has never seen independent tumors of this kind in the cerebral substance, though he believes, from the symptoms observed in some cases, that such deposits were very likely to have existed. A firm, tough, yellowish, lymph-like mass, of syphilitic origin, has frequently been met with intervening between and connecting the dura mater with the brain. And although it seems most probable that the primary seat of this is the dura mater, still it is desirable to mention it here on account of the serious way in which the brain matter is often implicated. Dr. Wilks records[1] a characteristic example of this kind of lesion, met with in the post-mortem examination of a woman of low character, who was believed to have suffered from syphilis. He says: "At the anterior fossa the dura mater was united to the bone by a firm, yellow lymph; here also the bone was slightly roughened, but not carious. The dura mater on the inner side was firmly and inextricably united to the anterior lobes of the brain, especially on the right side, and corresponding to the anterior fossa of the skull. On attempting to separate them, a quantity of hard yellow material was seen uniting them together. This filled up the sulci, and involved the cineritious substance. On the right side it had penetrated to the medullary matter, and here the adventitious substance formed a tumor, tolerably circumscribed on its deep side, the size of a walnut." In the liver were some of the characteristic tough masses, corresponding with a puckered and cicatriform condition of the surface above them.

3. *Cancer.*—Cancerous growths in the brain are, according to Lebert, decidedly more frequent in the second half of life, though they are met with occasionally in youth, or even in childhood. Dr. Walshe also found that out of 56 persons affected with cerebral cancer, 26 died between the ages of 40 and 60 inclusive, whilst 5 died before the 10th year, and 5 died between the 10th and 20th years. In about one-half of the total number of cases cancer of the brain is primary.

All three forms of cancer may occur in the brain, though encephaloid is by far the most common: next to this Lebert speaks of a lardaceous intermediate kind. It may exist either in the form of a distinct tumor, or it may infiltrate parts of the brain. The growths are usually solitary, though occasionally two or even more may be met with. The size of the cancerous mass varies from that of a pea up to an orange, or even larger. Occasionally the greater part of one hemisphere may be implicated. The cancerous mass is very rarely inclosed in a sort of fibrous cyst, but in the majority of cases it passes, at some part of its periphery, almost insensibly into the adjacent brain tissue. The color is occasionally the same as that of brain tissue, though various tints of rose, yellow, and even green may be met with either singly or intermixed: very many cancerous growths in the brain are said to have a yellow color. All parts of the brain are liable to be affected. Cancerous tumors are frequently found imbedded in the midst of the hemispheres, and, according to Lebert, those near the convexity usually attain the largest size, whilst those in the pons and medulla are usually the smallest, owing to the more rapid death of the patient when the growth occurs in these situations. The duration of life varies considerably; thus in 6 out of 11 cases inquired into by Lebert, the growth seemed to have proved fatal in about 6 months, whilst in 4 the symptoms extended over a period of from 2 to 5 years.

The consistence and amount of vascularity of the cancerous growth vary much in different cases. Effusions of blood may be met with in the midst of soft cancerous masses, and not unfrequently cysts are developed in their interior, which contain a thick glairy fluid. The surrounding brain tissue may be natural (which is frequently the case), or it may be softened, or, still more rarely, in a state of induration. The softening may be white, red, or of the yellow[1] variety.

Lebert records one instance of a cerebral cancer which, by its progressive growth, caused a large perforation of the skull, in the situation of the coronal suture.

4. *Melanotic Tumors.*—These are found in the shape of small nodules, generally varying in size from that of a pea to a bean. They may exist in the deeper parts of the brain, or at its surface, in the gray matter of the convolutions.[2] Sometimes these growths may be cancerous in their nature, but others are certainly not so. The black color is due to the infiltration of the cells of the growth with black granular pigment, similar to that met with in the choroid coat of the eye. Dr. Clendinning[3] found a mass of melanoid deposit in the upper part of the right corpus striatum, as large as a horse-bean,

[1] Guy's Hosp. Rep. 1863, p. 49.

[1] Dr. Ogle, in Journal of Mental Science, 1864, p. 229, cases 1 and 4.

[2] Hooper's Morb. Anat. of Human Brain, 1828, pl. xii. figs. 2 and 3.

[3] Trans. of Path. Soc., vol. i. p. 42.

and, external to this, a hard pea-sized mass. Similar new formations existed in the centrum ovale, and in the right lobe of the cerebellum. In this individual, growths of the same kind existed also, in great numbers, in the subcutaneous tissue, and in most of the internal organs except the lungs.

5. *Gliomata.*—These growths, to which the above name has been given by Virchow, take their origin in the neuroglia or interstitial connective tissue of the brain. They are, in fact, formed by a localized hyperplasia of the neuroglia, and contain no nerve elements in their composition. These tumors are never sharply defined from the surrounding brain tissue, to which they bear a certain superficial resemblance. On section, however, they are often seen to have a somewhat translucent, bluish-white appearance, whilst at the same time they may be firmer and rather more vascular than the brain tissue itself. Gliomata are usually solitary, and of slow growth, so that they may exist for a long time without producing any very appreciable symptoms. They often attain to a considerable size—that of an orange, for instance, and occasionally they are even much larger than this. They are most frequently met with in one or other of the posterior cerebral lobes, and after this, perhaps, they occur on some part of the upper and outer portion of the cerebral hemispheres oftener than in other situations. The tumors are composed of an intercellular substance, which varies in quality and consistence in different parts of the brain, and of an abundant mixture of cells and nuclei. The cells are variable in shape and size—the smaller ones occasionally possessing fine prolongations which are continuous with those of adjoining cells. There are two principal varieties of gliomata: the soft and the hard. The former, containing a soft basis substance, and numerous moderately large cells, are closely allied to medullary sarcomata; whilst the latter, having a harder and firmer basis substance, and small cells with highly refractive nuclei, have close affinities to fibrous tumors.

Dr. Cayley[1] has recorded an interesting case in which a tumor of this kind, about the size of a large walnut, involved all the deeper parts of the right side of the pons, the right processus ad testes, the corpora quadrigemina to some extent, the right half of the valve of Vieussens, and the fibres of origin of the right fourth and fifth nerves.

6. *Fibro-plastic Tumors.*—These growths most commonly arise from the pia mater; still occasionally they take their origin in the substance of the brain itself. They have been found in the midst of the hemispheres, in the pons, and in the cerebral peduncles. They vary in size from a pea to a hen's egg, are mostly spherical or ovoid in shape, and with a surface which is often mammillated or slightly lobulated. Their color is generally rose-red, mixed with yellowish and even greenish tints. The amount of vascularity differs in different tumors, and in different parts of the same growth. They contract no adhesions, and, in general, do not infiltrate neighboring parts, although they erode by their growth and consequent pressure.

Dr. Bristowe has recorded[1] a characteristic instance of the occurrence of a tumor of this kind in a man aged 33 years. The growth was irregularly spherical, and about one square inch in bulk. It arose from the right half of the pons and from the corresponding crus cerebelli, whence it extended for a certain distance into the medulla oblongata. The surface was lobulated, and had somewhat the appearance of brain substance, owing to its color and the arrangement of vessels on its surface. There was no defined limits to the deeper portion of the tumor, which passed insensibly into the surrounding brain tissue. On section, the substance of the growth was grayish and slightly translucent, interspersed with patches in which the vascularity was more marked than it was elsewhere.

Dr. Ogle[2] has reported a case of fibroplastic infiltration, in which the new product, instead of forming a distinct tumor, had infiltrated itself into the tissue of the left optic thalamus, so as to make this body almost twice its natural size.

7. *Fibrous Tumors.*—These growths are very rare, and comparatively few cases are on record. They are mostly small and spherical, varying in size between that of a small pea and a walnut. M. Reignier[3] found a pedunculated growth of this kind of the size of a large filbert, growing from the valve of Vieussens; and in the Trans. of the Path. Soc., vol. v. p. 18, an account is given of a fibrous tumor about the same size, which was found projecting into the left lateral ventricle, from the side of the corpus striatum. Lebert records two cases: in one a tumor of the size of a pea was found in the pons, composed of a firm, elastic, yellow, and somewhat gelatinous tissue, but presenting the usual microscopic characters; in the other, 17 fibrous tumors were situated upon the ependyma of the lateral ventricles, varying in size between a pea and a small cherry stone, and of a white or slightly yellowish, or even rose color in some places. On sec-

[1] Trans. of Path. Soc., vol. xvi. 1865, p. 23.

[1] Ibid., vol. vii. p. 28.

[2] Trans. of Path. Soc., vol. vii. p. 12, pl. ii.

[3] Bullet. de la Soc. Anat., t. ix. p. 120.

tion they were homogeneous, and somewhat translucent. Several tumors were situated on the septum, and superficially they were all covered by epithelial cells, similar to those of the ependyma. The substance of the brain, in other respects, was apparently quite healthy, and there was no excess of fluid in the ventricles.[1]

8. *Osseous Tumors.*—True bony growths in the substance of the brain are extremely rare, still they have been met with. Dr. Bristowe[2] found a growth of this kind occupying the position of the infundibulum and corpora albicantia. It was a hard conical mass about as large as a horse-bean, whose apex rested on the pituitary body, and whose base assisted in forming the floor of the third ventricle. It "was wholly unconnected with the dura mater or osseous parietes of the skull." On microscopic examination, it presented the characters of the true osseous tissue, with perfect lacunæ and canaliculi.

9. *Tumors of the Pituitary and Pineal Bodies.*—Both these bodies are occasionally found in a morbid condition, and more or less enlarged. Cysts are then frequently met with in their interior.

a. Pituitary Body.—Lebert considers the enlargement of this body to be a kind of hypertrophy. Several cases are on record. In one of them related by Rayer,[3] this body was about 1½″ in diameter, whilst its tissue was also more dense and resistant than natural. Vieussens[4] found the "gland" as large as a hen's egg, soft, and containing in its interior a grayish-white glutinous fluid. Heslop[5] records a remarkable case in which the tumor was soft, deep gray, and of the size of a large walnut, containing a small cavity with fluid in its interior. It occupied the region of the pituitary body, and also extended posteriorly as far back as the pons, and antero-laterally to the fissures of Silvius, so as to occupy the whole interpeduncular space. The corpora quadrigemina were flattened antero-posteriorly, from pressure. Abercrombie[6] also refers to a case described by Dr. Powell, in which there was found "a tumor of the size of a hen's egg, containing a thick purulent fluid under the anterior part of the brain, and interposed betwixt the optic nerves, which were much separated by it from each other. Below it was attached to the pituitary gland, which was very soft, and enlarged to five or six times its natural size." Davaine[1] records three cases in which small cysts (supposed to be hydatids) were found in the pituitary body.

b. Pineal Body.—Hooper[2] says, speaking of this body: "It is sometimes converted into a *cyst*, the whole of the natural structure being destroyed. This cyst is firm and membranous, and I have seen it of the size of a tamarind stone. The contents of one which I examined were, a turbid serous fluid, with small particles of solid albumen." Dr. Ogle[3] also relates a case in which "the brain and membranes were natural, excepting that the pineal gland was exceedingly enlarged and very adherent, posteriorly, and contained two cavities, each full of transparent fluid, situated immediately below its investing membrane."

10. *Formation of Gray Matter upon the Ependyma of the Ventricles.*—Rokitansky and Virchow[4] have both described the existence of cerebral gray matter upon the internal surface of the lateral ventricles, in situations where, naturally, gray matter does not exist. It occurs in the form of numerous small tubercles, from the size of a mustard seed to that of a cherry stone.

11. *Vascular erectile Tumors.*—These growths are very rare. Nevertheless, Lebert has given the particulars of five cases in which they were found.[5] In all they were discovered *post mortem*, but had given rise to no notable symptoms during life. In one of these cases the growth was lodged in the right lateral ventricle, and was a development from the choroid plexus, but in the other four the tumors were imbedded in the cerebral substance.

In the case related by Farre[6] the growths were multiple; two of the same size being met with in the medullary substance of the left hemisphere, and several small growths in the corpora striata and cerebellum. Lebert[7] himself has minutely described an erectile tumor, of the size of a hen's egg, found in the posterior lobe of the right cerebral hemisphere, and Luschka,[8] met with one of the same size

[1] Anat. Path., vol. ii. p. 71.
[2] Trans. of Path. Soc., vol. vi. p. 25.
[3] Archiv. Gén. de Méd., 1re Sér. 1823, t. iii. p. 350.
[4] Nov. Vasor. Corp. humani Syst.; Amstelodami, 1705, p. 248.
[5] Dublin Quarterly Journal of Medicine, Nov. 1848.
[6] Diseases of the Brain, &c., 3d ed. 1836, p. 438.

[1] Traité des Entozoaires. Paris, 1860, p. 656.
[2] Morb. Anat. of Human Brain, 1828, p. 43, pl. xii. fig. 8.
[3] Brit. and For. Rev., July, 1865, p. 235. He also adds that the Museums of King's College and of St. Thomas's Hospitals contain one specimen each of an enlarged pineal gland, hollowed out into a cyst.
[4] Würtzburger Verhandlungen, t. ii. p. 167.
[5] Guérard, Bullet. de la Soc. Anat., t. viii. p. 223.
[6] Leubuscher, Die Patholog. und Therap. der Gehirnkrankheiten, p. 413, Berlin. Original reference not ascertained.
[7] Anat. Path., t. i. p. 213.
[8] Archiv für Path. Anat., t. vi 1854, p. 458.

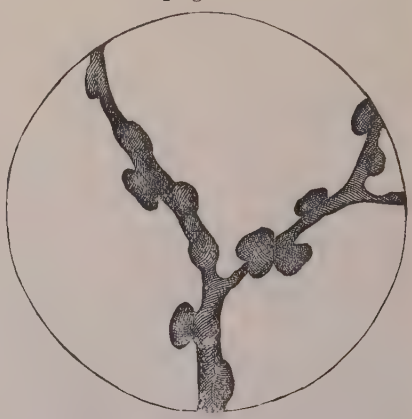
in the left anterior cerebral lobe, which was surrounded by softened brain substance. Lastly, Förster[1] alludes briefly to an erectile tumor of the size of a nut, found in the gray cortical substance of one of the hemispheres, the cavernous spaces of which were found to communicate with neighboring dilated veins. The tumors described by Lebert, Farre, and Guérard were made up almost entirely of fine vascular ramifications.

12. *Aneurisms.*—The intracranial aneurisms which are best known are those occurring on some one or other of the larger arterial trunks at the base of the brain, or on some of the branches of the circle of Willis lying in the midst of the pia mater and therefore whilst they are still on the surface of the proper brain substance. Such aneurisms belong to the meninges, and will not be further referred to in this place.

There are, however, aneurisms belonging to the encephalon itself, whose existence has only recently been discovered, and which are remarkable principally for their small size, and on account of their frequent numerical abundance within the same brain. These were first detected and examined by MM. Charcot and Bouchard,[2] the latter of whom has shown, not only their frequent and close association with the phenomena of intracranial hemorrhage, but also their apparent dependence upon a certain general pathological condition of the small encephalic arteries, which may exist alone, or may be associated, in various degrees, with the more familiar atheromatous degeneration. The pathological condition of the arteries favorable to the formation of these minute aneurisms is one of fibroid degeneration —a process of *sclerosis* in which there is brought about a great increase in the number of connective tissue nuclei on the perivascular sheaths, and also in the walls of the vessels, whilst the muscular fibre cells of the middle coat are gradually replaced by fibrous tissue. This change diminishes, or even destroys, the elasticity of the arterial coats, so that when, from any increase of the ordinary blood pressure, they have once become unduly dilated, they are unable to regain their normal calibre. In this way, by the incidence of increased pressure upon degenerated parts, are produced the various kinds of aneurismal dilatations, whose characters differ according to the degree and extent of the morbid changes in the parts involved. Thus, we may have uniform dilatation of an arterial branchlet, for a certain portion of its length, or this uniform dilatation may be interrupted by constrictions at intervals, owing to the presence of similarly situated sounder portions of the arterial walls. The kind of alteration with which we are more particularly concerned at present, however, and which is also the most frequent, is due to an altogether local and circumscribed change, and results in the formation of the minute and more or less spherical *miliary aneurisms*, as MM. Charcot and Bouchard propose to name them.[1]

These miliary aneurisms are very rarely met with before the middle of life, and

[Fig. 52.



Miliary Aneurisms. (Hamilton.)]

are most common in the very aged. They are visible to the naked eye, and can be seen readily with the aid of a pocket lens. Their diameter varies between $\frac{1}{125}''$ and $\frac{1}{25}''$, and they are attached to vessels which seldom exceed $\frac{1}{50}''$ in diameter. Sometimes only two or three can be detected in the same brain, though more frequently they exist in much larger numbers. Bouchard has found even more than one hundred in the same brain. They may be met with in all parts of the encephalon, though with different degrees of frequency in different situations. Hitherto they have been found most frequently in the optic thalami, and then, in decreasing order, in the pons Varolii, the cerebral convolutions, the corpora striata, the cerebellum, the medulla oblongata, the middle cerebral peduncles, and, lastly, in the white matter of the cerebral hemispheres. When abundant in the convolu-

[1] Lehrb. der Patholog. Anat., p. 418.

[2] Bouchard, De la Pathologie des Hémorrhagies cérébrales. Paris, 1867. See also joint papers by MM. Charcot and Bouchard, in the Journal de Physiologie, 1868–69.

[1] Cruveilhier (Anat. Patholog., Liv. xxxiii. pl. ii. fig. 3) figures and gives an accurate description, so far as it goes, of these very miliary aneurisms under the name of "apoplexie capillaire à foyers miliares." He was therefore ignorant of their real nature, though perfectly familiar with the naked-eye appearance.

tional gray matter, a number of minute and variously colored spots may be seen, after the membranes have been stripped off, lying exposed on the surface of the convolutions; whilst, when sections are made, others may be recognized in the deeper strata of the gray matter. Whether occurring in this situation or in the more central parts of the brain, the color of the minute aneurismal grains varies from a bright red or violet, to a yellowish or even black hue;[1] according as they contain in their interior normal fluid blood, or more or less altered blood pigment. Occasionally the aneurisms seem to undergo a natural process of cure. Their wall, as well as that of the enveloping and sometimes adherent lymphatic sheath, becomes thickened by an increased growth of connective tissue elements, whilst at the same time the white corpuscles of the blood have a tendency to adhere to their inner surface. The fibroid change creeps on, bringing about, sooner or later, a union between the wall of the aneurism and its sheath, the gradual thickening of these, and an extension of growth inwards, probably owing to a further organization taking place in the substance of the adherent and fused white corpuscles. Thus may the cavity of the aneurism be gradually diminished, till at last this, and even the minute vessels on which it is situated, may undergo complete obliteration.

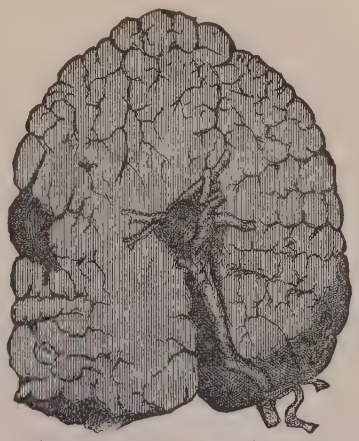
Such intra-cerebral "miliary" aneurisms may coexist with other and much larger aneurisms of the vessels of the pia mater. Bouchard speaks of a case of this kind where the aneurisms of the arteries of the pia mater were not only exceedingly numerous, but varied in size between a pea and a cherry stone. In other cases, minute aneurisms of these meningeal arteries may be met with precisely similar to those coexisting on the vessels in the midst of the brain substance. Frequently when miliary aneurisms are met with in the smaller cerebral arteries, the larger arteries at the base of the brain exhibit marked atheromatous changes: such a coincidence, however, is by no means invariable.

Although the majority of intra-encephalic aneurisms are minute, and such as we have described, occasionally others of larger dimensions are met with. The size of the largest, however, could rarely exceed that of a small walnut, seeing that the arteries of the brain are comparatively small before they leave the pia mater to penetrate into its substance.

[1] Occasionally this blood pigment, in the form of amorphous yellow grains mixed with hæmatoidine crystals, may be principally collected around one of these aneurisms (which has been ruptured), though within its enveloping lymphatic sheath.

We have collected the records of five cases of this kind of aneurism, and in each of these the patient's death was occasioned by the rupture of the sac. The

[Fig. 53.



Cerebellar Aneurism. (Bristowe.)

first is related by Dr. Crisp,[1] and in this a boy aged fourteen died from the rupture of one or two small aneurisms on the anterior cerebral artery, in the substance of the anterior lobe. The aneurism which burst was as large as a horse bean, whilst the other was about the size of a pea, and was filled with laminated fibrin. The next case was recorded by Dr. Van der Byl,[2] and was altogether remarkable from the fact that the aneurism, which was situated on the left posterior cerebral artery in the substance of the brain, was as large as a hen's egg, and was almost filled with laminated fibrin. In one case recorded by Dr. Gull,[3] a small pyriform aneurism, "having much the appearance and size of a withered grain of wheat," burst in the centre of the pons Varolii, and was found in the midst of a coagulum weighing two drachms. Dr. Gull gives the details of another case in which an aneurism about the size of a small filbert, situated on the middle cerebral artery, in the anterior part of the middle cerebral lobe, was found in a girl aged seventeen, surrounded by a large recent coagulum, and by softened brain tissue. The other arteries of the brain are said to have been healthy. Lastly, the writer has himself recorded a case[4] in which an aneurism, about $\frac{1}{3}''$ in length and of an elongated pyriform shape, with a distinct rupture in its larger extremity, was taken from the midst of an enormous effusion of blood

[1] Diseases of Arteries, p. 165.
[2] Trans. of Path. Soc., vol. vii. p. 129.
[3] Guy's Hosp. Reports, 3d Series, vol. v. (1859), p. 297.
[4] Trans. of Path. Soc., vol. xviii. 1867.

into the outer part of the right corpus striatum and adjacent portions of the hemisphere. In this case there was an atheromatous condition of the arteries at the base of the brain, and, besides the larger aneurism, four or five of the small miliary aneurisms were found in different parts of the organ.[1]

13. *Bloodclots.* See Art. "Apoplexy."

14. *Abscesses.* See Art. "Abscess in Brain."

15. *Plastic Lymph on the surface of the Ventricles.*—A well-marked instance of this has been related by Dr. Wilks.[2] It occurred in a man who had fractured the left orbital plate of his frontal bone by a fall. A portion of the broken bone had torn through the dura mater and had injured the anterior lobe of the brain, so as to lead to the subsequent production of an abscess in this situation. The man died after seventeen days: "on incising the roof of the ventricle (left) a membrane was found within it; and on cutting this through it was found to consist of a layer of lymph, which completely lined the cavity. Some purulent matter escaped from within it. It covered the roof, the floor, and extended from the anterior to the descending cornu, and was so tough that it was capable of being removed entire; it formed, indeed, a complete cast of the cavity, and resembled a croupous membrane, as seen on the trachea in inflammation of that organ. The surface of the ventricle was soft, and in parts tore when the membrane was removed; but in most places it could be cleanly taken off. The foramen of Munro was closed, and the right ventricle contained only some turbid serum." It was uncertain whether there was a communication between the abscess and the ventricle, but the lymph was undoubtedly produced on the surface of the latter.

16. *Cysts.*—It seems extremely doubtful whether simple serous cysts are ever met with in the substance of the unaltered brain tissue. Those most likely to be of this nature are small cysts from the size of a pin's head to that of a mustard seed, which are sometimes met with beneath or projecting above the surface of the lateral ventricles. They occur either singly or in groups.

The corpora striata, on section, sometimes present the appearance of small cysts, even as large as a pea. These may, however, be either sections of dilated lymphatic canals, or cavities left after minute softenings.

It is true that larger cysts are not unfrequently met with in the brain, but these, when not due to one of the two forms of cystic entozoa, to be hereafter described, should rather be termed pseudocysts, since they are not primary formations, but have, in all probability, resulted from the modification of pre-existing pathological states. Such cavities or pseudocysts mostly result from the later changes taking place in the seat of old effusions of blood,[1] or of circumscribed softenings; or else they are due to the softening of encysted tubercular[2] or cancerous nodules.

Cysts occasionally form in the substance of cancerous growths in the brain, and, as before stated, they have several times been met with in the interior of enlarged pituitary and pineal bodies.

17. *Calcareous and other Concretions.*—These are not unfrequently met with in the brain, and are mostly due to changes which have taken place in masses of tubercle or in old abscesses. Such concretions consist for the most part of phosphate and carbonate of lime, and only contain a small quantity of animal matter. More rarely concretions may be met with which seem to have resulted from previous effusions of blood: thus Lassaigne[3] analyzed a mass which was found to be composed almost wholly of fibrine, and contained only four per cent. of phosphate and carbonate of lime, with traces of cholesterine.

Concretions known as "brain sand" are very common on or in the pineal gland and its peduncles.

18. *Entozoa.*—Two kinds of parasites only have been met with in the human brain, and these always in an immature or larval condition. They are the *Cysticercus*, and the *Hydatid* or *Echinococcus cyst:* the first representing the second, or *scolex* stage, in the development of *Tænia Solium*, and the other an equivalent stage of *T. Echinococcus*, an animal which exists abundantly in its mature condition in the alimentary canal of dogs. Goeze and Zeder[4] have recorded two cases in which they suppose the *Cœnurus cerebralis* to have been met with, but they have not been supported by other observers, and Davaine believes these cases, in reality, to have been instances of hydatid disease. Certainly, as he says, the descriptions these writers have given are obscure and inexact, and we may well imagine mistakes to have been made, when we consider what was the state of knowledge concerning helminths at the time in which they wrote.

a. Cysticerci.—In the brain these vary

[1] See an article on Intra-cranial Aneurisms, by Dr. J. H. Hutchinson, in Penna. Hospital Reports, vol. ii. 1869.—H.]

[2] Trans. of Path. Soc., vol. xv. p. 5.

[1] Many cases are recorded by Dr. Ogle (Med.-Chir. Rev., July, 1865, p. 212).

[2] Trans. of Path. Soc., vol. v. p. 26.

[3] Clinique Méd., t. v. p. 8.

[4] Nachtrag zur Naturgesch. der Eingeweidewurmer, 1800, pp. 308 and 313, tab. ii. figs. 5–7.

in size from that of a pea to a small horse-bean, or even larger. The serous cysts, in which they are usually inclosed in other situations, are often absent entirely, so that they are bounded only by a smooth layer of unaltered or somewhat compressed brain substance. They often exist in large numbers in the same brain, and are very rarely solitary. From ten to twenty are frequently met with. Cruveilhier[1] records an instance in which more than 100 were found within the cranium of the same individual, and of these about 50 were lodged in the cerebellum. They may be found in almost all parts of the brain, but, speaking generally, they are by far the most abundant at the surface of the brain, in, or in close connection with the gray matter of the convolutions. They are extremely frequent in the pia mater, also where they press upon and partially imbed themselves in the surface gray matter. Sometimes they are lodged in the midst of the gray matter itself, whilst more frequently still they are found intervening between this and the white substance. They have, moreover, been seen in the midst of the white substance, in the central ganglia, in the pons, in the crura cerebri, and in the cerebellum as before stated; whilst Cruveilhier says he has seen real cysticerci in the choroid plexuses of the lateral ventricles. In the latter situation they have to be carefully discriminated from the small, non-parasitic cysts which so frequently occur in the same locality.

Although usually giving rise to but slight changes in the surrounding brain matter, the cysticerci themselves undergo important modifications with age. It is desirable that this fact should be known, in order that pathologists may recognize them in their different stages, and that individual developmental modifications may not be mistaken for specific distinctions. According to Davaine,[2] "Les altérations portent, d'une part, sur la vésicule qui est devenue plus ou moins globuleuse, plus volumineuse, sans jamais cependant avoir acquis un grand volume, irrégulière, quelquefois divisée en lobules ou même double; d'une autre part elles portent sur la tête dont le rostre et les ventouses sont envahis par une matière noirâtre, pigmentaire. Les crochets sont recouverts à leur base par cette matière. Dans une période plus avancée on les trouve en désordre, diminués de nombres ou même ils ont disparu. L'ouverture de la vésicule rétrécie ou oblitérée ne laisse plus sortir le corps; la tête invaginée dans celui-ci ne peut non plus en être extraite par une pression ménagée; sa présence ne peut être reconnue que par la dilacération des parties." It should also be added that in those cases where the cysticercus is non-encysted—as when it is lodged freely in one or other of the ventricles—it tends to grow more easily into the form of a tapeworm, by the elongation and segmentation of the neck of the larval animal.[1]

The cysticerci seem to occur pretty frequently in both sexes. They may be met with also at all ages beyond infancy, though, as Cruveilhier has remarked, they seem to be most frequent in the latter half of life, and have often been met with in very old people.[2]

b. Hydatids.[3]—In the brain, as in other

[1] Anat. Path. Gén., t. ii. p. 88. Paris, 1852.

[2] Traité des Entozoaires. Paris, 1860, p. 657.

[1] Thus constituting the third stage of development, when the animal is termed a *strobilus*. See Brit. Med. Journ. 1859, p. 272, where a specimen, apparently in this stage of development, is recorded to have been met with in connection with the fourth ventricle.

[2] Dr. Cobbold says that more than 100 cases of cysticerci in the brain are on record. References to many may be found in his *Entozoa*, p. 224, and Professor Griesinger (Archiv der Heilkunde, 1862) has analyzed the details of between fifty and sixty of these cases.

[3] The following remarks do not refer in any way to hydatids having their seat in or between the membranes of the brain. From various sources I have ascertained the details of thirty cases of hydatids contained within the cerebrum and cerebellum. I have seen references to a few other cases also, of which I have not been able to ascertain the details. For many of the references I have been indebted to Davaine's Traité des Entozoaires, Dr. Cobbold's Entozoa, and Dr. Ogle's paper in the Med.-Chir. Rev. July, 1865, p. 206. The references to these 30 cases are: 1. *Martinet*, Lond. Med. Repos. 1824, vol. ii. p. 408.—2. *Baily*, Lond. Med. Repos. 1826, vol. ii. p. 144.—3. *Morrah*, Med.-Chir. Trans., vol. ii. p. 262.—4. *Hooper*, Morb. Anat. of Human Brain, 1826, pl. xiv. p. 65.—5. *Dalgleish*, Lancet, 1832, p. 168.—6. *Guérard*, Lancet, 1835, p. 45.—7. *Bree*, Lancet, 1837, p. 53.—8. *Sturton*, Lancet, 1840, p. 494.—9. *Berncastle*, Lancet, 1846, p. 635.—10. *Barker*, Trans. of Path. Soc. 1859, vol. x. p. 6.—11. *Baillarger*, Brit. Med. Jour. 1861, p. 286.—12. *Risdon Bennett*, Med. Times, 1862, p. 80.—13. *Ogle*, Brit. and For. Rev. July, 1865, p. 207.—14. St. Thomas's Hosp. Mus. No. 101.—15. St. Barthol. Hosp. Mus. No. 60.—16. *Davaine*, Gaz. Méd. de Paris, 1862.—17. *Abercrombie*, Diseases of the Brain, &c., 3d ed. 1836, p. 447.—18. *Zeder*, Davaine's Traité des Entoz. 1860, p. 644.—19. *Barth*, Bull. Soc. Anat. ann. xxvii. 1852, p. 108.—20. *Calmeil*, Dict. de Méd., art. Encéphale, t. xi. p. 588, 1835.—21. *Faton*, Bull. Soc. Anat. 1848, p. 344.—22. *Becquerel*, Gaz. Méd. de Paris, 1837, p. 406.—23. *Rendtorff*, Dissert. de Hydat., cap. 10, p. 22, Berlin, 1822; and *Livois*, Rech. sur les Echinoc. p. 100, Thèse, Paris. 1843.—24. *Cazeaux*, Bull. Soc. Anat.

organs, the hydatid or hydatids are mostly inclosed within an outer sac or cyst. In this organ, however, it is generally very thin, and in some cases it has been stated to have been altogether absent, the hydatid membrane pressing immediately against the compressed brain tissue. When they occur in the lateral ventricles, the enveloping cyst is always absent. In the great majority of cases, only one hydated cyst is met with, though this may contain two, three, or more hydatids of different sizes; usually, however, a single cyst exists containing a single hydatid. The size of the cyst varies, generally, from that of a marble up to a large orange, though occasionally this limit is much exceeded. Thus, in a case observed by Mr. Headington and reported by Dr. Abercrombie,[1] an immense hydatid cyst was found within the left lateral ventricle, which nearly extended to the circumference of the brain on the same side, and "contained about sixteen ounces of limpid fluid;" and in another case, recorded by Rendtorff, an enormous mass of hydatids weighing two and a half pounds, was found in the same situation, in a girl only eight years of age. The cyst is frequently lodged in the centre of the white matter of one of the hemispheres, and it may increase in size till it occupies almost the whole of one of the lobes—anterior, middle, or posterior, as the case may be. Occasionally it occupies the greater part of two contiguous lobes, and may project towards the circumference, as well as into the lateral ventricle. In both these situations the cyst may be covered only by a thin layer of nerve substance; or it may be uncovered, owing to the brain tissue having disappeared under the influence of the gradually increasing pressure. I have only found one case on record in which an hydatid cyst was lodged in the substance of the cerebellum; in this instance, however, it was large, measuring three inches by two, projecting into the fourth ventricle, and extending transversely across from the right to the left lobe, so as to be covered by a coating of brain matter at each extremity not thicker than a wafer.

Although usually only one hydatid cyst is met with in the brain, still sometimes two or three, or even many, are encountered in different parts of the organ. In these cases their size is generally in the inverse proportion to their number; so that in some instances, instead of meeting with one large cyst, such as we have before alluded to, we encounter a number of little ones varying in size between a mustard seed and a hazel-nut.[1]

As an instance of multiple hydatids I may refer to a case recorded by Léveillé, and quoted by Davaine, in which many were found in the meninges and at the surface of the brain, in the corpus callosum, in the left middle cerebral lobe, in the right optic thalamus, and in other parts.

The increase in size of the hydatid being usually slow and gradual, little or no change is generally observed in the surrounding brain substance, which gradually atrophies under the pressure of the growing cyst. But occasionally congestion or softening does occur in the surrounding brain tissue, and, more rarely still, the presence of an hydatid in some portion of the brain seems to excite changes in the whole organ, and even in the cranium. Thus in the case of hydatid in the cerebellum, before alluded to, occurring in a man 24 years of age, the brain was found to be denser and firmer than usual, the ventricles distended with four ounces of clear fluid, and the skull-cap extremely thin, having a medium thickness of not more than about $\frac{1}{10}''$, and at the squamous portions of the temporal bone being quite wafer-like, and not more than $\frac{1}{30}''$ in thickness. In a remarkable case recorded by Dr. Barker (10): "The calvaria was healthy but exceedingly thin, so as to be transparent in numerous places; the outer surface was natural, but the inner presented a series of shallow depressions, separated by angular ridges, evidently produced by the long-continued pressure of the subjacent convolutions, of which they presented an accurate mould. The surface also was congested, rough, and softer than natural. The base of the skull and its dura mater were healthy. There was no sub-arachnoid fluid, the convolutions being compressed against each other, and against the parietes, so as to obliterate the sulci; the surface was not congested. In the posterior lobe of the right cerebral hemisphere was a hydated cyst, occupying nearly the whole

ann. viii. 1833, p. 106.—25. *Carrère*, Dict. de Méd. de Chir. et d'Hygiène Vétérin. 1839, t. vi. p. 157, art. Tournis.—26. *Tonnelé*, Bull. Soc. Anat. ann. xxvi. 1851, p. 165, case xxxi. —27. *Chomel*, Gaz. des Hôpit., t. x. 1836, p. 619.—28. *Montansey*, Bull. Soc. Anat. ann. ii. 1827, p. 188.—29. *Aran*, Arch. Gén. de Med., 3me. Sér. t. xii. 1841, p. 98.—30. *Leroux*, Cours sur les Gé. ér. de Méd. prat., t. ii. p. 12, Paris, 1825.

[1] Diseases of the Brain, &c. 3d edit. 1836, p. 447, case xxxiii.

[1] In cases of multiple hydatids, their small size may be explained by the fatal nature of the malady, and the early death of the patient. The duration of the life of the patient naturally varies according to the situation and number of the hydatids. Davaine records one case (loc. cit. p. 650) in which a large single cyst must have been four years old.

lobe, which was thus converted into a fluctuating cyst. It had rendered the lobe irregular, and lobulated, and increased its dimensions; but the hydatid was covered everywhere by brain substance, although in many situations it was a mere film. The lobulated character seemed to have been produced by the superficial veins acting as ligatures. The cyst was single, about as large as a middling-sized orange, and contained two hydatids, one nearly as large as the cyst itself, the other the size of a walnut. They contained no secondary cysts: the brain in all other respects was healthy."

The hydatids met with in the brain are almost always barren, and thus correspond with the *acephalocysts* of Laennec. Sometimes they are perfectly simple, but they may contain smaller secondary cysts in their interior, or bear them as buds on their exterior surface. The hydatids usually contain a clear, limpid fluid, and their walls are made up of the usual thin, structureless, and concentrically arranged lamellæ. In only two (12 and 23) out of the thirty cases of which I have read the details, is any mention made of the hydatids containing echinococci or their remains. In these fertile cysts, in addition to the echinococci, the remains of the fibro-granular germinal membrane may be detected on the internal surface of the hydatid. Many cases of so-called hydatids in the brain are recorded by old writers, which have but a very doubtful right to this title. The word was formerly used with great laxity; everything in the shape of a cyst receiving this appellation —even the vesicles so common in the choroid plexus, which are now known, in the great majority of cases, to be simple serous cysts.

Hydatids in the brain seem to occur as often in the one sex as in the other. As regards time of life, they seem to be met with, in the great majority of cases, in individuals between the ages of 10 and 30 years. I have found the age of the patient stated in 24 out of 30 cases: of these, 3 were below 10 years of age (5, 7, and 8 years), and 3 above 30 years (one "middle age," 37, and 38), whilst the remaining 18 were between the ages of 10 and 30 inclusive.[1] This is very notable, and in striking contrast with what is known concerning the cysticercus and its tendency to occur rather in the latter half of life, than in younger individuals. We know so little as to the mode in which the human body becomes infected with these cystic entozoa, that it is extremely difficult to explain such peculiarities. We do know, however, that the adult or fully developed condition of the echinococcus hydatid exists in the intestines of the dog, though, of course, not in that of all dogs, and perhaps we may also say that individuals between the ages of 10 and 20 years, have generally more to do with these animals than those of an earlier or more advanced age. This is a mere suggestion which, unfortunately, we are unable further to develop.

19. *Silver and Lead.*—In the 11th vol. of the Transactions of the Pathological Society an account is given by Mr. Sydney Jones of the post-mortem appearances in an old epileptic, who had for several years been in the habit of taking nitrate of silver as a remedy. "The choroid plexuses were remarkably dark: from their surface could be scraped a brownish black soot-like material; a similar substance was found lying quite free in the cavity of the fourth ventricle, apparently detached from the choroid plexus." A specimen of metallic silver was obtained from the plexus.

Lead has several times been detected by the aid of chemical analysis in the brain.

Hypertrophy.—The accounts given by Dance,[1] Andral,[2] and Rokitansky,[3] of the morbid appearance presented in the so-called hypertrophy of the brain, are so harmonious and striking as to point undoubtedly to some definite structural modification, differing altogether from the enlargement due to congestion. A similar condition has also been noticed, and more briefly alluded to, by Bouillaud[4] and Laennec.[5]

Dance and Andral give the post-mortem appearances met with in seven adults, one of whom was 39 years of age, whilst the others varied between 26 and 30. In these examinations the following pathological conditions were met with:—The skull was of average size and shape; a great turgescence of the brain was noticed on the removal of the skullcap, which became even more manifest when the dura mater was cut; the dura mater itself was rather thin, and the arachnoid and pia mater remarkably exsanguine, free from all moisture, and easily torn; the convolutions were completely flattened, and separated only by small lines of demarcation, instead of well-marked sulci; on section, the substance of the brain was found to be extremely anæmic, with much less than the usual distinction

[1] Several cases of hydatids in the membranes of the brain, of which I have read, have, however, been over this age.

[1] Répert. d'Anat. Patholog. par Breschet, 1828.

[2] Clinique Médicale, Trans. by Spillan, 1836, p. 174.

[3] Patholog. Anat. (Syd. Soc.), vol. iii. p. 373.

[4] Traité de l'Encéphalite. Paris, 1825.

[5] Journal de Méd. de Chirurg. et de Pharm., t. xi. p. 669.

between the gray and the white matter, owing to the extreme paleness of the former; the white substance presented an almost completely bloodless section, whilst its density and consistence were so much increased as to make it comparable to "the white of an egg hardened by boiling;" the ventricular cavities were very small, and quite devoid of fluid; and lastly, these changes, though affecting the whole of the cerebrum, did not extend to the cerebellum, pons, medulla, and cord, all of which had their natural consistence.

But there is another form of "hypertrophy" of the brain, which is of much more frequent occurrence. This is met with in some young children, who present obvious marks of being rickety, and is usually indicated by the existence of more or less enlargement and alteration in the shape of the cranium. The skull becomes especially prominent in the frontal region, and often approximates somewhat to the form met with in hydrocephalus. In this variety of hypertrophy, also, there is more or less compression of the brain, as indicated by the existence of anatomical characters similar to, though less strongly marked than those already described.

Whether any relationship exists between the hypertrophy of the brain as it occurs in adults, and that which occurs in childhood, is not known. Nor are we better informed as to the precise nature of the histological change. We neither know whether it is the same in both cases, nor what it is in either. Most pathologists seem to agree in the supposition, that the increased bulk is due to an augmentation of the interstitial substance or neuroglia of the cerebral hemispheres, rather than to an increase in the number or size of the proper nerve elements. So that, if this be true, the disease cannot be looked upon as a hypertrophy of the brain in any strict sense of the term. It has been held by Rokitansky that there is an actual increase in the amount of neuroglia, whilst Sir William Jenner and others are of opinion that, in childhood at least, and when associated with rickets, the enlargement of the brain is due to an infiltration, more particularly of the anterior lobes, with an albumenoid material similar to that met with in the liver, spleen, and other organs. If this be the case, then the brain substance ought to yield the ordinary reaction with iodine; and the characteristic changes in the coats of the arteries, peculiar to this albumenoid degeneration, should be recognizable. Accurate and careful microscopic observations as to the nature of the morbid changes have yet to be made, and without these no real light can be thrown upon the pathology of these remarkable affections.

There are, however, two other cases of hypertrophy of the brain on record, which differ notably in all respects from the forms to which I have just alluded. In both there was great enlargement of the cranium as well as of the brain; and, owing to this coincident enlargement of the brain and its case, there was not only an absence of the signs of compression of this organ during life, but also an utter absence of the pathological appearances peculiar to the other forms of hypertrophy. The particulars of one case, that of a child who died at the age of five years, have been narrated by Dr. Scoutetten;[1] whilst those of the other, a patient of Dr. Sweetman, who died when a little more than two years of age, have been detailed by Sir Thomas Watson.[2] In both these children, the head equalled that of an adult in size, the skull was somewhat thickened, the dura mater was unduly adherent to it, the arachnoid was moist, the pia mater fully injected, and the convolutions not at all flattened. The ventricles were small: in M. Scoutetten's case they contained a very slight amount of reddish serum, whilst in Dr. Sweetman's they were empty. In this latter case, also, the surfaces "of the medullary matter, exposed by repeated sections, presented very unusual vascularity." Nothing is said concerning the amount of vascularity in M. Scoutetten's case, and in neither of the reports is any mention made of an undue consistence or alteration in density of the nerve matter. The amount of brain substance above and behind the ventricles seemed to be more especially increased in the elder child, since to reach these, from above, an incision nearly three inches in depth was required. Regarding the nature and cause of this enlargement, we know even less than concerning the other forms. Is there an actual increased growth of brain substance—including a due proportion of nerve element proper, and of interstitial substance — or does the increased bulk, in these cases, also, result from an augmentation in bulk of the neuroglia alone? and even if this be the case, we may still inquire as to the nature of the change which it has undergone.

Much doubt also exists with regard to partial hypertrophies of the brain. Whilst admitting their extreme rarity, Rokitansky says:—"There can be no question that small portions of the brain really are separately hypertrophied. Many of the observations brought forward as instances of this occurrence are undoubtedly erroneous; adventitious formations infiltrated through the cerebral tissue may have occasioned at once the enlargement and the error. There are, however, some instances which may be relied on, in which

[1] Archiv. Gén. de Méd. 1825, t. vii. p. 44.

[2] Lect. on Princip. and Pract. of Physic, 4th ed., 1857, p. 427.

the optic thalamus and the pons were hypertrophied; and I have myself also met with a most remarkable case of hypertrophy of the medulla oblongata." Andral[1] also believed in the existence of a limited local hypertrophy of parts of the brain. These so-called hypertrophies were, in all probability, produced by a hyperplasia of the interstitial tissue of the parts, though it seems more than questionable whether we ought to follow Rokitansky, and apply this name to an increase of bulk which has been thus occasioned.

PROGNOSIS.—In all cases when the presence of an actual growth within the cranium is diagnosticated, the prognosis is very grave, but the degree of gravity depends upon the nature of the growth rather than upon the character or intensity of the special symptoms which it has occasioned. Thus, if there be reason to believe in the existence of carcinoma, the future prospects are as bad as they can be; if the conclusion be that syphilis is the cause of symptoms, there may be room for the hope of complete recovery. Without entering upon a discussion of the general grounds of prognosis in the several cachexiæ which have been enumerated (p. 887), as the cause of tumor, it may be well to direct attention to a few points with regard to some of them.

If tubercle is believed to exist, the prognosis is highly unfavorable; but the course of tubercle *en masse* in the brain, is sometimes exceedingly slow, and this is the case especially in children. The advance towards a fatal issue is to be apprehended when there is marked hectic, much elevation of temperature, and when the symptoms indicate the progressive invasion of different portions of the brain. On the other hand, when—although there may be distinct paralysis, or amaurosis—the general health is tolerably good, and the symptoms have shown but little tendency to increase in either intensity or extent, there may be considerable prolongation of life.

The hypertrophy of the brain which is met with in children is slowly progressive, but its prognosis, under all rates of advance, is eminently unfavorable.

In syphilitic diseases of the brain or its meninges there is much room for hope; and it seems to be of little moment that the symptoms are varied and severe. Those which are the least amenable to treatment are the losses of sight and hearing, which not unfrequently exist; paralyses and spasmodic affections are often removed with considerable rapidity. The length of time during which the symptoms have lasted is a further guide in the prognosis, the hope of restoration being in inverse proportion to the duration of the morbid state. Still, unless the general condition be one of highly marked cachexia, amendment may be confidently expected. The presence of disease in the kidneys is of unfavorable omen, but even it often disappears under an antisyphilitic treatment. There are no cases which appear so bad and which recover so well as some examples of intracranial syphilis. Until the diagnosis of the constitutional state is established, the case may appear absolutely hopeless; sometimes the only missing link in the history may be unattainable because the patient is insensible, or in such a state of mental incapacity that no reliance can be placed on his assertions, but yet from such condition he may completely recover.

In the case of lead-poisoning the prognosis is favorable, provided that after a few applications of either the continuous current, or of faradization, the muscles show some remnant of irritability. When the paralysis has existed for a number of years, and the wasting of muscular tissue is very great, it may be impossible to restore the limb, but yet, by continuous treatment, the advance of symptoms may be arrested.

In those cases where there is reason to suspect the existence of either aneurism, hydatids, or carcinoma, the prognosis is eminently unfavorable; but the forecast of a fatal termination is to be based upon the state of the general health of the patient, rather than upon the special cerebral symptoms.

Under all conditions of Adventitious Product uncontrollable pain and vomiting are the most unfavorable symptoms; the former deprives the patient of rest, and the latter renders food useless, and often worse than useless, through the fatigue occasioned by its rejection.

TREATMENT.—There is nothing special which can be said with regard to the treatment of adventitious products in the head, for under all circumstances it is simply that of the different dyscrasiæ upon which they depend. The only remark which it seems to me desirable to make, is one in favor of the administration of large doses of iodide of potassium when there is a belief in the existence of syphilis. I have repeatedly seen the most menacing symptoms removed by the exhibition of KI in doses of forty grains, three and four times daily. When this has failed, recourse to mercury has proved curative, and with especial frequency when in conjunction with the baths and waters of Aix-la-Chapelle. Small doses varying from $\frac{1}{24}$ to $\frac{1}{12}$ of a grain of the red iodide of mercury have appeared to me to be more generally useful than any other form of mercury; but when even those doses

[1] Précis d'Anat. Patholog., t. iii. 776.

cannot be borne by the stomach—an event which rarely happens—happy results may follow the exhibition of mercury by fumigation or inunction.

The pain of cerebral tumor may be palliated, and sometimes removed altogether by Indian hemp, or the application of ice; and sleep may be obtained by the chloral hydrate.

Sickness is sometimes treated most successfully by absolute rest to the stomach, the patient being fed by nutritive enemata.

Convulsions may be checked by bromide of potassium, in doses of ten or twenty grains; but the powers of the therapeutic art are, with the exceptions above mentioned, inclosed within painfully narrow limits, and all that can be done is to palliate evils which cannot be removed.

CEREBRAL HEMORRHAGE AND APOPLEXY.

By J. Hughlings Jackson, M.D., F.R.C.P.

The text[1] of this article is Cerebral Hemorrhage, using the term in the sense of escape of blood within the intracranial nervous centres. Since very little or very much blood may be effused, the symptoms vary extremely in degree; and since the parts in which rupture of vessels may take place are numerous, the symptoms vary much in kind. When large and rapid effusion occurs, there is the apoplectic condition. Cerebral Hemorrhage is one cause, and the most frequent cause of apoplexy. So frequently does hemorrhage cause apoplexy, that the name has got into use for hemorrhages in other organs. Thus we speak of Retinal Apoplexy and of Pulmonary Apoplexy. Since this use or abuse of the word leads to confusion, the term is often qualified by the word Cerebral. We shall restrict Cerebral Hemorrhage to effusion of blood into the brain, and reserve the word Apoplexy for the comatose condition which large effusion of blood and other causes produce. Cerebral Hemorrhage will be chiefly considered, and other causes of Apoplexy will be spoken of under the head of diagnosis. It will be absolutely necessary to make occasional reference to meningeal hemorrhage, although this has been considered in another part of this volume, since meningeal as well as Cerebral Hemorrhage may produce Apoplexy.

[1] In my collection of materials towards writing this paper, I have to acknowledge with my warmest thanks the help I have received from Dr. Anthony Roberts, Mr. F. M. Corner, Mr. Steggal, Dr. James Jackson, Mr. George Mackenzie, Mr. Frederick Mackenzie, Mr. Grubb, Mr. Norton, Mr. Llewellyn, Mr. Gordon Brown, Mr. Stephen Mackenzie, Mr. G. E. Herman, Mr. Louis Mackenzie, and others. I have to acknowledge also most valuable assistance in the investigation of cases of apoplexy from my colleagues Dr. Sutton, Dr. Woodman, Mr. Hutchinson, Mr. Waren Tay, and Mr. MacCarthy. I have to thank my other colleagues for their generosity in placing cases at my disposal for investigation. For valuable help in the revisal of this article, I am indebted to my friends Dr. Woodman, Dr. Gowers, and Mr. Stephen Mackenzie.

Morbid Anatomy.

Position.—The effusion of blood is circumscribed. With rare exceptions it occurs in but one side of the brain, nearly always in a limited part of that side, most frequently in the ganglia at the base. Occasionally, however, it breaks out of the substance of the brain, usually into the lateral ventricle, but sometimes on to the surface. It is of little use to take as a basis for statistics, records of published cases; from the Pathological Society's Transactions, for instance. No one would think of bringing before the Pathological Society an ordinary specimen of Cerebral Hemorrhage. Cases of special interest are published, such for instance as cases of hemorrhage into the pons Varolii simulating opium poisoning. A goodly number of cases of large hemorrhage into the cerebellum could be collected from medical periodicals, as many such cases are pretty sure to get into print. But large hemorrhage into the cerebellum is in reality exceedingly rare. During the seven years I have been attached to the London Hospital, I have seen but two cases. Again, statistics take count mostly of *fatal* cases of hemorrhage—of effusions of blood into the brain big enough to kill quickly—and of comparatively few of those cases so frequently seen in hospitals

of hemiplegic patients who have recovered from coma, and who go out of the hospital with more or less permanent palsy. Many of these patients die months or years after in workhouses. If we speak of hospital cases only, we thus exclude many cases of very great clinical importance, and besides those mentioned we exclude cases of rapid death from meningeal hemorrhage, for most of such cases are seen by those engaged in private practice. For these reasons I do not give nor refer to statistics, but it will, I think, be safe to say that *large* hemorrhage very often occurs in the corpus striatum and thalamus, often in the pons, rarely in the cerebrum, very rarely indeed in the cerebellum, and scarcely ever in the spinal cord.

We occasionally find blood in the lateral ventricles. In the vast majority of cases, it comes from a rent in the corpus striatum or thalamus opticus. It may extend to the fourth ventricle, and escape sometimes on the surface of the brain. I have, however, twice known blood effused into the ventricle without injury of the ganglia in its floor. These were exceptional cases; the blood came from bursting of large aneurisms. In one the aneurism was seated in the middle line of the hinder part of the circle of Willis at the divergence of the posterior cerebral arteries. In the other case an aneurism of a small artery of the posterior lobe had burst into the posterior cornu—blood had escaped also on to the surface of the brain. In the first edition of the article Convulsion, a case occurring in Mr. Gayton's practice was mentioned, the notes of which were supplied by Dr. Woodman. In this case Dr. Woodman found no aneurism, and no laceration of the brain.[1]

Multiple Effusions.—Sometimes two or more recent clots, even large clots, are found in different parts of the brain. My friend Mr. Llewellyn showed me a specimen in which there was a clot in the floor of the lateral ventricle, and another in the pons. The two clots came on at the same time, for the patient had gone to bed as well as usual and died next day of Apoplexy, which began in the night. Dr. John W. Ogle (Pathological Soc. Transactions, vol. xv. p. 8) has recorded a case in which there were three recent clots, one in the right corpus striatum, one in the left thalamus opticus, and one in the pons Varolii. Dr. Baümler has supplied me with notes of the case of a man, thirty-four years of age, who died three hours after a fit, in whose brain four recent clots were found: a large one in the centre of the right hemisphere, a small one in each of the optic thalami, and a small one in the right crus cerebri. The ventricle was also full of blood. After discovering the main clot, we should carefully search the rest of the brain, especially the pons and medulla, for small effusions, often only little specks.

So far we have spoken of recent clots. It is not at all rare to find effusions of blood of very different dates. Sometimes after discovering a recent clot on one side of the brain we may find much of the opposite motor tract damaged by old effusion when there has been no corresponding palsy for some time before death.

Size of Clots.—The size of a clot varies from that of a pea or less, to a mass the size of one's fist or more. Its size will depend of course on the size of the vessel ruptured, and its shape depends somewhat upon this also. In small capillary hemorrhages the blood may lie in streaks in the brain substance, rather pressing the tissues apart than destroying them. (We see by the ophthalmoscope in many cases of small hemorrhages into the retinæ, in cases of Bright's disease, that the blood is arranged in the direction of the retinal nerve fibres.) It is doubtful whether capillary hemorrhages in the brain give rise to symptoms. (A patient's sight may be quite good when there is considerable streaking of his retinæ with blood.) When from a large effusion the fibres are torn, the clot is rounder and more distinct, and gives rise to symptoms.

The larger the clot, not only the greater the local destruction of nerve tissue, but also the more squeezing there is of the parts not directly damaged. When a large quantity of blood has been effused in one side of the brain, we see *post mortem* that the affected cerebral hemisphere looks more voluminous, that the convolutions are flattened, and we find on section that the cerebral substance is more anæmic than on the healthy side. If after sawing round the skull we insert the knife in the line of the saw-cut, and remove the skull cap with the part of the brain it contains—a plan Mr. Hutchinson adopts to display the position, and effects of traumatic hemorrhage—we may find that the falx bulges to the sound side, and this is evidence that the other hemisphere has been compressed. These facts are of importance with regard to the causation of loss of consciousness from Cerebral Hemorrhage.

Changes in Effused Blood.—We find

[1] A man, 24 years of age, died rather suddenly twelve hours after a fit of convulsion. Although this man had recovered so as to take broth and to answer questions, his cerebral ventricles were found at the autopsy full of blood. Mr. Prescott Hewett says that a very, very slight laceration of the floor of the lateral ventricle may, if it correspond to the situation of a large vein, give rise to an extensive extravasation of blood into this cavity (Holmes' Surgery, p. 314). Blood in the fourth ventricle may come from a rent in the pons.

post mortem most varied appearances according to the age of the clot. Soon after its effusion there is seen a soft black jelly mixed at its edges with small specks of brain and lying in a bed of softened brain. In the later stages the appearances are widely different: there is a cyst—"apoplectic cyst"—filled with ochre-yellow fluid, or there is even a cicatrix. We sometimes find the two extremes, a recent black soft clot, and one or more cysts the relics of old effusions. In the progress from the recent clot to the apoplectic cyst, we have to consider changes in the blood effused and changes in the brain about it.

On removing as much as we can of a recent clot we mostly see, especially when the effusion is in the corpus striatum and thalamus, an irregularly-shaped cavity with a shaggy wall of soft brain intermixed for a short distance with specks of blood. The local softening results partly from imbibition of serum from the clot and partly from inflammatory changes excited by the clot. The inflammation—local encephalitis—may lead to extensive disintegration of brain ("the apoplectic clot is even transformed to an abscess of the brain," Niemeyer), but usually the process is limited; it is conservative, and leads to the formation of the cyst wall to be presently mentioned.

The progress to the final stage is gradual. The clot diminishes in bulk, becomes softer and browner; next the color becomes yellow; granules of blood pigment and hæmatoidin crystals form. Finally the clot is represented by thin ochre-yellow, or even clear fluid. Simultaneously, as a result of a slow inflammatory change, the wall of the cavity undergoes great alteration. An organized membrane forms from the neuroglia, and the apoplectic *cyst* results. This is the most common termination, but the process may go even further. There may be no cyst, but a hard pigmented patch, an "apoplectic cicatrix."

Traumatic hemorrhage.—In cases of injury the clot mostly affects the surface, and most frequently the convolutions of the base; there is rather a pulp of brain and blood than a distinct clot. An injury is to be suspected whenever blood is found effused close to the convolutions, especially if these be bruised, and if there are many small specks of black blood near the principal clot; above all, if distant from the principal lesion, there are very many little specks of black blood intermixed with, and round about pulpy patches.

At post-mortem examinations, especially of those who have died of chronic Bright's disease, we occasionally find evidence of former hemorrhage into the gray matter of the convolutions. There is often a cup-like depression with hardened walls containing ochre-yellow fluid. But if these be in several parts which we know to be often bruised by injuries to the head,—for instance, on the under surface of the anterior or sphenoidal lobes,—it is probable that there has been injury to the head. (See Bristowe, Path. Soc. Trans. 1869–70.)

Rupture of Aneurisms of large Cerebral Arteries.—We have to speak of aneurisms of the small arteries of the brain (miliary aneurisms, p. 894), but occasionally Cerebral Hemorrhage results from rupture of aneurism of the large arteries at the base. As a rule, however, their rupture produces meningeal hemorrhage. (See arts. Adventitious Products, Meningeal Hemorrhage, and Convulsions.)

Hemorrhage from Cerebral Tumors.—Cerebral Hemorrhage has occasionally, but very rarely, its origin from vascular tumors of the brain. I have recorded three such cases in the *Lancet*, Oct. 29, 1869. The tumor is the glioma of Virchow. As he points out, ordinary Cerebral Hemorrhage has its seats of election, and these are not precisely the places where glioma is most frequently found. Glioma occurs most frequently in the white mass of the hemisphere, especially in the posterior and anterior lobes, places where ordinary Cerebral Hemorrhage is rare. It is very important to bear in mind a remark he makes to the effect that it often requires a very attentive examination to distinguish hemorrhagic gliomata from traumatic hemorrhage, from red softening, and from rupture of cerebral aneurism. (See art. Adventitious Products and art. Softening.)

Lungs.—Of course all organs are to be examined *post mortem* for, as we shall see, the heart and kidneys are often diseased. There are, however, often striking post-mortem appearances in the lungs, which are owing to the apoplectic condition. These are not peculiar to apoplexy from Cerebral Hemorrhage, and, indeed, some of them at least occur in other modes of dying. They are very varied, and the variation depends on two factors—the rapidity of death and the condition of the patient (his age and state of health) when taken ill. I have not been able to make out any difference from *position* of the Cerebral Hemorrhage. I have known the lungs pale like "cholera lungs," and weighing only twenty-two ounces, in a patient who died in an hour of large Cerebral Hemorrhage. But as a rule even in patients who die more quickly—as in some cases of meningeal hemorrhage—the lungs are congested. They are often bulky and œdematous in their dependent parts, which easily break under pressure, and very emphysematous (vicariously) in front. If the patient be a robust, full-blooded man, the lungs may be black, from cramming with blood up to their anterior mar-

gins, and easily breaking into a pulp, as in cases of rapid death from traumatic hemorrhage. In some cases of slower death, we find pulmonary apoplexies. I have in two cases (one traumatic and one opium-poisoning; both patients young) seen lungs which on section looked like the "damson lungs" of heart disease. In other cases where patients die very slowly, we find on section granite-colored patches of various sizes slightly raised and well-margined. These lobular patches are often called lobular pneumonia, and when very numerous and almost confluent, the word pneumonia is sometimes used without any qualification. Yet these changes occur with rare exceptions, in both lungs and in the dependent parts of all lobes, and ought not to take the name which belongs to a well-marked independent disease. They may affect one lung more than the other — the right usually. Since, when we find these granite-colored patches, we occasionally find recent apoplexies also, and indeed patches of intermediate color, I believe they are, as Brown-Séquard has stated, altered apoplexies. Sometimes one or more of them are broken down into a grumous pulp; over some nodules near the surface there may be slight pleurisy.

Etiology and Pathology.

In speaking of Etiology and Pathology we for the present exclude cases where the bleeding is the result of injury to the head, where it occurs from rupture of an aneurism of a *large* cerebral artery, such as the middle cerebral or basilar, or where it starts from a vascular tumor of the brain. We cannot speak at all of cases of intracranial hemorrhage occurring in purpura (see art. Purpura, Part I. p. 461), or scorbutus (see art. Scorbutus, Part I. p. 456), or pyæmia (see art. Pyæmia, Part I. p. 335), nor of "red softening" (art. Softening). Obviously the above are in their etiology and pathology very different things; they only agree in that there is escape of blood in or upon the brain. Moreover they differ much clinically. We consider, in what follows, the common run of cases.

Age.—Cerebral Hemorrhage rarely occurs in persons under forty. This age is that at which one of the most important factors in the causation of Cerebral Hemorrhage, degeneration of arteries, begins to be common. Changes in the arteries of old men are scarcely to be considered morbid. "To degenerate and die is as normal as to be developed and live" (Paget). In the progress to healthy old age the body, *as a whole*, descends in vitality; the blood wastes, numerous capillaries obliterate, the lymphatic system undergoes involution, there is senile emphysema. Although the heart becomes somewhat bigger as years increase, there is not excessive cardiac hypertrophy. There is an increase in bulk of the whole organ, not an extreme hypertrophy of the left ventricle only, as there so often is in persons of middle age who die of Cerebral Hemorrhage. The degenerations we are especially concerned with in this article are premature, moreover they are often attended by disease of the kidneys. In a patient under forty the arteries may feel tougher than the arteries of another person of eighty.

Some of the exceptional cases of large Cerebral Hemorrhage in *young* people whose arterial system, as a whole, has not undergone degenerative changes, are cases of rupture of aneurisms of the larger cerebral vessels; for instance, of a branch of the middle cerebral or of the basilar. Indeed, if there be no evidence of the arterial and other degenerations in the body, to be presently mentioned, the probability is that large Cerebral Hemorrhage in young people (excluding cases of injury, purpura, and the like) is thus caused. Apart from such quasi-accidental cases, we must observe further that the matter of importance in considering the influence of age in diagnosis is to note the general constitutional state the patient has arrived at rather than the number of years he has lived. For Cerebral Hemorrhage quite like that which occurs so often after forty does sometimes occur at the age of twenty and even under, in people who are subjects of chronic Bright's disease, who have degenerated arteries and hypertrophy of the left ventricle of the heart. I have recorded such a case (London Hospital Reports, vol. iv. p. 337)[1].

Heredity.—It is asserted that in some families there is a tendency to Cerebral Hemorrhage. Obviously the transmission cannot be of a tendency to certain symptoms—hemiplegia and Apoplexy—but to certain tissue degenerations most strikingly manifested in the arteries. Cerebral Hemorrhage therefore can be inherited only indirectly. Much used to be said of a certain inherited build of body and of the "apoplectic constitution." Little importance is nowadays attached to this. Austin Flint says, "The larger number of persons attacked are either spare or of an ordinary build." Niemeyer says,

[1] In one case, that of a woman aged 73, who died in forty-seven hours, of hemorrhage into the substance of the left cerebral lobe; one lung, which was universally adherent, was solid by this sort of change; the other, which was not adherent, except by one or two tags, was a good specimen of senile emphysema, and presented scarcely any other morbid appearance.

"There is no such thing as an apoplectic constitution indicated by a short neck and broad shoulders."

CONSTITUTIONAL STATE PRIOR TO CEREBRAL HEMORRHAGE.—It is convenient to consider an extreme case in order that we may state the whole of the factors which *may be* concerned in causing Cerebral Hemorrhage. We are, as was said at page 905, not now considering cases of hemorrhage from rupture of aneurisms of the large cerebral arteries, from injuries, or from the like quasi-accidental causes.

At autopsies on patients who have died of Cerebral Hemorrhage we frequently, if not mostly, find three things: hypertrophy of the heart, chiefly the left ventricle, chronic renal disease, and degenerated[1] arteries (Bright, Johnson, Kirkes). A patient so much and so widely diseased has not a liability to Cerebral Hemorrhage only; he is liable to inflammation of serous membranes, to bronchial catarrh, to œdema of the lungs, &c. But in this article we have only to do with the triple association as it bears on Cerebral Hemorrhage. We are especially concerned with the condition of the vascular system, and can only speak incidentally of the renal disease. We have two tasks. Going the natural round of the circulation (heart, large arteries, small arteries, capillaries (and nervous tissue), venous system and lungs), we have first to consider the abnormal conditions of different parts of the vascular system, and next how, from the sums of these several conditions, it results that rupture of the smaller cerebral arteries is determined.

Heart.—The hypertrophy is of the left ventricle: it is of the kind called simple, because, although the wall of the ventricle is thickened, there is not dilatation; the capacity of the ventricle is, at all events, but slightly increased. Yet, as in other kinds of cardiac hypertrophy, dilatation may ensue. In patients long bedridden from palsy due to Cerebral Hemorrhage, we may find the heart decreased in size, notwithstanding that there is chronic renal disease.

In this form of hypertrophy there is obviously increased power acting on the *arterial* side of the circulation, and consequently we infer obstruction somewhere in the arterial system. We limit ourselves to cases where there is no obstruction at the aortic orifice, and no incompetence of the aortic valves.[2] The resistance to the heart is much further on. It is peripheral. Although this is agreed on, there are great differences of opinion as to the exact nature of the peripheral obstruction. Dr. Bright suggested that the "altered quality of the blood might so affect the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system." He suggested also that the blood in Bright's disease might act as an unwonted stimulus to the heart. Dr. George Johnson, believing that the blood in renal disease is more or less noxious to the tissues since it contains "urinary excreta," considers that its passage into the capillaries is resisted by *contraction of the small arteries*—the vessels most rich in muscular tissue. The muscular coats of these vessels therefore are hypertrophied in *antagonism* to the heart. Since the small arteries are hypertrophied throughout the body, the obstructions, though each is slight, are in their sum total so large, that in order that the circulation may be carried on efficiently, hypertrophy of the heart must ensue.

But whilst Dr. Johnson believes that the thickening of the walls of the small arteries is genuine hypertrophy, "an increased growth of a normal tissue without change of texture," Dr. Beale doubts whether there is real hypertrophy of the muscular fibre cells, and supposes that the changes in the small arteries are degenerative. He remarks[1] that "there is an increased bulk with altered structure, not simply increased bulk without change of structure (hypertrophy)."

Traube considers that the cardiac hypertrophy in Bright's disease is a consequence of increased tension of the arterial circulation, partly the result of the diminished calibre of the renal circulation, and partly the result of greater volume of the blood from diminished excretion of fluid. The objection which has been raised to this view is, that the hypertrophy of the heart mostly begins in the earlier stages of Bright's disease.

Occasionally, although rarely, we find great hypertrophy of the left ventricle in cases of Cerebral Hemorrhage, when there is neither obstruction at the aortic orifice,

[1] Retinal changes will be alluded to hereafter. The careful clinical observer considers minor degenerative changes,—baldness, grayness, state of skin, worn teeth. He inquires for history of gout, and of intemperance.

[2] For intracranial hemorrhage is not common in cases of *valvular* disease of the heart, excluding cases of aneurisms of the larger cerebral arteries, and ruptures of these usually produce meningeal hemorrhage. My observations confirm the statement of Dr. Austin Flint, that important nervous symptoms of any kind—excluding, of course, those produced by the process of embolism—are not common in cases of valvular diseases of the heart (Diseases of the Heart, 2d ed., p. 180).

[1] Beale on the Urine, 3d ed., p. 72.

nor renal disease, but simply wide-spread degeneration of the arteries. From these cases it seems clear that degeneration of vessels is a sufficient cause of hypertrophy of the left ventricle. It must be admitted that there are cases of hypertrophy of the heart, which we are unable to explain. We do mostly, however, find hypertrophy of the left ventricle in cases of Cerebral Hemorrhage.

Large Arteries.—From a degenerative[1] change the large arteries lose much of their chief property—elasticity; they become permanently wider, longer, and more tortuous. We see them move in curves on the temples, and we feel that they are tough and sometimes even "bony." When the large arteries which we can see and feel are thus changed, it is a reasonable inference, that the large arteries of the brain are similarly, although not perhaps equally, altered. Elasticity of the arteries, although it adds no new force, is an important aid to the circulation in equalizing the flow of the blood, gradually reducing it from intermittence in the large arteries to a nearly continuous stream in the small arteries. We readily understand, therefore, that the *absence* of elasticity of the larger arteries will be an important factor in leading to rupture of the smaller arteries. The circulation is carried on too much in systole. The smaller arteries will receive the impulse from the strong left ventricle intermittently, not remittently.

Small Arteries.—Here rupture mostly takes place, always in large hemorrhages. We have to consider several pathological conditions of the smaller arteries. (*a*) Fatty degeneration. Arteries of small size have a highly developed muscular coat, and this coat especially is the seat of fatty degeneration, a change which we may suppose will allow rupture when the vessels are unduly strained. However, not so much importance is attached to this pathological condition as was wont to be, for fatty degeneration is found sometimes in the arteries of very young people. Indeed, Billroth and Bouchard consider it to be most frequently a *result* rather than a cause of cerebral lesions. But even if so, degeneration of arteries, for instance, in a focus as softening, may favor the occurrence of hemorrhage into the part diseased. (*b*) Charcot, Bouchard, and Charlton Bastian have described what they term "miliary aneurisms:"[1] aneurisms mostly visible to the naked eye, of the size of a pin's head more or less. (These have been fully described, art. Adventitious Products.)

Capillaries.—These vessels may be found the seats of fatty degeneration. But ruptures allowing large hemorrhages do not occur here. If it be degeneration of the smaller arteries which produces obstruction and thus induces hypertrophy of the left ventricle of the heart, the diseased state of the smaller arteries may protect the capillaries from strain.[2]

[1] I speak of *results* under the general term "degeneration." Virchow (Cellular Pathology, Dr. Chance's translation) points out that the true atheromatous change in arteries begins by a slow inflammatory change of the tunica intima—an endarteritis strictly analogous to the inflammatory changes in endocarditis. I have in this article, however, to do with changes in the vessels so far only as, by affecting the dynamics of the circulation, they favor cerebral hemorrhage. I have not to do with the processes by which these changes are arrived it; therefore I use the general term "degenerative," which, with the above qualifications, need not mislead. I conclude the foot-note by a quotation from an able lecture by Moxon, Med. Times and Gazette, Nov. 12, 1870: "It is too much the fashion, at least in this country, to assume that all the processes in the arteries which lead to the deformation of their interior by yellow patches, swellings, petrifactions, or erosions, or to aneurisms or rupture of the vessels, are all of a degenerative origin, and that all are sufficiently described and defined in the common notions of atheroma. The truth is, that sub-inflammatory irritation plays a very important part in these changes." Since this was written, Moxon has considered the whole subject in a valuable article in Guy's Hospital Reports, 1870–71.

[1] Dr. Bristowe has drawn attention (Path. Soc. Trans. 1859) to ruptures of *small* aneurisms in the substance of nervous organs as a cause of cerebral hemorrhage, and gives a drawing of an unruptured aneurism, the size of a grain of wheat, which lay in the substance of the cerebellum. Dr. Henri Lionville has found miliary aneurisms in the retina (Gaz. des Hôpitaux, 1870). See also Dr. Gull (Guy's Hosp. Reports, 3d Series, vol. v. 1859).

[2] Here reference may again be made to Dr. George Johnson's view, mentioned p. 906. In one case of large Cerebral Hemorrhage he failed to discover any hypertrophy of the small arteries of the brain, but he found them much hypertrophied in the subcutaneous tissue. Inferentially, they were hypertrophied in other organs, although, unfortunately, examinations were made of the arteries for the brain and of the subcutaneous tissue only. In such a case, he suggests that the hemorrhage results, not from rupture of the arteries, but from rupture of the capillaries of the brain. They rupture because they are not protected from the force of the hypertrophied left ventricle by the hypertrophy of the muscular coat of the arteries delivering blood to them. It is, however, unlikely that rupture of capillaries would cause a large effusion of blood. "The apoplectic fit does not occur in capillary hemorrhages." (Niemeyer, Textbook of Practical Medicine; translation of the eighth German edition by Drs. Humphreys and Hackley, vol. ii. p. 198.)

Nervous Tissues.—The influence which changes in the tissues outside the vessels may exert in the causation of Cerebral Hemorrhage may be most conveniently considered here.

When the brain wastes slowly, there is, in order that the cranium may continue full, compensation. There is, in some cases of wasting of the brain, thickening, genuine hypertrophy, of the skull (Paget). There is, sometimes, increase backward of the capacity of the frontal sinus (Holden); but the most important compensation is by increase in the quantity of the cerebro-spinal fluid. To these sources of compensation Leubuscher and Niemeyer add dilatation of the vessels which will, they believe, favor rupture. And Niemeyer suggests that the frequency of Cerebral Hemorrhage, in advanced life, depends at least partly on the dilatation of the vessels induced by atrophy of the brain. Further, he thinks that since Cerebral Hemorrhage leads secondarily to atrophy of the brain, one attack favors the occurrence of another attack.

Softening of the brain, by diminishing the support of vessels, may favor hemorrhage, but it is almost universally agreed on that the softening we find *post mortem* near to a clot is nearly always the result of the effusion, not a pathological condition prior to the occurrence of hemorrhage.

Venous System.—We shall speak only of obstruction to the return of the blood from the brain by changes in the lungs, excluding cases like phthisis and senile emphysema, in which the volume of the blood is reduced, and in which, therefore, the pulmonary impediment is to a great extent compensated. From hypertrophous emphysema there results universal peripheral congestion, consequent on the difficulty the venous blood encounters in passing through the lungs—the vascular area of which is reduced by obliteration of a great number of capillaries.[1] From theoretical considerations therefore we might at first glance attach much importance to emphysema as a factor in the causation of Cerebral Hemorrhage. "In no other disease does the cyanosis attain such severity excepting in cases of disorder of the orifices of the right heart." (Niemeyer.) However, emphysema probably is not often an important factor in the causation of Cerebral Hemorrhage. There will, in chronic cases, be much compensation by increased power of the right ventricle. Even if there be not full compensation, the left ventricle will have less blood to send to the brain, and thus the *arterial* tension will be diminished. It is the venous system which is overblooded and strained in emphysema; the arterial, from which rupture in Cerebral Hemorrhage takes place, is underblooded and less strained. In the last stages of emphysema we have, Niemeyer says, the severest symptoms of hyperæmia of the brain. Yet Cerebral Hemorrhage is a rare termination of this or of any other form of thoracic disease. It is the tension on the venous side of the circulation which is increased in emphysema.[1]

The veins are of lower functional structure than the arteries—very few of the cerebral veins contain any muscular tissue—and thus probably they are less liable to disease. The degree of hyperæmia of the face and lips is no exact measure of the degree of venous congestion of the brain in sudden obstruction at least. The cerebral veins are protected from sudden backward strain by the large cerebral sinuses. Nevertheless obstruction at the lungs, especially when occurring quickly, will be a factor, if not an important one, in favoring rupture of cerebral arteries.

We have now to speak of the dynamics of such a person's circulation, in order to show how abnormal strains lead to rupture. To obtain clear ideas upon this point, it is well to run over the peculiarities of the cerebral circulation in health. The brain receives a large supply of blood (a large quantity passes through it), but there are provisions by which it is protected from suddenly increased afflux. There are turns of the carotid and vertebral arteries, the free anastomosis of the circle of Willis, the numerous subdivisions of the arteries beyond that circle, on the convolutions at least, and their small size before they enter the brain substance itself. Perhaps we may add the possibility of diverticular enlargement of the thyroid body, and of the facial arteries, by which in suddenly increased action of the heart the flow to the brain will not be in proportion to the increased quantity of blood sent into the carotid and vertebrals. Further, when there is increased afflux of blood, compensation can occur by diminution of the quantity of the intracranial part of the cerebro-spinal fluid. Some parts of the brain, however, are less pro-

[1] We do not here refer to the acute emphysema which we so often find *post mortem*, and which is especially well-marked in young, robust men who die in the apoplectic condition soon after traumatic intracranial hemorrhage. In these cases we find great posterior congestion—occasionally pulmonary apoplexies also—and vicarious emphysema of the front parts of the lungs.

[1] Dr. Hyde Salter never saw or heard of Cerebral Hemorrhage during an attack of asthma or as a result of asthma; for, although here there is acute obstruction to the return of venous blood, the arterial tension is very small: in severe attacks the pulse can scarcely be felt.

tected than others. The arteries which supply the corpus striatum are not capillary in size. They, or many of them, come off from the middle cerebral, which artery is almost the continuation of the internal carotid. Thus its branches lie more in the way of strain from the heart. But on the other hand we have to observe that here there is special compensation. As Hilton points out, the corpus striatum and thalamus (which parts we may presume to have greater and more frequent functional activity than most divisions of the nervous system) lie in great part in the large water-bed of the brain. By this means rapid compensation by diminution of the fluid in the lateral ventricle may occur when these highly vascular parts during their functional exercise swell by becoming for a while more vascular. There is even more. Dr. Bastian (on Tubercular Meningitis, *Edin. Med. Journal*, April, 1867), speaking of perivascular canals, suggests (but only as one hypothesis as to their use) that they may constitute "an apparatus for the distribution of cerebro-spinal fluid throughout the structure of the brain, in order that the same protective influence may be exercised over each individual portion of its structure which is exercised over the whole region by that portion of the fluid situated in the subarachnoid spaces." He says too, speaking of perivascular canals, three times the diameter of the vessels they contain,—"This large size of the perivascular sheaths occurs more frequently in the corpus striatum and optic thalamus." Then on the venous side there is the remarkable arrangement of the sinuses, which is such that a backward strain, if it be sudden, will not reach the veins of the brain — or will reach them in a diminished degree. The blood will pass, by preference, so to speak, into the outer parts of the head, face, &c. If the backward pressure be slowly exerted as in chronic emphysema, there will be cerebral hyperæmia, but it will be very slowly developed and very evenly distributed.

Let us suppose that all the abnormal conditions enumerated, p. 906 to p. 908, are present. Of course the whole of these factors are not present in all cases. There may be no renal disease, but there are usually degenerated arteries and hypertrophy of the left ventricle. But as the following is chiefly recapitulatory, we shall in its proper order mention each of the conditions which different observers have supposed as well as have proved to be factors in the causation of Cerebral Hemorrhage. The small artery is usually the seat of rupture. We take this as the fixed point, and first consider the development of undue strain upon it. Next we speak of negative circumstances which add to the influence of this strain—of impediments to the exit of blood from the arteries into the veins.

There is a strong left ventricle. The larger arteries are inelastic, and thus the wave of the blood sent forcibly into them is not equalized: as a consequence the impulse from the heart's jerks will be carried on strongly to the smaller arteries of the brain. The small arteries of the brain—normally thinner than arteries of other parts—are degenerated. Though this degeneration leads to resistance, it is not the resistance of power; it is a "weak obstinacy." The resisting arteries are fragile and may be actually aneurismal. Moreover if the brain be wasted, the arteries are dilated and less supported. So far for the forward strains.

If we hold that changes of nutrition in the penetralia of the body—in capillary regions—contribute largely to the forces of the circulation (*vis à fronte*), there will be from the imperfect nutrition which the disease of the arteries and capillaries causes (and which in some cases atrophy of the brain signifies), an obstruction (or more precisely a cessation of help) to the flow of blood. The capillaries will not readily empty into the veins. At this point too, as there is often renal disease, we have to recognize, if we follow Bright, Johnson, and Kirkes, a still further element of obstruction in the unwillingness of the tissues to pass impure arterial blood. Further, if there be emphysema or other obstruction at the lungs, the free return of blood from the brain is hindered. Perhaps a condition of plethora, or at least of transient plethora after large eating and drinking, may add to the tension of the circulation by increasing the volume of the blood. If all the above-named conditions be present, there is no wonder that the diseased small arteries, unable to empty readily into the capillaries and veins on the one hand, and jerked by a strong ventricle on the other, sometimes give way. Moreover, when we consider the local peculiarities of the circulation of the corpus striatum and thalamus, we can well understand that these bodies are the "seats of election" for large Cerebral Hemorrhage, notwithstanding the special provisions mentioned.

The degenerations have been slowly going on, and the diseased vessels are being subjected to increasing strain. A time comes when a vessel more diseased than another, perhaps one the seat of a miliary aneurism, or some vessels specially in the way of strain, mostly a branch to the corpus striatum, gives way. It is not at all rare to hear it said that hemorrhage is sometimes the result of *obstruction* to the *return* of venous blood from the head in paroxysms of convulsion. Dr. Todd (Diseases of the Nervous System,

Lects. vii. and xii.) held this view. But since the hemorrhage is nearly always in the arterial regions of the brain, and since in the most severe paroxysms of chronic epilepsy, Cerebral Hemorrhage is excessively rare, it is far more reasonable to suppose that the irruption of blood itself causes the convulsion and the subsequent apoplectic condition. No single instance of actual Cerebral Hemorrhage in an epileptic fit has presented itself in Reynolds' experience (On Epilepsy, p. 225).

Hemorrhage from Aneurism of the larger Cerebral Vessels.—In cases of aneurism of the larger cerebral arteries, a very local disease of the vessel may be the sole flaw in the system, excepting perhaps vegetations on the heart's valves (Dr. John W. Ogle and Dr. Church). If there be no misfitting of the valves, no hypertrophy of the heart will ensue, nor indeed any derangement of the dynamics of the circulation of the brain, possibly not even any change in the nutrition of the part supplied by the aneurismal artery if it remains pervious. In a few cases the fatal attack begins, if not in good health, at least in what appears very like good health. The patients feel well and are about at their work. We can say nothing as to their constitutional condition. I have only to do with rupture of these aneurisms in diagnosis. For rupture of them mostly produces meningeal, not cerebral hemorrhage. (See art. Adventitious Products.)

Localization of Lesions.

We have now to speak of lesions of the motor and sensory tract within the cranium, and of lesions of the two large masses therewith connected—the cerebrum and cerebellum. In this section we consider almost solely paralytic symptoms —those which localize; other non-localizing symptoms will be considered under the head of the Apoplectic Condition.

The parts of the motor and sensory tract from above downwards are corpus striatum, thalamus opticus, crus cerebri, pons Varolii, and medulla oblongata. All these parts are double, right and left, although the halves of the last two, pons and medulla, are welded together. In the vast majority of cases, the lesion is of but one *lateral* half of the sensori-motor tract, and its results are one-sided palsy—hemiplegia. In the parts we have spoken of as being welded together, the pons and medulla, the lesion occasionally affects both halves, and thus paralyzes both sides of the body. We have to do especially with lesions of one lateral half of the motor and sensory tract at different levels, and consequently with several varieties of hemiplegia. In all these varieties we are concerned with palsies of the limbs, and with palsies of parts supplied by cranial nerves.

The limbs, since their motor fibres have decussated in the lower part of the medulla oblongata, are always palsied on the side opposite to the lesion of any level of the lateral half of the sensori-motor tract. In lesions of the medulla, near the decussation, however, there is rather a general weakness, and only hemiplegia in the qualified sense that the limbs are more affected on one side. As Brown-Séquard has pointed out in lesions of one lateral half of the medulla, the nerve-fibres from the limbs may be caught in part before and in part after their decussation, and thus from a one-sided lesion there results bilateral paralysis.

Above this great decussation, cranial nerves emerge from the sensori-motor tract, and many, if not the whole, of these nerves have special decussations. Hence in hemiplegia, parts supplied by the cranial nerves are sometimes palsied on the same side as the limbs, and sometimes on the opposite side, according as the lesion affects the fibres of these nerves after or before their decussations. For instance, both in lesion of the lower half of the right[1] side of the pons and in lesion of the right corpus striatum, the arm and leg are palsied on the left, but in the former case the face is palsied on the side of the lesion (the right), because the fibres of the facial (portio dura) nerve are caught before its decussation, whilst in the latter the face is palsied on the side opposite the lesion, because fibres of the nerve are caught after its decussation. We must observe further that the decussation[2] of cranial nerves, the facial and hypoglossal nerves at all events, is incomplete, so that, as we shall have to insist on later, we find in lesion of the higher levels of the motor tract, that is to say, in lesions above the pons, and therefore above the decussations of these nerves, that the palsies of the face and tongue are not only on the side opposite the lesion, but that they are incomplete in degree—there is paresis rather than paralysis.

Cerebral Hemisphere. — Large parts of one cerebral hemisphere may be

[1] It will be convenient throughout this part of the article to suppose the right to be the side of the several lesions of which we have to speak.

[2] Lockhart Clarke has shown very clearly that there is a considerable decussation of the fibres of the ninth nerve. He finds that the nuclei of the facial are united by many fibres—commissural; but he has traced very few fibres of the *nerve* that decussate directly. (See Researches on the Intimate Structure of the Brain: Phil. Trans. 1868, p. 300, plate xiii. figs. 50 and 51.)

destroyed without producing obvious symptoms, either mental or physical. It is not said that *disease* in the hemisphere does not *lead* to symptoms; it very often does. (See art. Convulsion, p. 737.) In the article on Convulsion it was pointed out that hemiplegia frequently attends gross disease, syphilitic disease for instance, of the surface of the hemisphere, but in these cases the hemiplegia nearly always follows a convulsion, and depends probably on the nervous discharge in the convulsion itself.

It is, we repeat, certain that a large quantity of any part of either hemisphere may be destroyed while there are no symptoms, *with one important exception.* If the lesion involves convolutions near the corpus striatum—usually the left—there is, according to the size and *exact* position of the lesion, more or less defect or even complete loss of speech (aphasia).[1] The probability is that when a part of the brain is *slowly* destroyed, the undamaged parts take on the function of the part destroyed. Something similar is seen in the case of the eye. When the central vision is lost, the peripheral parts of the retina acquire greater acuteness of vision. In large and *sudden* lesions of the cerebrum there are symptoms, probably from the withdrawal of a part before this kind of accommodation can be effected.

We may have no symptoms from small clots in the hemispheres. In very large hemorrhages there are symptoms. These are usually, however, symptoms of Apoplexy; but if the Apoplexy be not very severe, we may discover some degree of hemiplegia. The palsy results either, as suggested, because a large part of the cerebrum is *suddenly* destroyed, or because the effusion by its mere bulk compresses the subjacent motor tract.

Lateral Ventricles. — In cases where blood breaks into the lateral ventricle from the corpus striatum or thalamus opticus, there is usually, not always, a convulsion and rapidly deepening coma. The convulsion, however, I believe, occurs not because blood is effused in the ventricle, but because under such circumstances the clot is usually a large one and has *suddenly* torn much of the brain: for severe convulsion (with tongue-biting) may usher in the Apoplexy, or may occur later when a large clot, starting in the bodies mentioned, is well walled in. If a patient be first hemiplegic with or without unconsciousness, the subsequent occurrence of a severe convulsion followed by universal powerlessness and deep coma is strong evidence of rupture into the ventricle, and is a very unfavorable sign.

We now come to the sensori-motor tract. In the first two divisions—corpus striatum and thalamus — the lesion is above the decussation, not only of the fibres for the limbs, but of all the cranial motor nerves, so that the paralysis produced is altogether on the opposite side of the body.

Corpus Striatum. — A lesion here produces what may be called "the common form of hemiplegia." As this is the most valuable symptom in the diagnosis of Cerebral Hemorrhage, it will be considered in some detail, but still only so far as bears on the diagnosis of Cerebral Hemorrhage.

It is very important, however, to observe that a *very* large and sudden effusion in this region leads to such deep coma that, although there must be paralysis of the limbs, we do not often *discover* any (just as in deep coma with fracture of the base of the skull we may not discover palsy of the face due to injury of the portio dura nerve until the patient's coma is passing off). We discover no local palsy, because there is palsy of both sides—universal powerlessness. This may be the result of squeezing of the opposite side of the brain by the sudden intrusion of a bulky mass into one side, the blood perhaps having also escaped into the lateral ventricles. Possibly the universal palsy may be in part owing to the destruction—the *sudden* lack—of those fibres which pass from the side of the lesion to the *same* side of the cord, as well as of those which decussate. (I have heard Dr. Brown-Séquard insist that many hemiplegic patients, some time after their seizure, were a little weak on what we usually call their sound side.) However, even in these cases we may learn that the case *began* in a hemiplegic manner, or we find some kind of one-sided symptoms, as tremor, rigidity, and the like.

As we usually see hemiplegia, always in chronic cases and most often in recent cases, the palsy is *incomplete* in range. It affects only the face, tongue, leg, and arm of one side. But in *complete* hemiplegia we have the following symptoms. We suppose the right to be the side of the lesion:—

1. The head turns to the right.
2. *Both* eyes turn to the right, and frequently both upper lids are fallen.
3. The muscles of the belly and chest are weakened on the left.
4. The muscles passing from the trunk to the left limbs are paralyzed.
5. The face is paralyzed on the left side.
6. The tongue on protrusion turns to the *left*.
7. The left leg is paralyzed.
8. The left arm is paralyzed.

Hemiplegia so complete only occurs

[1] This symptom is considered in art. Softening.

from a very grave[1] lesion, and even then, as a rule, the first two symptoms in the list pass off in a few hours or days. The symptoms in the list are given in what I believe to be the order in which the several parts suffer. It will be found that those parts suffer most and suffer longest (recover latest) which have the more voluntary uses. This is notorious of the arm and leg; the arm nearly always suffers more and recovers later than the leg. Of course the distinction into complete and incomplete hemiplegia is artificial. There are all degrees of paralysis according to degrees of gravity of the lesion. But there is an order in which paralysis increases in increasing gravity of lesions. We observe that the graver the lesion, not only are the more voluntary parts (arm and leg) *more* paralyzed, but that the further spread in *range* is the paralysis, and the method of its spreading is from the more voluntary to the more automatic parts—to parts higher up in the list. Thus, neglecting very small clots, a considerable lesion (I cannot use a more exact term, suddenness of effusion as well as size of clot being a factor) paralyzes only the most voluntary movements of one side of the body, those of the face, arm, and leg, and these parts in degree according to their degree of voluntary use. A larger lesion not only causes a deeper and more permanent palsy of these three parts, but it leads also to implication of more automatic parts; it causes the additional symptoms 1, 2, and 3 in the list. In still larger lesions the palsy spreads to the *most* automatic parts of the body, even to parts supplied by ganglionic nerves. It produces stertor from palsy of the palate and palsy of the respiratory muscles and of the heart—the palsy of respiration and of the heart showing itself chiefly in *slowness* of movement. There is also abasement of temperature. So we see that degrees of hemiplegia are "compound degrees." Not only are there degrees of more or less amount of loss of power of the face, arm, and leg—there is also, along with increasing degrees of loss of power of these most voluntary parts, increasing spreading of palsy to the more automatic parts of the body. There are degrees of hemiplegia, compound degrees as we have seen, from palsy of the most voluntary parts of one side only to almost universal paralysis, when, of course, "hemiplegia" is a misnomer.

Lateral Deviations of the Eyes and Head.[2]—This is not strabismus, as both eyes are still parallel, although both are turned to the right side—to the side of the lesion; to the non-paralyzed side. Indeed there never is, except as an accidental complication, strabismus from palsy of the third or of the sixth nerve in hemiplegia from a lesion above the crus cerebri. The patient,[1] especially when there is deviation of the head also, seems to be looking fixedly to one side. If the patient be sufficiently himself, we can get him to follow movements of our hand, and we may find that he can bring the two eyes to the middle line or even beyond it, but they soon fall again into the condition of deviation.

Along with this deviation there is often more or less turning of the head to the same side. There is, according to Vulpian and Prévost, "rotation," as there is after experimental lesion of one side of the brain in lower animals. But in man the rotation is only rudimentary; there is only a slight twist, not a real turning.

The clinical importance of these symptoms is that they may be, as Prévost suggests, valuable evidence of a local lesion—of a clot, for instance—in cases of coma where we can make out no paralysis of the limbs on one side, and when we are in doubt whether the coma is owing to a very large *local* lesion, such as extensive cerebral hemorrhage, or to a general condition, such as uræmia or opium poisoning. Two things further are to be noted. In cases of convulsion of one side of the body, the two eyes and the head turn to the side convulsed, and they may be permanently turned to the paralyzed side if it become rigid, and may strain still further in that direction when a convulsion or a higher wave of rigidity comes on. (Deviation of the head and eyes may occur in meningeal as well as in cerebral hemorrhage.)

The Muscles of one side of the Chest.[2]—Whether the muscles of the chest suffer

[1] This word is used to include two equally important factors, size and suddenness of lesion.

[2] In this country attention was first called to this symptom by Gull, Lockhart Clarke, and Hutchinson. It has been described by Vulpian and Prévost of Geneva (Gazette Hebdomadaire, Oct. 13, 1865). Cases by Drs. Humphry of Cambridge, Lockhart Clarke, Broadbent, and Russell Reynolds, and some interesting remarks on the symptom by Dr. Elizabeth Garrett (now Mrs. Garrett-Anderson), will be found in the *Lancet* for 1866.

[1] It has been likened to the conjugate deviation of the heads of two horses when an omnibus driver drops one of his reins, the other rein being "in tone." As, however, his reins do not decussate, that deviation is from the side of the "lesion."

[2] Dr. Broadbent has (Medico-Chir. Review, April, 1866) advanced an important and very valuable hypothesis to explain how it is that whilst the muscles which can act quite independently of their fellows on the other side of the body (those of the arm, for instance), suffer in disease of the corpus striatum, those which must act together (the intercostals, for instance) do not suffer.

or not in this form of hemiplegia, is disputed. Niemeyer says: "Patients who, as a result of apoplexy on the left side, cannot move the right arm or leg, move the right side of the thorax just as well as the left during respiration." Todd says that "it must be an extensive lesion which will paralyze the intercostal and abdominal muscles." In some cases of recent hemiplegia, when the patient voluntarily draws a deep breath, the side of the chest paralyzed certainly sometimes moves less than the other (see Wilks' Pathology of Nervous Diseases, Guy's Hospital Reports, 1866); probably, however, because the muscles passing from the trunk to the chest on the side of the paralysis will not act so strongly. At all events palsy of the muscles of one side of the chest is little, and soon passes off.

Face.—The muscles paralyzed are those supplied by the facial (portio dura) nerve. The whole of these suffer, but they suffer slightly. Moreover we find that the several parts of the face do not suffer in the same degree. And it is to be particularly noted that there is only slight weakening of the orbicularis palpebrarum. The patient can close his eyes, although not so strongly on the paralyzed side when urged to close them both tightly; sometimes, especially in chronic cases, we discover no difference. Hence this cerebral facial palsy differs remarkably from the facial palsy owing to affection of the trunk of the nerve (Bell's paralysis). The side of the cheek is the part most paralyzed, so that when the patient is asked to "show his upper teeth" the mouth is decidedly drawn to the sound side.

The tongue is not paralyzed; it is only weakened on one side, and on protrusion it turns but a very little to one side; it turns *to the side of the paralysis.* But sometimes, in case of loss of speech with hemiplegia, especially soon after the attack, the tongue seems to be much paralyzed, as the patient does not put it out when asked, even when he knows what is asked of him; he may try to get it out with his fingers. The fact that he can utter plainly some one or more words —mostly "yes" or "no"—that he masticates and swallows well, and that he may now and then put out his tongue to catch a stray crumb, shows that the tongue is not paralyzed in the ordinary sense of the word. There is loss of the most voluntary movements of it, and we may find that the patient cannot do other simple things that he is told; *e. g.*, open his mouth, shut his eyes, or frown.

Arm and Leg.—The arm suffers more and recovers later. We must not infer that the leg is not affected when the patient seems to move it as well as the other when he lies in bed. If we cannot get him up to walk, we can only say that the leg is not much paralyzed. Indeed it is an exceedingly rare thing to find paralysis of the arm without *some* weakness of the leg soon after the attack, although not at all uncommon to find that the leg recovers when the arm remains much paralyzed.

Thalamus Opticus.—Disease of the thalamus opticus produces hemiplegia which, in regard of the motor symptoms, is very like that produced by disease of the corpus striatum. In grave lesions there is lateral deviation of the head and eyes. Probably, however, the palsy is not similarly distributed. I think, for instance, that the arm suffers less, and the leg more, the further back the lesion is placed in the two divisions of the motor tract, the corpus striatum and optic thalamus.

But in disease of the thalamus there is also diminution, or, soon after the attack, loss of sensation, and not of the arm and leg only, but of the whole half of the body, quite up to the middle line. This loss of sensation has been denied; one reason no doubt is, and it is important to bear this in mind, that sensation returns much more quickly than does power of movement.[1]

Crus Cerebri.—Damage to the crus cerebri causes hemiplegia on the opposite side like that above described, there being loss of motion if the under part of the crus only be damaged, and loss of sensation also if the upper strands be damaged as well. From the crus emerges the first cranial nerve, and at this level of the motor tract we may have "*cross paralysis.*" If the under and inner part of the crus be involved, the third nerve is paralyzed on the side of the lesion—on the side opposite the paralysis of the limbs. The nerve is, so to speak, caught as it goes in,[2] and of course before any of its fibres can have decussated, whilst the fibres from the arm and leg are caught long after their much lower decussation in the medulla oblongata. But we can only make the diagnosis of lesion of the crus when the two symptoms—the palsy of the third nerve and the hemiplegia—come on at the *same time.* If they come on at different times, it is just as likely that there are two lesions,—one of the trunk of the nerve and the other in the thalamus or corpus striatum. This is important in the diagnosis of the *nature* of the lesion, for when the two symptoms—the palsy of the third nerve, and the hemiplegia—come out at different times, or if the palsy of the third nerve be on the same side as the hemi-

[1] For important observations on this subject, see Broadbent, Med.-Chir. Review, April, 1866.

[2] It is convenient to speak of the fibres of both the motor and sensory nerves as passing from their external distribution to the nervous centres.

plegia, we may be practically certain that there is not Cerebral Hemorrhage. In these cases the disease is mostly syphilis.[1]

So far the sides of the motor tract are distinct. In the next two divisions the two sides are welded together.

Pons Varolii.—Hemorrhage may be limited to one lateral half, or it may occupy both sides of the pons. (It may extend to the crus cerebri or crus cerebelli.) In the former case there is hemiplegia; in the latter double hemiplegia, or rather a condition of universal powerlessness, in which, as there is usually deep coma, we can make out no *local* paralysis—a condition, as we shall see, very like that of uræmia and opium poisoning. In other cases the palsy, although on both sides, is more marked on one side.

We speak here of hemiplegia from lesion of one lateral half of the pons,[2] and we speak of lesions of the right half. Here again we have to do with cross paralysis, because we have to do with cranial nerves which decussate in the pons itself above the great decussation of the fibres for the limbs. The cranial nerves are the fifth, sixth, and facial. First, for general remarks on the effect of lesions at different levels of one lateral half of the pons, taking the facial nerve (portio dura) for illustration. In the right half of the pons, there are fibres of the right and also of the left facial nerves, those of the right before its decussation, and those of the left after its decussation. (1) A lesion in the lower part of the pons will catch the fibres of the right facial nerve before its decussation, and will also involve the fibres from the left limbs, which have crossed lower down in the medulla. There is then palsy of the right side of the face, on the side of the lesion, and of the arm and leg on the left, the side opposite the lesion. (2) A lesion in the upper part of the pons (Brown-Séquard, op. cit. p. 153) may destroy fibres of the left facial nerve which have crossed in the pons, and will also involve the fibres for the left limbs. Here then the facial paralysis will be on the side opposite the lesion, and therefore on the same side as the palsy of the arm and leg. The hemiplegia will be like the hemiplegia from disease above the pons. Hemiplegia of this kind is very rarely caused by disease of the pons. (3) Lastly, Brown-Séquard points out, that a lesion in the middle of the pons will catch the fibres of the right facial nerve *before* its decussation, and those of the left *after* its decussation. Then the face is palsied on *both* sides from lesion of the right half of the pons and the arm and leg on the left.

To repeat, a lesion of the right half of the pons affecting the facial nerve near its implantation causes palsy of the face on the right side and of the arm and leg on the left. A lesion in the upper part of the right half of the pons causes the palsy of the face and of the limbs on the left side. A lesion of the right half of the pons may be so extensive as not only to affect the right facial nerve before its decussation, but to extend to the fibres of the left facial nerve after its decussation. Then there is palsy of the face on both sides, more marked on the right, and palsy of the left arm and leg.

What has been said applies to the fifth nerve. An extensive lesion may involve both the facial and fifth nerves, and then there is palsy of the region supplied by the fifth nerve and portio dura nerves of the right side and of the leg on the left.

Here it is to be observed that in most cases not only does the palsy of the face in cross paralysis from disease of the pons differ from the facial palsy in the common form of hemiplegia, in that it occurs on the side opposite the limbs paralyzed, but it differs in that it is much more decided in degree. The facial palsy may be as extensive as that which results from disease of the trunk of the portio dura nerve. There is, in short, Bell's paralysis of the face. And when the fifth nerve is affected, the face is anæsthetic, the masseter and temporal muscles are much palsied, and after a time they waste as they do in disease of the *trunk* of the fifth.

There are, in actual practice, combinations of symptoms which are more difficult to understand, but which can be resolved by a consideration of Lockhart Clarke's researches. (See especially Philosophical Transactions, Part I., 1868.) When the portio dura nerve is paralyzed, we often find paralysis of the sixth nerve on the same side too. The opposite sixth may be also paralyzed in a less degree (Brown-Séquard, op. cit.).[1] The relation of these two symptoms—the facial and ocular palsy—is easy to understand when we remember that the facial and sixth nerves, although they emerge at different places,

[1] An important case of hemorrhage into the crus cerebri has been published by Dr. Hermann Weber in the Med.-Chir. Society's Transactions, vol. xlvi. This report gives a careful account of the condition of sensation and of temperature on the two sides of the body; there is a reference to cases previously published.

[2] In an article on Cerebral Hemorrhage, we can only do so in outline. For a full account of the symptoms resulting from disease of the pons Varolii, see Brown-Séquard's Lectures in the *Lancet*, 1861, vol. ii.; and Lockhart Clarke's papers, Phil. Trans., 1868.

[1] We should carefully examine the condition of the opposite sixth; and if it be not markedly paralyzed, we may see oscillations in the eyeball when the patient tries to avert it, showing that the external rectus on that side also is weakened.

arise, as Stilling and Lockhart Clarke have shown, from a common nucleus.

Again, we must observe that in limited disease in one half of the pons, the parts supplied by the *motor* division of the fifth nerve may be paralyzed on the side of the lesion when there is no diminution of *sensation* on that side. We find diminution of sensation on the *opposite* side of the face to that on which the limbs are affected. (This has occurred, in the cases I have seen, only when there has been palsy of the sixth and facial also.) Since Lockhart Clarke has found that the bundle of fibres of the sixth and the bundle of fibres of the portio dura nerve, after arising from their common nucleus, diverge so as to inclose the motor nucleus of the fifth—the fibres of the portio dura separating it from the sensory nucleus of the fifth—it is easy to understand why we have, from a single lesion, palsy of the portio dura sixth, and the *motor division* of the fifth all on one side. But the occurrence of the diminution of sensation on the opposite side of the face is not easy to understand unless we assume a crossing of the sensory and motor fibres of the fifth in the pons, on separate levels, analogous to the crossing of sensory and motor fibres of the limbs on separate levels, which Brown-Séquard has discovered.

As to the limbs in hemiplegia from disease of the pons, there is usually affection both of sensation and motion. We find, of course, differences in the amount of loss of power, and of degree of anæsthesia. Moreover, we find differences in distribution of the two. I have seen the leg very much palsied and anæsthetic when the arm was scarcely weakened but nearly altogether anæsthetic. These points do not specially concern us in an article on Cerebral Hemorrhage, although they are of very great physiological interest, as showing relations of strands of motor and sensory nerve-fibres for the limbs in the pons.

Whilst the diagnosis of hemiplegia from hemorrhage on one side of the pons is usually very easy, the diagnosis of hemorrhage into both sides of the pons is sometimes very difficult. A large effusion in this part usually produces death rapidly—in a few hours. In large effusions there is usually marked contraction of the pupil on both sides, and there is universal powerlessness—a condition so like that of opium poisoning, that treatment for opium poisoning has been adopted in cases of hemorrhage into the pons. (See Special Diagnosis.)

Medulla Oblongata.—Of the effects of effusion of blood limited to the medulla oblongata little is known. A large effusion would no doubt be very rapidly fatal, but I have never seen a large effusion here. I have made but one autopsy on a patient who had had a small effusion limited to the medulla. I saw this patient with Dr. Lockhart Clarke and Dr. Morell Mackenzie. The patient had recovered from an attack of hemiplegia due, as we afterwards found, to a clot in his right thalamus opticus, when all at once he lost power to articulate from paralysis of his tongue—remaining able to write well. A few years later the patient died, and Dr. Lockhart Clarke,[1] to whom I gave the medulla oblongata, found in it remains of past effusions of blood.

The symptoms which would lead us to infer disease of the medulla oblongata are paralysis of the lips, tongue, palate, and vocal cords. These symptoms, however, mostly come on very slowly, and usually both sides are equally affected. This is so in the "Paralysie labio-glosso-laryngée" of Duchenne. There can be no clot nor any kind of sudden lesion in such cases. If any of these palsies are on but one side, they must be attributed to tumor or to syphilis, if they come on slowly; but whether on one or on both sides, if they or any of them come on suddenly, they must be attributed either to clot or to softening from thrombosis.[2]

Cerebellum.—Nothing definite can be said as to the special symptoms produced by hemorrhage into the cerebellum. Sometimes there is loss of consciousness, and sometimes there is not. Sometimes there is hemiplegia, and at other times none. And when there is hemiplegia, it is sometimes on the side of the lesion and sometimes on the opposite side. Of course these differences depend on differences in the exact part of the cerebellum injured, on the size of the clot, and on the rapidity of the effusion, but these differences have not yet been put in order. When there are no paralytic symptoms, we can make no diagnosis. Sometimes there is a conjugate deviation of the eyes—not lateral deviation: one eye is turned upwards and outwards, and the other downwards and inwards. When this symptom is present, we may diagnose sudden lesion of the crus cerebelli. When there is hemiplegia, the palsy is not diagnostic as to the seat of the lesion unless perhaps we can ascertain the absence of facial and lingual palsy. For Brown-Séquard (*Lancet*, Nov. 2, 1861) says that in hemiplegia from hemorrhage in the cerebellum there is neither lingual nor facial palsy, although there is a loss of facial expression. In cases of deep

[1] See Phil. Trans., part i. 1868, where the case is published.

[2] Here I would refer the reader to very important remarks by Lockhart Clarke (Phil. Trans., part i. 1868, pp. 316–17, &c.), and to a case illustrating his views which I have published (London Hospital Reports, vol. i. 1864, p. 361).

coma, however, we might be unable to tell whether there was facial or lingual palsy. But if there be hemiplegia of any kind in a case of apoplexy, we can at all events say there is a local lesion, and this with other symptoms would be good evidence of the existence of large Cerebral Hemorrhage—and this is the most important matter—in some part of the encephalon. Vomiting occurs in Cerebral Hemorrhage, but *very* urgent vomiting would point to Cerebellar Hemorrhage: severe pain at the back of the head would supply still further evidence, but this symptom would only be presented when the patient was not unconscious. Fortunately, hemorrhage in the cerebellum is very rare, so that we have not often the chance of being wrong.

[Since this article was written, an important advance has been made in the direction of inquiry above pointed out. Localization of brain-lesions (not only hemorrhagic, but also traumatic, inflammatory, and degenerative) has been during the last decade (1870–80) extensively studied, by combining the results of experimental physiology with those of clinical observation and morbid anatomy.

Since 1861, Dax, Bouillaud, and Broca have obtained the general recognition in pathology of the causative association of lesion of a certain portion of the left cerebral hemisphere with aphasia.[1] Besides the now neglected but ingenious hypothesis of Gall (popularly known as phrenology), other suggestions had been often previously thrown out, of a similar kind. One of the most noticeable of these was that of Serres,[2] based upon two actual cases; to the effect that "if the right arm be affected, the lesion will be in that portion of the brain which corresponds to the upper left parietal bone, and *vice versa*." The study and discussion by Bravais in 1827 of "hemiplegic epilepsy," and somewhat later, by Todd, of "epileptic hemiplegia," followed by Hughlings Jackson's able analysis of unilateral convulsions, and of epilepsy connected with cortical brain lesion, prepared the way for the new era in localization. In 1870 were published the investigations of Fritsch and Hitzig upon the effects of electrization of the surface of the cerebral hemispheres in animals. These were followed by similar experimental inquiries by Ferrier, Schiff, Braun, Nothnagel, Eckhard, Carville, Duret, Burdon Sanderson, Lussana, Vulpian, and Dupuy. In the connected clinical and pathological observations and studies, contributions have especially been made by Charcot, Bouchard, Pitres, Brun, Lépine, Meynert, Huguenin, Proust, Broca, and Hughlings Jackson.

Upon the physiological portion of the subject, Ferrier may be considered as the representative authority. From the carefully studied effects upon animals of the stimulation by electricity of different portions of the cerebral hemispheres, he concludes that, in the gray matter of the convolutions, there are *psycho-motor zones*, which have a functional relation to the voluntary movements of certain groups of muscles. These are not exactly the same in all animals; being, for example, differently located in monkeys, dogs, and cats. From experiments upon monkeys (confirmed by a considerable amount of evidence from morbid anatomy), it is inferred that the psycho-motor zone is, upon each side, in the immediate neighborhood of the fissure of Rolando; comprising the ascending frontal and ascending parietal convolutions, the paracentral lobule, and probably the base of the frontal and superior and inferior parietal convolutions.[1] "Broca's convolution," the now accepted seat of the faculty of speech, is the third frontal convolution of the anterior lobe of the left hemisphere.

The other "centres" in this zone are, by the advocates of localization, regarded as governing the volitional movements of the head, eyes, eyelids, pupils, angles of the mouth, arm, hand, leg, and foot. Ferrier draws an important distinction between the central determination of voluntary, and that of automatic, instinctive, or reflex movements. The latter he refers to the lower centres, at the base of the brain; ascribing to the gray matter of the limited region above named those actions only which involve conscious discrimination. He also asserts (with less amount of plausible evidence, however) the existence of sensory centres, in the parieto-temporal region, between the motor-zone and the occipital lobes. To these, the occipital lobes, he ascribes, hypothetically, a governing relation to the viscera, and the functions of organic life. The most anterior frontal and orbital regions of the cerebral hemispheres he believes may be the seats of true *psychical* centres.

Somewhat confirmatory of these views, as to a regional subdivision of the functions of the hemispheres, are the observations of Duret, Cohnheim, and Charcot, upon the distribution of the arterial circulation of the brain. Duret asserts that the areas or territories of vascular supply are to a considerable extent independent of each other. This has great consequence in connection with cerebral em-

[1 See Aphasia, in the article in this volume on Softening of the Brain.]

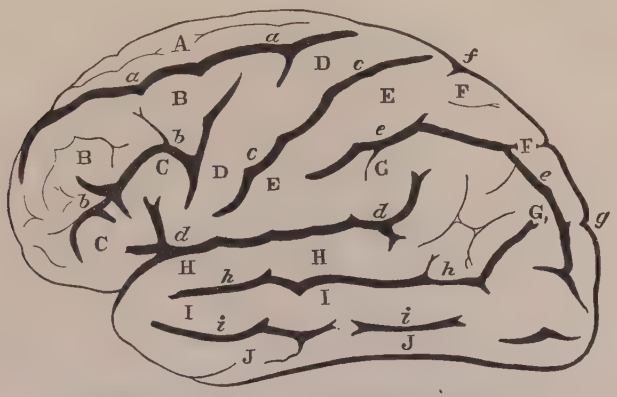
[2 Anatomie Comparée du Cerveau, 1824–26.]

[1 See Ferrier on the Functions of the Brain, 1876; on Localization of Cerebral Disease, 1878.]

bolism. The "motor zone" is supplied by the superficial branches of the Sylvian artery. If thrombosis or embolism of this vessel occur after it has given off the branches going to the corpus striatum, on either side, softening may occur in the psycho-motor region alone; producing hemiplegia of the opposite side as one of its results.

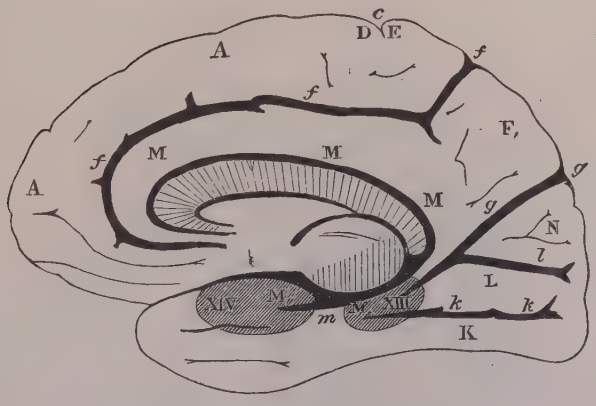
Notwithstanding the amount of evidence accumulated in favor of this theory of localization, it cannot be considered

Fig. 54.



Lateral view of brain, showing fissures and convolutions.

Fig. 55.



Inner surface of hemisphere, showing convolutions and fissures, and Ferrier's centres of touch, smell, and taste.

Fissures (Figs. 54 and 55):—*a*, superior frontal; *b*, inferior frontal; *c*, fissure of Rolando; *d*, fissure of Sylvius; *e*, inter-parietal; *f*, fronto-parietal; *g*, parieto-occipital; *h*, first temporo-sphenoidal; *i*, second ditto; *j*, inferior ditto; *k*, occipito-temporal; *l*, calcarine; *m*, hippocampal.

Convolutions (Figs. 54 and 55):—A, superior, or first frontal; B, second ditto; C, third ditto; D, ascending frontal; E, ascending parietal; F, superior parietal; F_1, præcureus; G, supra-marginal; G_1, gyrus angularis, or *pli courbe;* H, first temporo-sphenoidal; I, second ditto; J, third ditto; K, fusiform lobule; L, lingual lobule; M, gyrus fornicatus; M_1, gyrus hippocampi; M_{11}, uncus gyri fornicati, or subiculum cornu Ammonis; N, cuneus.

yet as a finally established doctrine in Physiology. Brown-Séquard has brought the weight of his immense experience in cerebro-nervous experimentation and morbid anatomy to bear against it. Vulpian, Lussana, and Lemoine prefer to ascribe to the fibres of white substance, connected with the cortical gray matter of the brain, the motor control in question. Goltz, Schiff,[1] Munk, Luciani, and Tamburini[2] have obtained considerably different experimental results from Ferrier's.

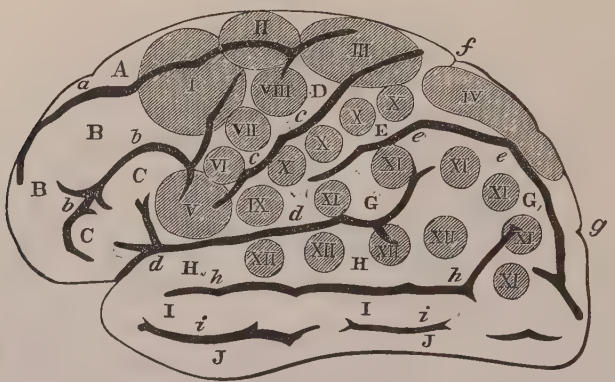
Brown-Séquard's position on the subject affords a remarkable exemplification

[1 See also Lauterbach, Amer. Journal of Med. Sciences, October, 1877, p. 371.]

[2 Brain, July, 1879, p. 189.]

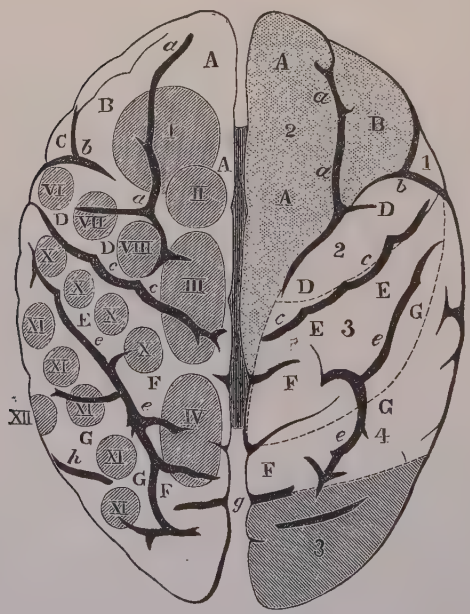
of the liability of vivisectory experimentation to complicate the problems it proceeds to solve. After having arrived at quite different results in his previous investigations, they, and those of other physiologists, have been, by his last demonstrations, swept away; with the deduction of conclusions such as the following: a lesion of one side of the brain can produce symptoms either on the same or on the other side of the body; a lesion on both sides of the brain may cause symptoms limited to one side of the body; and most extensive lesions may occur in any or all parts of the brain without corresponding symptoms. Instead of a few restricted cerebral centres governing special functions, he believes that very numerous brain-cells related to each of such functions must be located throughout the hemispheres; acting in *solidarité* by means of intercommunication amongst them.

Fig. 56.



Lateral view of brain, showing Ferrier's psycho-motor centres.

Fig. 57.



Upper aspect of brain, showing on left side Ferrier's centres; on the right, the arterial areæ.
The Roman numbers in Fig 56, 57, and 55, refer to Ferrier's centres.

I, lateral movements of head and eyes, with elevation of eyelids and dilatation of pupils; II, extension of arm and hand; III, complex movements of arm and leg, as in climbing, swinging, &c., IV, movements of leg and foot, as in locomotion; V, movements of lips and tongue, as in articulation, VI, depression of angle of mouth; VII, elevation of angle of mouth; VIII, supination of hand and flexure of forearm; IX, centre of *platysma*, retraction of angle of mouth; X, movements of hand and wrist; XI, centre of vision; XII, centre of hearing; XIII, centre of touch; XIV, centre of smell and taste. (From Bristowe.)

Each hemisphere, especially, he asserts to be in itself complete for all brain functions, for both sides of the body; although both are not nearly always alike developed and actively used. Minute anatomy has not yet furnished a complete explanation of the intercommunication of all parts of the brain; the exact nature and functions even of the neuroglia cannot be said to be fully understood. Analogy furnishes an important suggestion, in the discovery by Gerlach, confirmed by Boll,[1] of a *network* of extreme fineness in the spinal cord, composed of the union of the ramifying nerve-filaments of the spongy substance of the cord.

Dupuy, by his experiments, has placed a great difficulty in the way of the adoption of the hypothesis of cortical motor centralization. Having exposed the "motor zone" of the left hemisphere of a dog, he produced the usual movements by electrical stimulation, and then cauterized the cortex of that region. Electricity applied to the cauterized part still caused the same movements. Leaving the animal for four weeks, it presented no symptoms affecting the motor functions. Then, reopening the wound, he found a dry eschar, with meningeal adhesion. Electricity applied to the eschar produced no movement; but application of the current to the parts around the eschar brought on muscular action. This certainly shows great insufficiency in the proof of the *dependence* of the motor function upon the condition of the cortical substance. This, indeed, is not exactly what Ferrier has asserted, as he refers a large class of movements to the corpora striata and other lower brain centres, and dwells on the fact that actions which at first are volitional, may become subsequently automatic; the superintendence of the higher centres being sometimes, when incomplete, supplemented, or even substituted by the lower centres. This happens with especial facility in the *lower* animals.

On the whole, all that has been previously known and rendered probable of the functions of the cerebral hemispheres makes it reasonable to emphasize what Ferrier recognizes as the *psychical* element in the history of these phenomena. Other reasons exist, which have been very generally overlooked, for concluding that the anterior portion of the cerebrum is emotional in function; the intellectual powers having their seat in the posterior, or postero-median, portion.[2] For emotional expression there must be a relation to the muscles, through their immediately controlling centres; and this relation appears to be well established in regard to certain parts, at least, of the anterior region of the brain. As a question in Physiology, this is all that should be claimed as settled; the *nature* and *extent* of the relation being yet open for farther investigation. In experimentation upon the brain and nervous system, one of the most important facts is, the frequent *diffusion*, not only of electrical stimulation, but of *all* impressions. Action at a distance, through nervous conduction, directly stimulant, reflex, or inhibitory, is, rather than exceptional, almost the rule. This introduces a great difficulty into the interpretation of results, both of vivisection and of accidental injury or disease.[1]

Coming to our immediate subject of the pathology and diagnosis of brain affections, this must be considered upon the merits of its own evidence, while awaiting the explanations of Physiology, to be hereafter completed. It has been known since the time of Andral, that extensive lesions of the cerebral cortex may occur without motor symptoms. Andral, Vauttier, Marot, Herpin, Sabourin and others[2] have reported cases of this kind. Mention has been made already of the names of those who have especially studied of late the coincidence of marked muscular symptoms (paralysis, convulsions, &c.) with lesions of a limited portion of the surface of the hemispheres.

It is necessarily admitted[3] that, as yet, it is not always possible to make during life a certain diagnosis between paralysis from lesion of the cerebral cortex and that from lesion of the corpus striatum, alone, or involving a portion of the internal capsule. In both, those movements are most affected which are the most volitional. Sensation is not affected in either, if the lesion be confined, in the one case, to the cortex, in the other, to the anterior two-thirds of the capsule. In neither is the nutrition or electric contractility of the paralyzed muscles impaired. In both, a tendency exists to the development, sooner or later, of descending sclerosis of the motor tracts of the crus, pons, medulla oblongata, and spinal cord, with late rigidity or contraction of the paralyzed limbs.

Characteristic, however, of paralysis of

[1 See Erb, in Ziemssen's Cyclop. vol. xiii.]

[2 Since 1860, I have taught this in lectures to students of physiology; having been led to it by facts in embryology and comparative anatomy, to which this would not be the place to allude in detail. See my Manual of Physiology for Students, 2d edit., Philada., 1874.]

[1 To make the most of this argument, one might ask what more of *demonstration* is there of purely motor centres in the cortex of the brain, than there is of *epilepto-genetic centres* in the skin of the guinea-pig, under Brown-Séquard's experiments?]

[2 Charcot and Pitres, Revue Mensuelle de Médecine et de Chirurgie, Jan. 1877.]

[3 Ferrier, Localization of Cerebral Disease.]

cortical origin seems to be the occurrence of dissociated paralyses, or monoplegiæ. An arm and hand alone, or the face, or one leg, may be affected with paralysis, which may successively extend to other parts. A comprehensive summary from Ferrier[1] may conclude our account of this subject:—

"While we cannot be quite certain of the position or extent of a cortical lesion causing a sudden and complete hemiplegia, we may take a monoplegia of the leg or of the arm and leg as an indication of lesion of the upper extremity of the ascending convolutions close to the longitudinal fissure; brachial monoplegia as a sign of lesion of the upper part of the ascending frontal convolution, or, if the paralysis affect the hand more particularly, of the ascending parietal convolution; brachio-facial monoplegia as indicating lesion of the mid-fronto-parietal region; while facial and lingual monoplegia, or this combined with aphasia, indicates lesion of the lower part of the ascending frontal convolution, where the third frontal unites with it."—H.]

The Apoplectic Condition.

Apoplexy, as was remarked in the Introduction, p. 902, is not peculiar to Cerebral Hemorrhage. It will be seen when we come to Special Diagnosis that it is sometimes difficult and occasionally impossible to tell whether Apoplexy is owing to a fatal lesion of the brain, or to the comparatively minor cause, deep drunkenness. Again, from clot there are all degrees, from slight and transient mental obscuration to profound and rapidly fatal coma. It is not *à priori* likely, when we consider that the clots vary in size, in the suddenness of their irruption, and in their seats of effusion, that there would be any uniformity in the conditions produced by Cerebral Hemorrhage, and, as a matter of fact, the conditions vary very much indeed. We can here only speak of *severe* cases, admitting that they are not typical. Under the head of Diagnosis we shall notice cases of Cerebral Hemorrhage in which there is no loss of consciousness; and under the head of Special Diagnosis, various degrees of impairment, or "loss of consciousness"—if the phrase *degrees* of *loss* of consciousness be permissible—will be spoken of.

The striking symptoms of the apoplectic condition are, (*a*) loss of consciousness, (*b*) states of pupil, (*c*) stertor, (*d*) alteration of pulse, respiration, and temperature.

Loss of Consciousness.—We are considering a severe case—apoplexy, the result of a large and sudden hemorrhage—and as we admit that such a case is not typical, we shall only discuss how it happens that from a *circumscribed* lesion in but one side of the brain there results *total* abolition of consciousness.

We know that loss of consciousness cannot result from mere lack of the comparatively small part which the hemorrhage has *destroyed*, for, as autopsies show, much of the brain may remain lacking for years in patients who have been unconscious only a few hours or days at the time when the destroying lesion occurred. But Cerebral Hemorrhage is a bulky lesion—it squeezes; it is also a brusque lesion—it not only destroys, but it destroys suddenly—there is shock.

Niemeyer attributes the loss of consciousness to squeezing. He does not suppose it to depend on squeezing of the nerve-fibres and cells, but on squeezing of the capillaries; in other words, he attributes it to rapid anæmia of the brain, produced mechanically. It is true that there is no loss of consciousness in cases of very large cerebral tumor when there is evidently increased intracranial pressure; but clot and tumor are manifestly not comparable lesions. Tumors increase slowly, and probably, as Niemeyer suggests, the fibres of the part of the brain directly compressed become atrophous, and thus more room is made. Again, there is compensation by diminution in the quantity of cerebro-spinal fluid. But blood is rapidly effused, and the clot will squeeze before either of these modes of accommodation can take place, and thus the accommodation is, according to Niemeyer, obtained at the expense of the capillaries. They are emptied. In estimating the gravity of a lesion, *rapidity* is to be considered almost as important a factor as *size*, as will be best seen when we come to speak of sudden death from intracranial hemorrhage.

Mr. Hutchinson (Lectures on Compression of the Brain, London Hospital Reports, vol. iv., 1867) also believes that the apoplectic condition is the result of *extensive* and *rapid* squeezing of a large quantity of the brain, and that the direct cause of the loss of consciousness is sudden anæmia *quoad* arterial blood. He points out that an enormous effusion, if it occurs very slowly, need not be attended by any insensibility whatever.

The above theory of the occurrence of loss of consciousness from anæmia, mechanically caused, may serve in the explanation of many cases of *large* Cerebral Hemorrhage, but will not serve in all cases. It does not explain, as Jaccoud insists, the transitory loss of consciousness which sometimes occurs from small clots, the squeezing from which can be very trifling. Moreover, there are many

[1 Op. citat.]

cases of loss of consciousness from other causes in which there can be no squeezing, for instance in some cases of embolism[1] of the middle cerebral artery, and in the fit of epilepsy. In cases of laceration of the brain from injury—cases without any considerable effusion of blood being now considered—the apoplectic condition comes on although there is no squeezing. Thus Mr. Hutchinson says: "It is sometimes quite impossible to make any diagnosis by the symptoms alone [cases where there is no history of the mode of onset, for instance] between cases of sudden compression of the brain and those of laceration of the brain." We must consider the shock-producing element — that of suddenness of lesions.

We may here avail ourselves of what Jaccoud has written in his important work, "Pathologie Interne," vol. i. p. 164. The normal function of the brain, he says, depends on the joint and simultaneous activity of its two halves. When one is injured, the other can in a certain measure *compensate*, provided the lesion occurs slowly, as in tumor. But the disturbance of a *sudden*, although local, lesion reacts on the whole brain, its two halves being *united by "powerful" commissures*. The torn brain receives a *shock* on the side injured directly, and this is transmitted and reflected on the other side, and then there is produced the "névrolysie"[2] which is apoplexy.

In unconsciousness produced by uræmia there may be no arterial anæmia in the ordinary sense of the expression, but still in effect the action will be the same. The blood is not good arterial blood. But, if we accept Traube's view that Bright's disease leads to œdema of the brain, there will be veritable anæmia in uræmia from the squeezing which the exuded fluid will cause.

Pupils.—There seems to be a wide-spread impression that when a patient is comatose, his pupils must be either "contracted" or "dilated."[1] It is quite certain that there may be a very large clot on one side of the brain when the pupils cannot be declared to be normal. Extreme contraction or extreme dilatation of the pupils are rare symptoms in Cerebral Hemorrhage. It is to be observed that in the many cases of coma, although the pupils are very small when the patient is left still, as small as in healthy sleep, they may become much larger when attempts are made to rouse him. From not considering this, different accounts are given as to the condition of the pupils in the same case by different observers, or there are supposed to be remarkable variations in the signs of the pupils. On the whole, the conditions of the pupils are of little value.[2]

We shall speak of contraction of both pupils under the head of Diagnosis of Apoplexy, owing to hemorrhage into the pons, from opium poisoning. The following further remarks on the pupils belong strictly to diagnosis, but they are most conveniently considered here.

Importance must be attached to difference in the size of the two pupils, but only when the difference is great; for the pupils are often of slightly different size in healthy people. Difference in the size of the pupils points to a local lesion. Thus, were one pupil very minutely contracted and the other presumably unaltered, the contraction would be some evidence of disease of the pons Varolii on the side of the contraction. But I have not yet seen minute contraction of the pupil on one side from clot in the pons when there has been coma. I have seen it in cases of hemiplegia from disease of one side of the pons: in one of these cases there was a clot. Were one pupil *very widely dilated*, it would lead me to search most carefully for signs of injury to the head, as Mr. Hutchinson has found very wide dilatation of one pupil in cases of blood effused

[1] He says, op. cit. p. 139, of the apoplexy which occasionally occurs from embolism, that it may be attributed "soit à une *névrolysie* produite par la perturbation subite de l'équilibre circulatoire (Jaccoud), soit à un œdéme aigu généralisé par suite de l'augmentation de pression dans les artères perméables (Niemeyer). Cette dernière interprétation me paraît difficilement admissible pour les cas où l'apoplexie dure à peine quelque minutes."

[2] Niemeyer, op. cit. vol. ii. p. 184, admits that "the entire loss of consciousness, the apoplectic attack, which usually accompanies the commencement of hemiplegia when the arteria fossæ Sylvii is stopped by an embolus," is difficult to explain. He thinks it is "most probably due to the diseased hemisphere being decidedly swollen by collateral œdema, and that, as occurs in large extravasations of blood, the opposite hemisphere is not sufficiently protected from the pressure by the falx, which only offers a limited amount of resistance."

[1] In case-taking, I prefer the terms *small* and *large*, as they have not the misleading implications the more technical terms have.

[2] Dr. Wilks says (Guy's Hospital Reports, 1866, p. 177) that "we cannot connect the conditions of the pupils with any definite lesions, for their state is very variable and liable to be influenced by very slight causes." Speaking of the contraction of the pupils observed in disease of the pons, he adds, "just as we see this produced by effusion of blood at the base or into the ventricle." Callender (St. Bartholomew's Hospital Reports, vol. iii. p. 430), speaking of the pupils in cases of injury to the head, says, "their condition varies remarkably in these cases, and no sort of reliance can be placed upon the appearance they may seem to present."

under the dura mater in the sphenoidal fossa from fractured base; the dilatation is on the side of the effusion.

During convulsive seizures the pupils may dilate very widely; this is not always the case.

Stertor is the sign of a grave lesion, or more generally of serious implication of the brain, and, like the other symptoms of the apoplectic condition, is not of diagnostic value. There may be uræmia, alcoholic poisoning, or large Cerebral Hemorrhage. It will occur in any one of these conditions. It may be absent in any of them, and is often absent in apoplexy from Cerebral Hemorrhage. The noise made will depend on the condition of the respiration, and varies with it in the same case. It shows that the lesion is large enough or widely enough spread in the brain to affect muscles supplied by or through the ganglionic nervous system. It is of value in prognosis. It shows grave lesion.

Pulse, respiration, and temperature.—This is the most important and at the same time the most difficult part of the subject. It is really impossible to give a proper account of the condition of pulse, respiration, and temperature without citing numerous cases, and these would show extreme differences, differences so great that it is most difficult to make generalizations. The great point to observe is that the condition of the pulse, respiration, and temperature varies very much according to the time elapsed from the seizure. So that so far as the pulse, respiration, and temperature are concerned, the patient may be in opposite conditions according as he is seen early or late. And since the lesions differ greatly in gravity—in size and suddenness—we can say nothing definitely as to time. What is early in one case is late in another. This is obvious when we consider that a clot may destroy life in an hour, or may not kill for days, or that the patient may recover from it. Moreover, alterations of pulse, respiration, and temperature, depression of them at all events, are not peculiar to Cerebral Hemorrhage; they are found in cases of meningeal hemorrhage, alcoholic poisoning, and even in some cases of tumor of the brain. We are obliged then to speak most generally. For convenience we make two stages of the apoplectic condition: one we call collapse and the other reaction. There is no absolute demarcation betwixt the two. Loss of consciousness continues through both. Of course, if the clot be small there are no stages—neither collapse nor reaction. Again the patient may recover from his collapse, if it be faintly marked, without any obvious reaction; he may die rapidly, and then there is practically no question of stages.

First stage:—Soon after the effusion, even of a large clot, the pulse, respiration, and temperature may be absolutely normal, the patient seeming as if gently asleep. But we shall speak of cases in which they *are* abnormal; the symptoms are those of depression. The face is pale, the pulse is slow and labored, the respiration is shallow, and the temperature is lowered. The pulse may be 60 or under, the temperature may be in the axilla 96 or lower.

In the second stage, the stage of reaction, the pulse quickens, respiration quickens, and the temperature usually rises, for instance, to 101, or 103. Shortly before death, it may rise to 107 or more. These points concern us most in prognosis: the quicker the pulse and respiration, and the higher the temperature, the less likely is the patient to recover. We frequently observe also that the pulse becomes irregular or intermittent. The respiratory action undergoes great variation in frequency. Thus the patient for a while lies breathing quickly, but pretty evenly in rate, and then for a short time there is a series of more rapid respirations, with loud stertor, after which comes a period of comparative calm.

Again, not only is the rate of respiration to be considered, but the character of the respiratory movements are to be noted. As they quicken in *rate*, so do they become more extensive in *range*, though each respiration is still short. Thus, in the first stage there may be only quiet action of the diaphragm, but at length the sides of the chest evert strongly in inspiration, the abdominal movement being less obvious, and at length the upper thorax takes part in the process. In severe cases the epigastrium sinks in during inspiration. This is probably partly owing to elevation of the attachments of the diaphragm from increased action of the sides of the thorax, and partly to pushing down of the diaphragm by the increasing bulk of the lungs from congestion or œdema.

Diagnosis.

An account has been given of the general bodily state of the patient who is especially liable to Cerebral Hemorrhage. We have described the symptoms which we know that hemorrhage produces—both the local, such as varieties of hemiplegia, and the general, the apoplexy which occurs from large and sudden hemorrhages; but other lesions produce exactly similar local symptoms, and many conditions cause Apoplexy. We now come to the most difficult part of our subject—Diagnosis. Under this head we speak first of Premonitory Symptoms; next of Modes

of Onset, under which will be considered the comparatively simple cases of patients who have hemiplegic symptoms; and lastly, under Special Diagnosis, of the condition of those apoplectic patients in whom we can discover no hemiplegia. The separate consideration of those cases of Cerebral Hemorrhage in which there is, and of those in which there is not hemiplegia, is justified by convenience; for practically they are different things. When called to a patient who is hemiplegic, whatever other difficulties we have, we are certain, since there is clearly a *local* lesion of some kind, that there is not only drunkenness, uræmia, poisoning, &c.; but if we make out no hemiplegia, we may be in doubt whether the apoplexy be owing to a very large clot in the brain, to a very central one as in the pons, or to some one of the more general conditions mentioned.

Premonitory Symptoms.—The general bodily condition already described (p. 906 et seq.) furnishes the best basis for premonition, and in no nervous affection in people of, at least, middle life, however trivial the symptoms may be, do we neglect to examine the heart, arteries, and urine; but that condition leads to, or is associated with, disease of many parts of the body. We here speak of slight *nervous* symptoms which point more expressly to the future occurrence of Cerebral Hemorrhage, and which, when occurring in a patient who has degenerated arteries, hypertrophy of the left ventricle of the heart, and chronic renal disease, show it to be very likely that this unsound state in his particular case is about to lead to decided hemiplegia, or fatal apoplexy, from the rupture of vessels in the nervous centres. Some of these patients, however, die after having had slight warning symptoms—some evidently due to hemorrhage, for we may have seen clots in retinæ and have heard of epistaxis—in other modes, as by pericarditis, uræmia, &c.

Before we speak of special premonitory symptoms, we must remark that some patients who die of Cerebral Hemorrhage have had none, at least we hear of none; and this is sometimes the case when examination both during life and after death reveals signs of most extreme degeneration.

The degeneration of arteries of which we have spoken (p. 907) leads to two pathological states of nervous centres; to softening by thrombosis, and to hemorrhage by rupture. The slight symptoms of which we are about to speak as premonitory of Cerebral Hemorrhage may depend on either; practically it matters little, since the symptoms to be mentioned may be taken as warnings of the possible supervention of *large* Cerebral Hemorrhage, whether they signify small hemorrhages or very limited softening. It may, however, be denied that there is any local lesion when such slight symptoms as those I have to mention pass away altogether in a few hours or days. Besides the reply that local symptoms of necessity imply local lesion, it may be added that it is quite certain that even decided hemiplegia, whether from clot or from softening, will pass away even when, as subsequent post-mortem examination shows, there remains a permanent, although a small, void in the motor tract.

One general remark may be made: the premonitory symptoms of Cerebral Hemorrhage are owing to affections of nervous centres, and not of nerve-trunks. In cases of Apoplexy from rupture of aneurisms of the larger cerebral arteries, there may have been palsy of a nerve-trunk—third especially—from compression of that nerve-trunk by the aneurism; but rupture of such aneurisms, with very rare exceptions, leads to meningeal, not to cerebral hemorrhage. There are, however, some seeming exceptions to the rule laid down. We may have palsy of the tongue from hemorrhage into the medulla oblongata, and palsy of parts supplied by the facial nerve from thrombosis or small clot in the pons;[1] but these are, as stated, only seemingly exceptional. They are *not* owing to affections of nerve-trunks; and practically, when we are consulted for palsy of any cranial nerve, we do not attach much importance to it as a warning of Cerebral Hemorrhage, we think of syphilis, and, if the nerve palsied be one of those to the muscles of the eyeball, of locomotor ataxy also.

So far negatively. The symptoms which are premonitory of Cerebral Hemorrhage are innumerable. We may divide them into two classes, local and general. The local ones to be mentioned are:

Defect of Sight[2] occurs now and then before Cerebral Hemorrhage. In these cases we find mostly the degeneration of the retinæ which occurs with Bright's disease, and usually linear clots are to be seen in the retinæ too; indeed it is an affection of a nerve centre,[3] and of one

[1] Dr. Moxon has recorded (Path. Soc. Transactions, 1869–70) a case of paralysis of the portio dura nerve from hemorrhage into the aqueductus Fallopii.

[2] I do not know that deafness is of value as a warning of Cerebral Hemorrhage; it is a rare symptom in cases of serious brain disease of any kind.

[3] In many of these cases of retinal hemorrhages there has been nasal hemorrhage also. Epistaxis, however unimportant in itself, is a serious warning if there be chronic Bright's disease. It is of very great importance to use the ophthalmoscope in all cases of brain disease. There are often changes significant of Bright's disease, so well marked that the

supplied by the same arterial system of the brain, and is not owing to affection of a nerve-trunk. The existence of these changes makes us take a very gloomy view of the case of a patient who has even the slightest nervous symptoms.[1] If we find either optic neuritis or any kind of optic atrophy, we cannot infer liability to Cerebral Hemorrhage unless we know that the atrophy has followed the neuro-retinitis of Bright's disease. Indeed optic neuritis (there being no albuminuria) is rarely associated in any way with Cerebral Hemorrhage. It rarely precedes, and it very rarely follows it. As, however, optic neuritis is frequently associated, especially in young persons, with tumor of the brain, there is to be considered the liability to hemorrhage from tumor; but this is of very rare occurrence.

Limited Facial Palsy.[2]—This is really a part of an attack of hemiplegia. It is the kind of facial palsy which occurs in hemiplegia which is so common in disease of the higher motor track. (See p. 913.) We must observe, however, that a facial palsy of exactly the same kind occurs after certain epileptiform seizures; but in these cases there is occasional spasm of the paralyzed part. The patient is usually young, and we have often a history of syphilis. The facial palsy which is a warning of Cerebral Hemorrhage comes on suddenly without spasm, or is found on waking, and usually passes off in a few days. It is a very unfavorable sign, because it shows *central* disease.

Speech.—There may be *loss* of speech with the above-mentioned kind of facial palsy, but more often there is *defect* of speech only; a difficulty of articulation for which the degree of palsy of the face and tongue does not account; and we find that the patient writes, that is expresses himself in writing, about as badly as he talks. Again, there is central disease. Speech defects are not of special value as warnings of Cerebral Hemorrhage unless they come on suddenly. We must also bear in mind that temporary loss of speech occurs from embolism; at all events it occurs in young patients who have valvular disease of the heart. Occasional mistakes in words occur in many presumably healthy people, and their significance as evidence of coming cerebral disease of any kind is, I think, overrated. Suddenly occurring difficulty of articulation is the condition of most evil import.

Slight weakness or numbness of one arm and leg points to a local and central lesion, although to a minute one; but many people are subject for years to a slight numbness and to queer feelings on one side, who seem to be otherwise in good health, or the symptom obtrudes itself when they are slightly out of health—dyspeptic, for instance. It is not uncommon in young and apparently healthy people. A slight weakness of one side is only of value as a warning of Cerebral Hemorrhage when it occurs suddenly without spasm, and even then we do not attach great importance to it unless the patient be past middle age and show signs of degeneration.

Successions of slight local symptoms are of more value than any single symptoms. From syphilis also there are successions of nervous symptoms: they are such as palsies of cranial nerves, optic neuritis, partial convulsion. But preceding fatal Cerebral Hemorrhages we may find epistaxis; defect of sight from degeneration of, often with clots in, the retinæ; sudden numbness or weakness on one side (without spasm); occasional difficulty of articulation, and drawing of the face. These are due to affections of nerve-centres; or the patient may have many epileptiform paroxysms of very different kinds, sometimes slight without loss of consciousness, sometimes severe with tongue-biting, sometimes local, sometimes general. Here we suspect small meningeal hemorrhages.

General Premonitory Symptoms. — A page might be filled by the enumeration of symptoms of this class which authors give as warnings of Cerebral Hemorrhage. They are such as drowsiness, loss of memory, especially for recent events, irritability of temper. Such symptoms point only to general deterioration of brain, to slow wasting, for instance—and do not point especially to a liability to Cerebral Hemorrhage. More valuable symptoms of this class are giddiness, pain in the head, and vomiting. But these again may be found in the onset of many kinds of brain disease; for instance, in young people the subjects of cerebral tumor: indeed, if the headache be intense and continued for weeks, and if the vomiting be urgent, it is, provided there be no albuminuria, likely that there is tumor. If, however, the symptoms come on suddenly in a person of middle age, especially if there be slight confusion of mind at the time, and above all if there be any paralytic symptom, however faintly marked, such as thick speech or unilateral weakness, they may be taken as warnings of

ophthalmic surgeon is the first to discover that the patient has Bright's disease. *There may be no impairment of sight* when the ophthalmoscopical appearances are extremely well marked. This is to be strongly insisted on.

[1] I may here refer to a record of several cases in a lecture on Cerebral Hemorrhage (London Hosp. Reports, vol. iii. 1866). In some cases of coma the detection of these changes may enable us to make the diagnosis of Cerebral Hemorrhage.

[2] See Trousseau (Bazire's Trans.), vol. i. lect. 1.

Cerebral Hemorrhage. Doubtless they are owing to small clots or to limited thrombosis. If there be albuminuria, they are often ascribed to uræmia. Patients with chronic Bright's disease are prone to attacks of headache and vomiting, especially on getting up in the morning. Even if these symptoms are dependent on uræmia, they may still be considered as warnings — indirect, it is true — of the possible future advent of Cerebral Hemorrhage, when they occur in a person past middle age who has tough arteries, hypertrophy of the left ventricle of the heart, and no notable dropsy with his albuminuria.

The Mode of Onset of Cerebral Hemorrhage.—This is often the only diagnostic evidence of value, and in many cases when it is not forthcoming we cannot make a diagnosis at all—as when a patient is found by the police in the streets "drunk and incapable."

When we consider that the clot differs in seat, in size, and rapidity of effusion, we cannot *à priori* expect any great uniformity of manner of onset; as a matter of fact it varies greatly indeed. A patient may not be unconscious from a hemorrhage large enough to produce permanent hemiplegia, or he may, minutes or hours after being hemiplegic, become apoplectic, or he may become apoplectic almost without prior symptoms. I say *almost*, because however quickly Apoplexy from Cerebral Hemorrhage comes on, there are nearly always *some* prior symptoms. As Trousseau says, Apoplexy, in the classical sense of the word—a sudden falling — is rare in Cerebral Hemorrhage. There is nearly always *something* wrong before the patient becomes unconscious, and often the interval is considerable—minutes, or even hours. Trousseau excepts cases beginning by convulsion, and also cases of hemorrhage into the pons Varolii; but even in cases of hemorrhage into the pons, there are, I believe, mostly some symptoms before the loss of consciousness. Thus, I have notes of a case of a man who came off a scaffold because he was giddy before he became insensible, and of another patient who, when taking a drink, cried out that he was poisoned. Dr. Hare relates the case of a patient who was able to knock at a door and say she was going to die before she became insensible. In each of these cases there was large hemorrhage into the pons. In considering special modes of onset we shall give further illustrations. The first statement as to mode of onset is that Cerebral Hemorrhage, even when large enough to produce Apoplexy, does not as a rule cause Apoplexy instantly.

To consider mode of onset more particularly we must make a grouping of cases. The following, although in actual practice we see all degrees of intermediateness, is convenient.

Rapid Death.—This practically includes onset and termination. It is a rare thing for Cerebral Hemorrhage to cause rapid death; within half an hour, for instance. From theoretical considerations we might suppose that when the clot is effused near to the medulla oblongata—in the pons—death would occur very rapidly; but as a matter of fact it rarely does. Yet cases of sudden death are frequently put down to "Apoplexy," Cerebral Hemorrhage being meant. Since this term is often made to include meningeal hemorrhage, the statement is not altogether wrong, for meningeal hemorrhage may cause death in a few minutes—five, for instance, and probably in less time. Yet, since meningeal hemorrhage may lead to death very slowly, we have to infer that it is *rapidity* of effusion which is rapidly fatal, and not the position of the hemorrhage. If the patient dies rapidly, within half an hour let us say, *cerebral* hemorrhage is most unlikely. If he be young and healthy-looking, the probability is that there is *meningeal* hemorrhage from rupture of an aneurism of a large cerebral vessel. Usually there is a convulsion in these cases, and if death occurs "in a fit," or very quickly after, we still incline in a young person to the diagnosis of ruptured aneurism; but it is quite certain that in some cases of death in a first convulsion we discover nothing abnormal post-mortem. If death occurs instantly — the patient dying in a minute—we infer failure of the heart, rupture of aneurism of the aorta into the pericardium, or rupture of the heart itself. Death by intracranial hemorrhage is never so exceedingly rapid as it often is from these causes.

Convulsion.—This mode of onset has been considered in the article Convulsion, (p. 737). What further is to be said will find its place best when the cases of patients who are apoplectic without discoverable paralysis are spoken of. However, convulsion from clot is frequently followed by apoplexy with hemiplegia; such cases will be considered under the head of "Hemiplegia with loss of consciousness."

Hemiplegia without loss of Consciousness.—If we are called to a patient of, or past, middle age who is hemiplegic without loss of consciousness, we have to consider two possible kinds of lesions: softening from thrombosis;[1] clot from rupture of a vessel. In the great majority of cases hemiplegia without loss of consciousness is the result of local softening. I believe we can say very little more than this, for a

[1] For further points in diagnosis, and especially for the diagnosis of softening from embolism, see art. Softening.

clot occasionally causes permanent hemiplegia without producing loss of consciousness at its irruption. However, a very deliberate mode of onset strongly favors the diagnosis of softening. If, for instance, a patient, when he gets up in the morning, finds his arm weak, next his leg numb, and half an hour later is paralyzed on one side, little or much, we diagnose softening. What has been said before as to constitutional state—or as to premonitory symptoms—helps us but little in this difficulty, for degeneration of arteries leads either to softening or to clot, and any local premonitory symptoms the patient may have had may have been the result either of thrombosis or of rupture of small arteries. Still, the existence of chronic Bright's disease is much in favor of clot; and if we see clots in the retinæ and hear that the patient has epistaxis, we are warranted in inferring from these visible hemorrhages that the encephalic lesion is also hemorrhagic.

When the hemiplegia is on the right side, and now and then, but very rarely, when on the left, there is loss or defect of speech. This furnishes no further diagnostic evidence. I think, however, that frequent mistakes in words during recovery in young[1] people favors the diagnosis of plugging of vessels.

Hemiplegia with loss of Consciousness.—Hemiplegia with deep loss of consciousness (Apoplexy) is nearly always owing to Cerebral Hemorrhage. These cases are therefore more important than any other, and we must consider the mode of onset in some detail. The attack may begin either by special nervous symptoms, such as one-sided numbness, loss of speech, defect of speech, or by such symptoms as pain in the head, vomiting, and confusion of mind, and, of course, it may begin by both sorts of symptoms at once. It may begin by convulsion. We here consider the special symptoms only; the general symptoms are of additional diagnostic value, but we could only repeat what has been said (p. 924) when speaking of them as premonitory symptoms.

When a patient has suddenly decided, although very slight, local palsy (for instance a little thickness of speech, a trifling drawing of the face, or loss of use of one arm, one-sided weakness, or even numbness), it is clear enough that he has some kind of *local* lesion of his nervous system. We should believe hemorrhage was that lesion if any one of these symptoms were followed quickly by deep loss of consciousness, or if after some deliberation, or even if after partial recovery, a convulsion occurred. If the patient be above middle age, if he have tough arteries, if there be albuminuria, we are almost certain that hemorrhage has occurred; and if, after such a mode of onset, hemiplegia is found with deep coma, we are practically quite certain. If the mode of onset has been by convulsion, we still think it most likely that there is hemorrhage, if there be decided, and especially if there be *complete* hemiplegia—complete *in range*, that is. (See list p. 911.[1])

We now consider the case of a hemiplegic patient when he is fully apoplectic. As before said, the degree of the coma in cases of Cerebral Hemorrhage varies; the deeper it is, the more is the diagnosis of hemorrhage warranted: but loss of consciousness, accompanied by stertor, slow pulse, lower temperature, is not diagnostic of Cerebral Hemorrhage. If we have no history of the mode of onset, or only that the patient was taken with a fit of convulsions, the first thing we do is to inquire for hemiplegic symptoms. While hemiplegia is certain evidence of the existence of local lesion, and with other circumstances of the existence of clot, we must not suppose that its absence negatives clot. (See p. 911.) Hence we have often difficulty in saying whether there is cerebral hemorrhage, or poisoning, or uræmia. We usually discover some kind of one-sided symptoms if we do not find definite hemiplegia; we find *some* difference in the two sides when we raise the arms and let them fall, and when we pinch the legs. There may be spasm, or there occur occasional waves of tremor down one side; or we may find both eyes or the head turned strongly to *one side*. These symptoms point decisively to a one-sided lesion at all events; and when there is no history of injury, no evidence of embolism,[2] they mostly, in persons past middle age, signify clot. Yet there may be meningeal hemorrhage, and if the limbs of one side be continuously rigid, with or without occasional higher waves of rigidity, the probability is that there is meningeal hemorrhage, although perhaps cerebral as well. However, and this really is the important matter, very deep coma occurring suddenly or quickly with *one-sided* symptoms of any kind, point at least to intracranial, if not to cerebral hemorrhage, in the vast majority of cases of patients past middle age.

[1] Plugging of cerebral arteries in older people is not the same thing as plugging of cerebral arteries in young patients, as the vascular condition of the brain is different at different ages. There is in older people less free anastomosis from obliteration of capillaries, and also from the atheromatous condition of small arteries. The vessels of the optic disk become fewer in number as age advances.

[1] See, however, art. Convulsion, p. 753, "Epileptic Hemiplegia."

[2] See art. Softening.

Special Diagnosis.

Apoplexy without Local Paralysis. — When there are no local symptoms in the apoplectic condition, no hemiplegia for instance, it is most difficult to make a diagnosis. We shall here discuss only the difficulties we most frequently encounter. We shall suppose that we are called to a case of coma, and try to show by what means we may arrive at the diagnosis of Cerebral Hemorrhage. We often cannot; but even then we may be able, at all events, to decide whether there is a fatal lesion, or the comparatively minor condition, drunkenness, and to exclude violence and poisoning. Speaking generally, the difficulty is to determine whether a patient is suffering from *local* lesion so large and sudden, or placed so centrally (as in the pons), as to produce coma with universal powerlessness, or whether he is suffering from some condition such as uræmia, poisoning by opium, drunkenness, &c., which, as it were, imitate the effects of the grave local lesions mentioned.

It is important to bear in mind that we may have combinations of states. I have known an "epileptic" fracture his skull by a fall in a fit, and die from hemorrhage the result of rupture of the middle meningeal artery. A drunken man may have been struck on the head. A drunken man falls like a log, and a seemingly slight blow on the curbstone, for instance, will lead to hemorrhage into the arachnoid cavity. I have seen two cases of this kind in drunken people. Prescott Hewett says that extravasation of blood in the arachnoid cavity is much commoner than is usually supposed; that the injury causing such extravasation is often a trifling one; that it may occur without any apparent lesion of the brain or membranes. Mr. Stephen Mackenzie has known a patient who died of garroting to be treated for alcoholic poisoning; the patient *was* drunk when attacked. If the patient be often drunk, a fit of drunkenness may not improbably coincide with, and perhaps be the direct cause of, rupture of cerebral arteries.

But the difficulty is not nearly so great practically as it is logically; for when we know the constitutional history of an apoplectic patient (see p. 906), and if we are told, as we mostly are, the mode of onset (see p. 925), we are very rarely in doubt as to the cause of Apoplexy. We may know that the patient has had an attack of hemiplegia or some other paralytic symptom (see p. 923) before; and this will favor very strongly the diagnosis of hemorrhage. Then cases of Apoplexy without hemiplegia or without some hemiplegic symptom are comparatively rare.

In most cases the patient becomes ill, at home among his friends, or at his work, or he is found comatose in bed, and in the great majority of instances the *circumstances* negative drunkenness, poisoning, violence, and the like, when the symptoms do not. Indeed, in most cases the diagnosis is really easy; or, to speak strictly, the prediction is usually verified. Suppose, however, there is no history. Suppose the patient, as is pretty often the case in hospital practice, is found in the streets universally powerless and deeply comatose, we very often *cannot tell from what he is suffering.* Or let us suppose we are called to a guest at an inn, who is found comatose in bed or in the water-closet—the discovery of an empty laudanum bottle may be the only clue to the nature of the case. In such cases we can only say there is apoplexy; we cannot declare the cause of it, and simply because there is not evidence. I would most earnestly beg young practitioners not to trust blindly to the fact that the patient is found at the bottom of a scaffold in the diagnosis of injury, nor to the smell of drink, nor to an "uproarious condition," for the diagnosis of intoxication; and, above all, not to conclude, from bitten tongue, that the patient has "only had an epileptic fit." It is true enough that if he be led entirely by these circumstances, he will *mostly* be right, but he must run no risk of being wrong. Most painful mistakes are occasionally made because a practitioner concludes from insufficient evidence.

Having first examined the apoplectic patient for hemiplegia (the existence of which we are now supposing that we cannot determine), we next inquire for convulsions. If we obtain no history of a convulsion, we search for evidence of tongue-biting. However, we cannot often get a look at the tongue, but we may judge by the presence of blood on the gums or by bloody foam. If the foam be very frothy in large bubbles, it no doubt comes from the bronchial tubes, and is no evidence of tongue-biting. The tongue may have been bitten but not lacerated. And when we do find evidence of convulsion, we must remember that to use the words of Gull,[1] "*general* convulsion with insensibility is in itself of *little value* in the diagnosis of *any* brain disease." (The italics are mine.) If we feel sure that there has been a convulsion either before or after the onset of the symptoms, we can only exclude drunkenness and poisoning. If there be no convulsion, we have still these two causes to consider. We next examine the urine for albumen. We speak first of the comatose patient who is not

[1] Abscess of Brain, Guy's Hosp. Reports, vol. iii. 3d Series.

hemiplegic and who has had no convulsion, so far as we can tell, and whose urine is not albuminous. The first question is—

Drunkenness.[1]—The smell of drink must only lead us to a very careful examination for evidence of drunkenness, as patients who suffer Cerebral Hemorrhage may have been drinking, or may have taken spirits for premonitory symptoms. Oddly enough, patients soundly drunk, their real condition not being recognized, are now and then treated by doses of brandy-and-water. This shows in another way the difficulties of diagnosis. A drunken man may be in one of two conditions. (1) He may be insensible without excitement; he may, indeed, be as deeply comatose as if he had extensive and fatal Cerebral Hemorrhage. This is so when the patient has been "sucking the monkey," *i. e.* sucking raw spirits out of a cask by aid of a gas piping, or when he has drunk off a large quantity of spirits for a wager or out of bravado. In these cases, from the condition of the patient alone we cannot make a diagnosis, although, fortunately, it is usually made for us by the history. If we hear that the insensibility came on very slowly while the patient was drinking, especially if it were preceded by excitement of talk or manner, we should suppose we had to do with drunkenness. If, however, the insensibility began suddenly, or if there were a sudden increase of stupidity, or if the patient all at once staggered and fell insensible, cerebral or meningeal hemorrhage is almost as likely. Let us now suppose there is no history of the mode of onset, the patient being found in the streets by the police. We try to rouse him, and we may get him to give his name or his address. This is, perhaps, some evidence that the case is not one of Cerebral Hemorrhage, but it had better be disregarded, as patients comatose from fatal cerebral lesions of several kinds can be roused so far. That he resists our endeavors to examine him, or swears when roused, is of no value at all as excluding fatal lesion of the brain. The patient may vomit (as he may in Cerebral Hemorrhage), and the vomit may reveal the nature of the case. If he does not we are justified, in doubtful cases, in using the stomach-pump. Then, the drunken patient oftener passes his urine and feces than do other apoplectic patients. Again, we may find alcohol in the urine. The mere presence of alcohol in the urine is not to be relied on to show that the apoplectic patient is suffering from a *poisonous* dose of alcohol only. As before said, a drunken man may owe his coma, in part at least, to hemorrhage into the arachnoid cavity. However, Dr. Anstie tells me that it would be possible to recognize the presence of a *poisonous* dose of alcohol in the system if one drop of the urine itself, added to 15 minims of the chromic acid solution,[1] turned the latter *immediately* to a bright *emerald* green.

(2) The other condition is one of excitement, of which there are all degrees; as we have seen, the patient, who when left to himself is insensible, may be roused to resist and to swear, but the main features of a case to which we are called may be one of "uproariousness." If the patient be violent, and struggle, he is probably drunk. A cautious man will still continue his examination for other causes, because it is certain that after severe and fatal[2] injuries to the head the patient may struggle and swear, and even, as I saw in one of Mr. Hutchinson's cases, make replies as definite as "What's that to you about my tongue?" when asked to put his tongue out. I have recorded a case supplied to me by Mr. Stephen Mackenzie,[3] in which violence and swearing were the striking symptoms in a case of death from meningeal hemorrhage. As in this case, we have often a history of a mode of onset under circumstances which exclude the diagnosis of drunkenness. But to make a diagnosis from the condition of the patient only is quite a different thing. We can only make a diagnosis by exclusion, and the most important thing is to exclude injury to the head. The young practi-

[1] Here I would refer to papers on Alcohol Poisoning by my colleague, Dr. Bathurst Woodman, in the *Medical Mirror*, July, 1865, and February, 1866. Dr. Woodman has had an unusually large experience of cases of apoplexy from numerous causes, and to him I have to acknowledge myself greatly indebted for facts serving in the diagnosis of causes of coma. I have recorded (Lond. Hosp. Rep., vol. i. p. 35, from notes by Dr. Woodman) a case of death by hemorrhage in the pons, in which, when the patient was first seen, the symptoms were like those in some cases of deep drunkenness. He could move all his limbs, put out his tongue when asked, and, although insensible, was roused by shouting to answer, "What's that to you?" when asked his name. He had been found in the street by a policeman.

[1] The chromic acid solution is made by dissolving one part of bichromate of potash in three hundred parts by weight of strong sulphuric acid. Of course Dr. Anstie does not represent this test as a certain one for alcohol, but there is not likely, he tells me—and his experience on this point is very great indeed—to be any practical objection to the conclusion when the reaction is so sudden and decided on the addition of but a drop of urine to the test solution.

[2] See Callender, St. Bartholomew's Hosp. Rep., vol. iii. p. 415, and especially Case 5 of his series of cases.

[3] Medical Times and Gazette, April 1, 1870.

tioner must not hastily conclude that a patient is "only drunk," even if he be only confused, or if he swears or is violent, or if he lies on his back insensible, growling or swearing if disturbed. If he does, I am quite certain that he will have now and then bitterly to regret trusting to such circumstances. To have said that a patient was "only drunk" when a post-mortem examination shows a fatal lesion of the brain is very painful to all concerned. Besides, deep intoxication is itself a serious matter.

Injury.—We need not speak of cases where there is a clear history of very severe injury, because then the diagnosis is made for us. However, when the diagnosis has blindly rested on the fact that the patient has been in the way of injury, it is sometimes wrong. Prescott Hewett says (op. cit.), "There is no doubt that many a case reported as one of traumatic effusion of blood in the brain was simply a case of apoplexy."[1] In all cases of coma we search for bruises on the head and face. We examine the ears for discharge of blood, watery fluid, or even brain matter; the face for evidence of palsy of muscles supplied by the portio dura nerve—two things the frequent result of fracture of the base. We must be especially careful to note the condition of the conjunctivæ and eyelids, as effusion of blood here coming on *after* the injury, or after the patient was comatose, is evidence of fracture of the orbital plates. (By itself this is not, my colleague Mr. Hutchinson teaches, a serious symptom.) The absence of external signs of injury unfortunately does not negative serious and fatal injury to the brain. As before said, a slight fall may cause hemorrhage into the arachnoid "cavity;" the heavy fall of a drunken man, or a fall in an epileptic fit. Even in cases of bruising and laceration of the cerebellum, the accident is not, Prescott Hewett says, always severe. In several cases the cerebellum was thus injured by the patient falling in the street when drunk.[2]

Even if we hear only that the patient has been in the way of injury some time before the symptoms set in, we must still consider the possibility of injury, as symptoms due to traumatic effusion of blood on the surface of the brain, especially if it be betwixt the dura mater and the bone, may come on, or at least develop largely, especially by a convulsion, sometimes hours or days after an apparently trifling injury.

If there be hemiplegia immediately after a fall, especially if the palsy does not follow a convulsion, non-traumatic hemorrhage is most likely. Yet it is not quite certain, for there may be laceration of the hemisphere.

Opium Poisoning.—In both poisoning by opium and *large* hemorrhage, especially into the pons Varolii, there may be minute contraction of the pupils, universal powerlessness, and deep coma. "Contraction of the pupils is the most constant of all the effects of opium."[1] Hence there are on record cases of hemorrhage into this part of the nervous system, mistaken for and treated as cases of opium poisoning. Unfortunately, there is not always minute contraction of the pupils in effusion into the pons, nor are they always contracted in opium poisoning. And in either condition, contracted pupils may dilate shortly before death[2]—"full active dilatation, which is uniformly observable when death (from opium) is imminent."[3]

When we learn that the symptoms set in when the patient was with his friends, we must bear in mind that he may have taken the poison half an hour or even an hour before. We may detect the odor of opium in his breath. If there be a convulsion at the outset or soon after—cases of children are not here spoken of—we may almost certainly decide that there is not poisoning. My friend Dr. William Proctor, of York, however, has supplied me with notes of a case of rapid death of a woman in convulsion, after taking six grains of morphia. Caspar[4] says, "There are fits of spasms extending even to general convulsions." Scoresby Jackson[5] says that occasionally convulsions precede death. But these accounts of the symptoms refer to cases of children as well as of adults. I think we may say that in an adult, a convulsion—a severe convulsion at least, and certainly if it markedly affects but one side of the body—especially at the beginning, or soon after the beginning of the attack, nearly always negatives opium poisoning.[6] If we hear that

[1] Holmes' Surgery, vol. ii. p. 265.
[2] Op. cit. p. 312.

[1] John Harley, The Old Vegetable Narcotics, p. 137.
[2] See Anstie, Stimulants and Narcotics.
[3] John Harley, op. cit. p. 138.
[4] Forensic Medicine, Syd. Soc. Translation, by Dr. Balfour, vol. ii. p. 63.
[5] Materia Medica, p. 330.
[6] It is right, however, to state that Tardieu describes one rare form of opium-poisoning which is not, so far as I can judge, to be distinguished from a case of large and rapid intracranial hemorrhage. "Dans la forme foudroyante l'ingestion du poison est presque immédiatement suivie d'un sommeil comateux que rien ne peut vaincre; la respiration est stertreuse et de cet état de narcotisme profond individus empoisonnés passent sans transition à la mort dans l'espace de trois quarts d'heure à une ou deux heures. Rarement celle-ci est precédée de quelques mouvements convulsifs. Une remarque est pourtant à faire dans cette forme, c'est que les pupilles sont constamment dilatées."

the onset of the symptoms was very gradual—there being no albuminuria—we think that the patient was poisoned. Coma from effusion of blood into the pons Varolii will, it is true (see p. 925), come on deliberately, but not so deliberately as opium poisoning. In hemorrhage the symptoms usually develop in a few minutes, or there is a sudden development of coma after slight symptoms. Soon after the poison has been taken the patient may be roused to give his name, but later he is in a state of as profound insensibility as clot ever produces. Moreover, the test is of little, if any, diagnostic value in cases of coma of any kind. If, however, we have no history, suppose the patient is found comatose in bed (we are supposing there are no local symptoms—such as palsy of the sixth nerve, turning of the two eyes or of the head to one side—that there is no convulsion), *we cannot make a diagnosis.* If the patient be a young adult, poisoning is probable; if past forty, apoplexy is more likely; and I know of nothing in the pulse, in the respiration, or in the condition of the skin, which is of certain diagnostic value. An extreme slowness of the pulse, thirty or forty in a minute for a long time—say an hour or more—is said to favor the diagnosis of poisoning. But the pulse is sometimes rapid in opium-poisoning.

If the patient were dead when we were consulted, we should think he had not died of opium-poisoning if death occurred in less than six hours.[1] Poisoning by opium proves fatal in from six to twelve hours (Taylor). Effusions of blood into the pons, extensive enough to cause deep coma, will kill at varying times, from a quarter of an hour, which is rare, to twelve hours or more. However, we often have exceptional cases. Dr. William Proctor, of York, has recorded a case in which an ounce of laudanum killed a woman fifty years of age in less than two hours.

We have next to exclude epilepsy (see arts. Epilepsy and Convulsion).

Uræmia.—We now suppose that we find albumen in the urine. We have many times insisted on the fact that patients who are prone to Cerebral Hemorrhage have frequently chronic renal disease. We cannot therefore logically attach much diagnostic importance to the mere presence of albumen in the urine. Practically it is not of value when the patient is past middle age, for his coma may be due either to uræmia or to Cerebral Hemorrhage; and this is so whether the illness begins by convulsion or not. However it begins, we are sure there is not uræmia *only*, if there be hemiplegia, for then, if the patient be past middle age, we are practically certain that there is clot. But we are now supposing there is no discoverable paralysis. There are two chief ways in which uræmic coma comes on, without convulsion and then usually slowly, or rapidly and with convulsion.

If the patient, known to be the subject of chronic Bright's disease, gradually becomes languid, and stupid, and as it were sleeps into coma, we may fairly diagnose uræmia. If the coma comes on suddenly, the diagnosis of clot is more likely; and if the coma be very deep, and the patient never moves nor can be roused to move any of his limbs, the clot is probably in the pons, especially if the pupils be minutely contracted. If, however, there is no history, we cannot tell when the coma is deep. I have known a patient found comatose in the street, from whose symptoms it would have been impossible to make a diagnosis betwixt clot in the pons (this was found *post mortem*), uræmia, and poisoning by opium.

Further, uræmia may begin suddenly, in the midst of seeming good health, by convulsion; but so may Cerebral Hemorrhage (hemisphere or pons); and, to make the matter more difficult, these are the cases of Cerebral Hemorrhage in which we often cannot make out any hemiplegia. If the convulsion were strictly limited to one side—most convulsions affect one side a little sooner and a little more than the other—I should for my part feel certain that there was not uræmia only, although of course we could not under these circumstances say there was Cerebral Hemorrhage. (See art. Convulsion, p. 759.)

Serous Apoplexy.—This term is rarely used nowadays. Most cases so called were doubtless cases of uræmia. Yet we occasionally hear of cases of death by Apoplexy ascribed to "effusion of serum on the brain." If there be Bright's disease and, inferentially, uræmia, this may not be an altogether inaccurate description; for Traube considers that uræmic symptoms are directly dependent on œdema of the brain. These are doubtless, when there is no renal disease, cases of what are here called "Simple Apoplexy." By this term is to be understood cases of Apoplexy in which no lesion is discovered; that is to say, no lesion which we can suppose to have been the cause of so dramatic a mode of dying. One reason why these cases are called serous is probably that there is not unfrequently found at the autopsy a large quantity of serum in the meshes of the pia mater. But this is rarely a chronic state of things, and so far from the fluid exercising pressure, it has simply been "effused" very gradually to take up the room vacated by wasting of the brain. This is seen strikingly in cases in which there is wasting of but one

[1] See, however, Tardieu, quoted in preceding footnote.

cerebral hemisphere; here the serum is "effused" on one side only.

Simple Apoplexy.—Now, supposing we have excluded drunkenness, injury, epilepsy, and uræmia, we have still to determine whether the case be not one which for want of better knowledge we can only name from its negative post-mortem appearances, Simple Apoplexy. We have already (art. Convulsion, p. 761), when speaking of patients dying after attacks of convulsion, stated that in some we find marked changes *post mortem*, and in others we discover nothing abnormal in any part of the body. But patients pass into deep coma when no convulsions have been observed, or after apparent recovery from a convulsive seizure. A patient, sometimes even a young man, quickly becomes apoplectic and dies in a few hours, and in the whole body we find nothing abnormal which can reasonably be supposed to have been the cause of the symptoms. This class of cases is well recognized. Dr. Todd[1] says, speaking both of delirium and coma, that "both these formidable states may take place in a brain which shall reveal on the minutest scrutiny no appreciable aberration from the natural standard." Dr. Wilks says in his Lectures on Pathology: "Occasionally you may be called to a case where the patient is insensible or suffering from apoplexy, and on examining the brain you find nothing. During the last two years I have seen two cases where the post-mortem revealed nothing." It seems certain that these patients die from the brain. At all events they die in the same way as patients do who die in coma from Cerebral Hemorrhage, and in such cases during life Cerebral Hemorrhage is frequently diagnosed. The post-mortem appearances of the heart and lungs are such as those we find in patients who have died with large cerebral hemorrhage.

I freely confess that I know of no rules by which to distinguish simple from sanguineous Apoplexy, or other forms of coma. We cannot rely on the kind of pulse, nor on the temperature, nor on the state of the pupils, nor on stertor. I have, in short, nothing to say of diagnosis here.

I have observed that some medical men seem, if I may use such an expression, to be disappointed in not finding in the head of a patient who has died in an apoplectic manner, anything which can be supposed to have given rise to his symptoms. In these cases the suspicion of poisoning will occur. Indeed, this possibility ought to be carefully considered. Yet this part of the question is legal rather than medical, and at an inquest we can assure the coroner—who, if he be a medical coroner, requires no strong assurance on that matter—that the profession recognizes such cases as cases of natural death.

Some of these cases are put down to congestion of the brain. But this conclusion is often drawn from the distension of the cerebral veins, which is a very common appearance in patients who have died rapidly from any cause; and in all cases, even in cases of death from hemorrhage, we find fulness of the veins in the occipital region.

For the diagnosis of Apoplexy from *Congestion of the Brain* and from *Sunstroke* see those articles.

Aneurism of the larger cerebral vessels. (See arts. Adventitious Products and Convulsion, p. 759.)—Cerebral aneurism has been incidentally considered in several parts of this article.

Hemorrhage from Tumors.—Occasionally fatal hemorrhage occurs from cerebral tumors. We can only make the diagnosis from the evidence supplied by a history of tumor of the brain (see art. Adventitious Products), and if there be no history we cannot make a diagnosis. If, however, in a young patient we discover double optic neuritis, we should suspect tumor.

Occasionally apoplectic symptoms come on suddenly from *Abscess of the Brain* (see art. Abscess of the Brain). We can only make the diagnosis from such facts as the history of a blow, presence of "puffy" tumor, disease of the ear, &c., and when these facts are not forthcoming we cannot make a diagnosis.

Prognosis.—Here we speak of Cerebral Hemorrhage only. It is again to be insisted on that Cerebral Hemorrhage is not a constant quantity; the clot varies in size, in suddenness of effusion, in position, and there are differences in the ages of the patients attacked, and in their constitutional condition. Obviously then we can only speak very generally on prognosis, and what would probably come under this head has been already in chief part considered. Thus, under Etiology, we pointed out that in many cases the constitutional condition of the patients who suffer Cerebral Hemorrhage is one of widespread degeneration. If therefore the symptoms which we attribute to Cerebral Hemorrhage be in themselves trifling and transitory (see Premonitory Symptoms, pp. 923–4), they are of very evil omen if the patient be past middle age, and if there be hypertrophy of the heart, degenerated arteries, and chronic renal disease. But here the evil omen is as to the future. We speak next of cases of larger hemorrhage, and of prognosis as to recovery from hemiplegia, or apoplexy, or, as is usually the case, from both. Of course the graver the lesion, the worse the prognosis. We estimate its gravity

[1] Nervous Diseases, chap. viii.

by the degree of the paralysis and by the degree of the apoplectic condition. Under Localization, p. 537, it was pointed out that the more *complete* the paralysis the graver the lesion. Thus if the patient has, besides palsy of the face, arm, and leg, lateral deviation of both eyes and of the head, the worse the prognosis; if he escape with his life, palsy of the face, arm, and leg will almost certainly remain. If the palsy be incomplete, the prognosis is less grave, both as to life and recovery from paralysis. But we cannot judge by the paralytic symptoms alone. The degree of the apoplectic condition is to be considered also, although it is usually greater in degree the more complete the palsy. The less, and the more transient, the loss of consciousness, the better the prognosis; the deeper the loss of consciousness, the worse the prognosis. The prognosis is very grave indeed if the patient, after being simply hemiplegic, becomes suddenly profoundly unconscious and universally powerless, and it is graver still if the change sets in by convulsion, for this mode of ingravescence points to rupture into the lateral ventricle. The other symptoms of the apoplectic condition are to be considered. The more the pulse, respiration, and temperature are implicated—either depressed in the first stage or raised in the second—the graver the prognosis. In other words, the more the automatic processes are involved, the worse the prognosis. We have seen (p. 911), that along with degrees of loss of consciousness there are in different cases all degrees of range of palsy, palsy of the most voluntary parts (face, arm, and leg), palsy of these and of more automatic parts (deviation of the eyeballs and head, &c.), and even palsy of the *most* automatic parts as evidenced by stertor and depression of pulse, respiration, and temperature.

When the patient has come round from the apoplectic condition, his condition varies. Since there are all degrees of gravity of the lesion, there are all degrees of the conditions left when the apoplectic symptoms have passed off. The deeper and the more continued the apoplexy has been, the worse the after condition of the patient is likely to be. He may be, especially when speechless, in a state of complete imbecility, lying in bed, taking no intelligent notice of what goes on, and passing urine and feces in bed. Although frequently he eats voraciously, he gets gradually thinner and often dies in a few weeks or months. In other and less severe cases, there is great defect of memory, especially for recent events, and great emotional instability; the patient is easily made to laugh or to shed tears, though he does not laugh with any healthy ring, and his crying is a blubbering, very painful to witness. There is also great irritability of temper and often a heedless selfishness; the patient's disposition, his friends tell us, is quite changed, his mental field is narrowed: he seems to care much for his own immediate wants, and cares little about his family or business concerns. In other cases there is little more than paralysis, although the patient's mental condition is not so good as before.

The palsy often diminishes, and improvement follows a certain order. The more automatic parts recover first. Thus the lateral deviation of the two eyes and the head usually passes away in a few hours or days. The leg is the next part to recover, although it rarely recovers completely after severe apoplexy, and the lingual and facial palsy diminish or pass away altogether. When rigidity of the limbs comes on, we fear no further improvement will follow. We may find the patient speechless[1] (aphasic), on recovery from the apoplectic condition, and he usually remains so if the apoplexy has been deep and continued.

If there has been no loss of consciousness, or only transient loss of memory, the patient has a good chance of recovering altogether from the paralysis and the affection of speech. But as we have seen (p. 925), it is not easy to be sure that hemiplegia without loss of consciousness is owing to clot. Most cases of this kind are owing to softening from embolism or thrombosis. Recovery from hemiplegia will occur from any kind of lesion if it be a small one. We can only judge by the early beginning of the recovery. If the patient begins to move the arm next day, he is likely to get well altogether. We cannot infer so much from early recovery of the leg, as this is very often not completely paralyzed at the outset, and we know that it frequently recovers when the arm remains much paralyzed.

TREATMENT.—The recovery of the patient, it is most probable, depends altogether on the quantity and seat of the hemorrhage. If the ventricles be opened, if there be a large clot in the pons, the patient will die. But, as in many cases we cannot be absolutely sure that there is any hemorrhage, we must treat the apoplexy (see arts. Softening and Renal Diseases). We must particularly bear in mind that if the cause of the apoplexy be alcoholic poisoning, recovery usually follows, even in very severe cases. When in doubt we should use the stomach pump.

There is unfortunately little to be done, in cases of large Cerebral Hemorrhage, and the chief thing is simply to keep the

[1] See art. Softening for an account of Aphasia.

patient quiet, especially when we see him soon after the attack. Rousing him may lead to such increase in the size of a clot in the brain that it breaks into the ventricle.

Bleeding used to be almost a routine practice. In this country it has fallen into disuse. Although I have observed very many cases of Cerebral Hemorrhage, not only in my own practice but in that of others, I have seen but one patient bled for it. I quote, however, part of what Niemeyer says on this point. It will be observed how carefully he tries to distinguish the cases in which bleeding is admissible from those in which it is hurtful: "If the impulse of the heart be strong, and its sound loud; if the pulse be regular, and no signs of commencing œdema of the lungs exist, we should bleed without delay. Local bleeding by leeches, behind the ears, or to the temples, or by cups to the back of the neck, cannot replace general bleeding, but may be used as adjuvants. If, on the contrary, the heart's impulse is weak, the pulse irregular, and rattling in the trachea has already begun, we may be almost certain that bleeding would only do harm, since the action of the heart, which is already weakened, would be still more impaired, and the amount of arterial blood going to the brain would thus be still more decreased. When the latter state occurs, the symptomatic indications require just the contrary treatment, in spite of the original disease being the same, and being due to the same causes. We must strive with all our skill, by the use of stimulants, to prevent paralysis of the heart. If we cannot give wine, ether, musk, &c., internally, we should apply large sinapisms to the chest and calves of the legs, rub the skin vigorously, sprinkle the breast with cold water, or drop melted sealing-wax on it."

It must be difficult to select the right time as well as the right case, as the pulse, respiration, and temperature are in very different conditions at different stages in the same patient (see p. 922). In the first stage the pulse may be very slow, and the temperature greatly reduced. We should rather give stimulants than bleed in this condition, but I think it is better not to do this, unless the pulse be very feeble, and the temperature much reduced; we may also apply mustard plasters to the calves. When the pulse and respiration become very quick, when there is evidence of engorgement of the lungs—as shown by the loud rattles we hear from œdema of the lungs—we might, from theoretical consideration, suppose that bleeding would be of service by relieving the venous system, which is evidently overcharged. But at this time the pulse is really feeble, and occasionally it is irregular.

It is, I think, good practice to give croton oil in either stage, unless the alteration of pulse, respiration, and temperature be extreme. In the second stage—the stage of reaction, and when the clot is producing a local encephalitis—it is well to apply cold to the head. Blisters relieve the severe headache in the cases of cerebral tumor (no doubt often one of the symptoms of a local encephalitis), and it is possible that blisters to the back of the neck, and behind the ears, are of service when the patient is recovering from the apoplexy, and has pains in the head.

Let us now suppose that the apoplexy is past, or that there has been no unconsciousness, or a very temporary confusion when a hemiplegia, indicating effusion of blood, came on. The more vividly we realize the fact, that a mass of blood is lying abroad in softened and torn nervous tissue, the less confident do we feel in our power to interfere for the patient's good. The feeling of helplessness is greatest, when we are looking at a clot lying in nervous tissue, *e. g.* in the retina. There is, to my knowledge, no treatment for effusion of blood in nervous tissue. There are no drugs which assist in the absorption of the clot. However, it is quite certain that some patients recover satisfactorily from hemiplegia, the result of Cerebral Hemorrhage. But recovery from hemiplegia will follow when damage to the motor tract remains. From not recognizing this fact, erroneous conclusions may be drawn as to the effects of remedies. We must particularly bear in mind that anæsthesia disappears or diminishes quickly when no drugs are given, and also that there is a natural order of recovery, as stated under Prognosis (p. 932), which probably is not interfered with by treatment.

We have still, as in many other diseases, to improve the general health. This is, however, not unfrequently, rather general disease, and the local lesion—let us say epistaxis, paralysis for a few days, or a day's thickness of speech—is sometimes a small matter in comparison with the state of the system of the patient who comes to us for such slight symptoms. The proper care of a patient who has a clot of blood in his brain, and who is liable to have further effusions, consists in attending to his diet, excretions, sleeping and exercise. Care in diet is especially important. In this connection we may quote what Niemeyer says (op. cit. vol. i. p. 314), under the head "hypertrophy of the heart." We have seen that in most cases of Cerebral Hemorrhage, there is cardiac hypertrophy, and in many cases the nervous symptoms, when the clot is small, may be almost unimportant in comparison with the unsound state of the sys-

tem: "Such patients must beware of immoderate eating and drinking, in order to avoid the plethora which, although but transient, always follows upon a free use of food or drink. How often does the long-threatening apoplexy set in in the midst of the plethora which has developed after a long and hearty meal! . . . In this connection I may mention an act of folly which I have often seen practised by tavern-keepers and itinerant wine dealers. The latter often suppose that, by a free use of water, they can counteract the pernicious influences to which they expose themselves, although it is evident that the plethora arising after a full meal would only be increased by an immoderate addition of fluid. Besides this, however, the patient must avoid all the causes which, independently of plethora, stimulate the action of the heart, and further distend the already overcharged arteries. Under this head come the use of stimulating drinks, mental excitement, and immoderate bodily exertion. Hot water must be included in this class, and there is no wonder that the use of the Karlsbad Sprudel should make victims every year who die of apoplexy."

ABSCESS OF THE BRAIN.

By William W. Gull, M.D., F.R.S., and Henry G. Sutton, M.B.

Abscess of the Brain is comparatively a rare disease, and it falls to the lot of no man to see a great many cases. We have collected seventy-six cases in all from various sources, and the details in this paper are based upon these records. Many of the cases have not before been published. We have arranged the different parts of this subject in the following order: A description of the various conditions that are known to give rise to cerebral abscess, the morbid anatomy, the symptoms, pathology, diagnosis, and treatment.

Suppurative inflammation of the brain may be caused by injury to the head, especially where the skull is fractured and the brain contused. Mr. Prescott Hewitt says: "All traumatic inflammation of the brain substance may end in suppuration and abscess."

Cerebral abscess may follow a penetrating wound of the brain substance, by a knife, by a splinter of wood, or by some sharp instrument being forced through the skull.

Abscess of the brain may follow a fracture of the skull where there is no displacement of the bone; acute suppurative inflammation of the membranes and brain substance being set up by the injury. In many cases, caused by fracture of the skull, the abscess in the brain is seated immediately under the injured bone, and close to the surface of the hemisphere. In others the abscess is not seated near the surface; for instance, a person may receive a fracture of the skull, symptoms of compression may set in, and the skull may be, in consequence, trephined; the portions of depressed bone may be removed, and the patient go out of the hospital apparently well. But after a few weeks or months, cerebral symptoms may again appear, and the patient may die; and the autopsy reveal an encysted abscess embedded in the substance of the brain, and seated at some distance from the surface.

Cerebral abscess may follow an injury to the skull, where there is no fracture of the latter, and with or even without a scalp wound. In such cases the injury excites inflammation and suppuration of the deploë of the bone, and the suppuration extends and involves the brain.

Cerebral abscess may follow contusion, or, as it is sometimes expressed, concussion of the brain, without there being any fracture or other discoverable injury to the skull. Mr. Prescott Hewett says that he has seen two cases of this kind, and the abscesses were large.[1]

This is a very important class of cases, for it probably embraces not a few of the so-called idiopathic abscesses of the brain.

In two of our cases, abscess was found in the brain, though in neither was there any evidence to show that the skull had been fractured or otherwise injured. With both patients the symptoms followed directly after the injury; one had a fit on the same day as the accident, and the other suffered from almost constant pain in the head for a fortnight after the accident, and was otherwise generally indisposed. The abscesses were encysted

[1] Holmes' Surgery, vol. ii. p. 185.

in both instances, and, during the time they were forming, there were symptoms indicative of cerebral disease, although, in the second case, the symptoms were, for a while, obscure. One patient died seven weeks, and the other three months after the accident.

Cases might be given to show that abscess may follow injury to the head, without any fracture or other discoverable injury to the skull; and the abscess may remain latent for months or even longer.

One of the commonest causes of cerebral abscess is disease of the internal ear. The clinical history of this class of cases is usually as follows: the patient has a discharge from the ear for some time—for months—and, in many cases, for years; the discharge being continuous or intermittent. It is common to hear it said that the discharge began in childhood, after an attack of measles, scarlatina, or smallpox; and since has returned, more or less. With the discharge there is often deafness and pain in the ear, but more often the patient makes no complaint of either. In some cases, the discharge is very offensive, and has been so for some time past. The extension of the disease to the brain is often very insidious. There may be no indications that the brain has become seriously involved until acute symptoms set in a few days before death. Very often the first sign is a great increase of the pain in the ear. The pain is often very severe, and comes on in paroxysms, so violent in some cases, that the sufferer screams with it. Occasionally the acute mischief in the brain is ushered in with rigors; at other times with nausea and vomiting.

Sometimes an epileptiform convulsion ushers in the acute symptoms, and a few days after this the convulsion is repeated, and followed by hemiplegia.

The accession of acute symptoms appears, in many cases, to correspond with the commencement of acute inflammatory softening, either primarily in healthy brain, or secondarily around an old abscess. Then the skin becomes hot, the pulse quick, tongue dry and parched; great prostration, drowsiness, and stupor set in—such symptoms as resemble continued fever, and have been mistaken for it in some cases. The discharge from the ear varies very much during the acute symptoms. It is common for it to subside, or even entirely to disappear.

Chronic changes, dependent upon diseases of the internal ear, may be insidiously going on in the brain substance, without there being any symptoms of cerebral disease.

Mr. Toynbee was of opinion that the inflammation extends to the brain, from the pus not escaping from the cavity of the tympanum externally. He says: "So long as there is a free exit for the discharge, I believe the disease rarely extends to the brain."[1] He also remarked: "In all fatal cases the discharge has been deprived of a free egress." Mr. Toynbee further stated, in cases where the disease attacks the mastoid cells in early life, the cerebrum is the part of the brain which is most likely to suffer, while in later periods of life the cerebellum is the part most generally affected. Long experience has clearly shown that, when disease of the internal ear has gone on for a long time, the temporal bone is very liable to become diseased. When the patient dies with cerebral symptoms, it is common to find caries of the petrous, or mastoid, portion of the temporal bone. It is also common to find suppurative inflammation of the dura mater covering the diseased bone, with or without sloughing of that membrane. There is, in some cases, no direct extension of the disease from the bone to the contiguous parts. In such cases the bone, membranes, and surface of the brain are healthy. A portion of healthy brain may lie between the abscess and the bone. The diseased action is considered to extend by a vein. It is rare to find abscess of the brain following acute disease of the ear; but one case is alluded to by Mr. Toynbee.

In cases of chronic disease of the ear, the causes of the acute brain mischief are various. A blow on the head, violent exercise, or other depressing influence; also cold air, or some irritating application, is sufficient to engraft acute changes upon the chronic disease.

Cerebral abscess may be associated with, and apparently dependent upon, chronic disease in the lungs, but in two of our cases the morbid appearances were such as to indicate acute changes in the lungs, extending, however, over several weeks.

In a case that occurred in St. Bartholomew's Hospital, the lung presented the appearance of acute pneumonia in the third stage; but the symptoms indicated that the disease had been going on about two months and ten days.

In all the other cases which have come under our notice, the morbid changes in the chest had evidently been going on several months and even years. In one, there was a large suppurating chronic empyema. In another, there was a large cavity at the apex of the right lung, which was firmly adherent to the chest walls by a thick layer of indurated tissue. Another patient had had flattening and general contraction of the left chest for years, signs of dilated bronchial tubes, and of disease in the left lung.

Suppuration in any part of the body

[1] *Vide* Diseases of the Ear, by Mr. Toynbee, p. 303.

may give rise to secondary abscess in the brain. In one of our cases there was an abscess in the sheath of the left rectus abdominus muscle, and several abscesses without cyst in the brain. In this case it is instructive to notice that the lungs, the common seat of pyæmic abscesses, did not contain any abscesses, nor were there any in the liver or spleen. In another case there were pyæmic abscesses in the brain, apparently the result of chronic suppuration of a mesenteric gland and coexisting recent abscesses in the spleen and kidney. In a case of acute necrosis of the tibia, which occurred in St. Thomas's Hospital, there were numerous abscesses in the brain, and pyæmic abscess in the lungs, liver, and spleen. In a case given by Dr. Bright, a whitlow was the source of general pyæmia and abscess of the brain. In another case, referred to by Lebert, the drawing of a tooth was followed by inflammation of the upper part of the face and cerebral abscess. Dysentery was the cause in one instance; abscess near the uterus; suppuration in the Fallopian tube; carcinoma of the face; abscess in the liver, and the phagedenic ulceration, following amputation of the breast, were the causes in other cases. Dr. Ogle relates a case of secondary purulent deposit in the brain, apparently the result of ulceration of the cæcal appendage. There is also another recorded case following amputation of the forearm.[1]

In chronic disease of the bones of the nose, and in cases of syphilitic disease of the bones of the skull, there is a liability to cerebral abscess.

Morbid Anatomy.—An abscess may form in any part of the brain. Usually it forms in the white substance, and when in the gray it is formed by extension from the white. The middle cerebral lobes are the most frequent seats of abscess. One hemisphere is as frequently attacked as the other. Of 80 cases, abscess was situated in the left hemisphere in 23, and in the right in 29. Practically, therefore, one hemisphere would appear to be as liable to be attacked as the other. In 12 cases abscess was situated in the middle lobe, but it is not stated in which hemisphere. The middle lobes were the seat of abscess in 23 out of 74 instances. Abscess was found in the cerebellum in 13 cases, in the pons Varolii twice, in the corpus striatum twice, in the optic thalamus twice. Abercrombie mentions an instance of abscess in the medulla oblongata. In several of the 74 cases the abscesses were multiple, and found in more than one part of the brain. The appearance of the abscess varies according to its duration. If it have been recently formed, the pus is not inclosed in a cyst, but directly surrounded by ragged suppurating brain tissue, and there is not a trace of lining membrane to the cavity. If the abscess have been formed some time, the pus is inclosed in a cyst of variable thickness. In very old abscesses the cyst wall has been found a quarter of an inch, or more, in thickness. When the abscess is a few weeks old, the cyst wall is usually a line or two in thickness. The wall of the cyst is formed of fibro-cellular elements, and, in some cases, well-formed spindle-shaped fibres are seen; in others the fibro-cellular tissue has undergone granular degeneration, and the fibre cells are very indistinct. The cyst, when of old date, may be divided into three parts—an outer layer, which is made up of loose, fine, fibrous tissue; a middle layer, which is firmer and more coarsely fibrous than the outer; and the inner surface of the cyst is formed by a smooth, pyogenic membrane, in which some small irregular dilated veins may be seen running in different directions.

In abscesses of recent formation, the pus is generally of a greenish hue, and may, or may not, have a disagreeable smell. In old abscesses, the pus is green, fetid, mucoid, and is decidedly alkaline. The pus removed from old abscesses, when placed under the microscope, shows few or no well-developed pus corpuscles; there is a large quantity of granular fat and granular matter without any nuclei.

There may be several encysted abscesses in the brain. In one of our cases there were no less than four; in another a large encysted abscess in each hemisphere.

The condition of the brain substance immediately around the abscess may vary very much; it has commonly undergone a process of softening. Rokitansky, speaking of recent abscess, says, round the abscess the brain substance is in a state of inflammation, producing red softening, yellow softening, and in more distant parts œdema of the brain tissue.

When a large abscess is situated in one of the hemispheres, the brain is often altered in shape; the convolutions being packed together and flattened; the hemispheres bulged at the side, and if the abscess be very large, the hemisphere containing it may feel more like a bag of pulpy thick fluid than solid brain substance. Collections of pus, in the hemispheres, tend to make their way towards, and discharge themselves into, the lateral ventricles, or on the surface of the brain.

[1] From analogy we should expect that an hydatid tumor, or a so-called strumous deposit in the brain, would cause abscess. We have, however, no record of such a case. Abscess is also said to have occurred when the carotid artery was tied. Probably it was softening of the brain, and not abscess.

Pus, like blood, may fill one lateral ventricle only, or escape into the ventricle on the opposite side. In abscesses, as in very vascular, soft, gliomatous tumors of the brain, hemorrhagic effusions are occasionally met with, and a coagulum of blood may be seen surrounded by pus.[1]

We have already stated that several abscesses may exist together in the brain; this is common when a patient has died of pyæmic cerebral abscess. In such cases every part of the brain may be studded with minute collections of pus; they may be found in the cerebrum, in the cerebellum, in the optic thalamus, in the corpus striatum, and pons Varolii. The size of these abscesses may vary from a pin's head to a hazel-nut, or even larger. They are usually situated near the surface of the brain. The cerebral substance around these pyæmic abscesses may be softened, at other times it is firm and comparatively healthy. When abscess of the brain is dependent upon disease of the internal ear, the morbid appearances are much as follows: the dura mater, situated over the diseased petrous or mastoid portion of the temporal bone, is often found highly congested, softened, and ulcerated; or of a dirty green color, and evidently sloughing, and the bone laid bare. In other cases the dura mater is simply thickened and covered with purulent lymph, and betwixt the dura mater and the bone there is often a collection of pus. The lateral sinuses are frequently involved and plugged, especially when there is disease of the mastoid cells; the sinus is often seen enveloped in pus and purulent lymph. The suppurative inflammation may extend along the internal jugular vein, and set up suppurative pleuritis and abscess in the lung.

In abscess of the brain due to disease of the ear, there is, in the majority of cases, caries of the temporal bone; the latter is seen of a dark color, with an irregular roughened surface. The abscess in the brain may have direct communication with the diseased bone, and the contents of the abscess make their way through the ulcerated openings in the dura and bone into the tympanum, and then escape through the perforated membrana tympani into the external meatus, thus constituting what has been termed "otorrhœa cerebralis." A similar communication and escape of the pus is said to have occurred in cases of abscess in the brain caused by diseased ethmoid bone. At other times there is no such direct communication, for there is a layer of brain substance separating the abscess from the membrane of the brain. This layer is often softened, of an ash-gray or yellowish appearance, and looking as if the pus were about to burst and discharge itself on the surface of the brain.

In some cases of abscess dependent on disease of the internal ear, there is no caries of the bone, as we have already mentioned, the membranes may be healthy, and the abscess may be situated at a distance greater or less from the surface of the brain.

Symptoms.—In 73 cases of abscess of the brain, the symptoms were as follows: —Pain in the head in 39 cases; epileptiform seizures in 38; coma in 30; heaviness, stupor, and drowsiness in 30; paralysis in 24; rigors in 17; pyrexia in 13; delirium in 13; vomiting in 12; incontinence of urine, or of feces, or both, in 15; vertigo in 8; disordered sensibility, not including pain in the head, in 6; defective articulation in 4; defective sight in 3; an apoplectic attack in 1.

That some of the symptoms may have existed in greater proportion, we should be prepared to expect, especially such symptoms as vertigo, pyrexia, emaciation, and probably in a greater number of cases, defect of sight would have been discovered had the eye been tested. The symptoms, therefore, that are most frequently observed in cases of abscess in the brain are pain in the head, epileptiform attacks, paralysis, coma, heaviness, drowsiness, stupor, rigors, pyrexia, delirium, vomiting, and incontinence of urine and feces. In a few cases defective articulation was met with. The records show that the intellect was very little affected. Paralysis was observed in 24, that is in about one-third, whereas in Lebert's cases it was observed in about one-half. He included, however, not only local paralysis, but also general loss of muscular power, whereas we have confined the term to local paralysis only, such as loss of power on one side of the body, of one arm or leg, one side of the face, or some other part.

The first symptom, in many cases, is pain in the head; it may be the only indication of cerebral disease present for months. The pain is often very agonizing.[1]

[1] See Guy's Hospital Reports, vol. iii. 3d Series, Case No. 6, p. 291.

[1] One patient lay in bed continuously holding his head with both his hands: another walked about with his hands pressed against one side of his head, crying out constantly, "Oh, my head! oh, my head!" The pain is often so severe that the patients shriek from the agony they suffer. A patient, who was perfectly sensible, said he could not help screaming; and, although he tore and bit anybody or anything near him, he at the same time expressed contrition for what he was doing, and said the pain in his head was unbearable; it felt as if some one was knocking it with a hammer.

An intense neuralgic pain situated over one spot is occasionally the first symptom; sometimes the pain is seated almost immediately over the region of the abscess. A boy, having an abscess in the anterior lobe of the right hemisphere, complained of almost constant burning pain over the front and right side of the head, but this localization of pain over the seat of the abscess is by no means constant. In some cases the pain is very remote. In one patient there was an abscess in the cerebellum, and the pain was felt in the forehead; in another there was an abscess in the right middle cerebral lobe, and the pain was referred to the left side of the head.

The pain often comes on in paroxysms; in other cases it is continuous, remittent, or intermittent. It is not present in all cases of cerebral abscess, as the statistics of our 76 cases show. It is very commonly associated with pain in the ear, when the abscess is due to disease of the auditory apparatus.

Instead of pain preceding, it may follow the convulsive attacks. Cases of this kind are by no means few.

Occasionally the first indication of cerebral mischief is a sudden and unexpected epileptiform seizure. The epileptiform seizures are occasionally the most prominent symptoms from the time of seizure to the patient's death. The epileptic attacks do not necessarily come on every day; occasionally some days elapse between the seizures.

After each convulsion the side affected is often left weak, and this increases until there is complete hemiplegia. The convulsive movements are sometimes unattended with insensibility, and are confined to one extremity, especially the arm. This has been long noticed.

Abercrombie alludes to a case of Lallemand's, in which there was pain in the right side of the head and tremor of the left arm. This was followed by continued convulsions, flexion, and extension of the left arm, which after some days ended in palsy.

Instead of convulsive movements, the first indications of brain disease may be numbness and tingling in one extremity.

The symptoms in other cases of cerebral abscess are like those that are said to indicate cerebral softening. There is sudden loss of power on one side of the body without any loss of consciousness; the leg being less affected than the arm.

In several instances rigors were very prominent symptoms throughout the attack. A patient, suffering from suppuration, was noticed to be getting thinner and weaker; then he was seized with rigors, diarrhœa, a dry brown parched tongue, and a hot skin; he became comatose and died. Pyæmic abscesses were discovered in the brain.

In some cases of pyæmic abscesses, there are no special symptoms to show that organic disease is going on in the brain; but only the general indications of pyæmia. In others the accession of convulsive seizures, paralysis, or coma indicates disease in the cerebral organ. Rigors were noticed in a few instances so severe, and returning with such regularity every day, that they closely resembled those of ague. One patient had headache, rigors, and vomiting, returning every day for five days, and then became unconscious. Rigors do not occur, in some instances, until after convulsive seizures have indicated cerebral mischief. Imperfect articulation, to a marked degree, was noticed in some cases, and in one there was loss of language.

With respect to the eye, Dr. Hughlings Jackson has mentioned to us that he has seen changes in the retina (optic neuritis?) in a case of cerebral abscess. Dr. Jackson thinks such changes are common to several kinds of cerebral disease.

Mental disturbances were observed in some cases. Now and then, the only symptoms noticed were a heavy expression, a disinclination to speak, and indifference to surrounding objects. In some cases with disease of the ear, it was stated that the patients had attempted to commit suicide. One patient appeared to become hypochondriacal. Emaciation setting in rapidly was a marked symptom in several cases. Similar emaciation is seen in some cases of tumor of the brain; but is not so frequent as in abscess.

Patients suffering from cerebral abscess may have symptoms so closely resembling continued fever, that it is exceedingly difficult, if not impossible with any degree of certainty, to say whether it be a case of fever or of organic disease of the brain.

PATHOLOGY.—Cerebral abscess may be produced by direct injury, or by contre-coup, contusing or lacerating the nervous tissue, and setting up inflammation and suppuration. It may be produced by suppurative inflammation in some tissue in the neighborhood of the brain which spreads to a contiguous part; namely, in the ear or nose, which extends, and invades the dura mater, pia mater, and brain substance. Or the diseased action may spread by continuity of structure, as along a vein, and thus to the brain. Disease of the ear or nose, or of other cranial bones, may give rise to cerebral abscess in this manner. Again, abscess may be produced where there is disease of the cranial bones, or some growth involving them, by the veins communicating with the diseased bone becoming plugged. The process of coagulation extends and invades the veins communicating with the sinuses of the dura mater. These become plugged,

as also the veins of the pia mater and probably some branches entering the brain tissue also, and inflammation, terminating in suppuration, is thence set up in the brain. In other cases, minute coagula, or thromboses, are supposed to be detached and carried along by the circulation until they are arrested in the capillaries of the brain, and often of the lungs, kidneys, and other organs.

Pyæmic abscesses are occasionally found in the brain, and not in any other organ of the body. Besides the coagula, some of the elements of pus may be carried by the circulation to aid in, or be the means of, setting up suppuration in the parts where the thrombosis is arrested. In this way abscesses in the brain are probably caused by abscess or suppuration in the liver, lungs, bowels, or in other parts.

We next inquire if every form of cerebral inflammation, or encephalitis, no matter what its origin, be liable to end in suppuration and an abscess. It has been many times stated that such is the case; but it would appear that the inflammation must be set up by a special cause, and unless it be so, it does not end in suppuration and abscess. Suppuration may apparently be excited by local injury, or by the elements of pus or thrombosis; but experience shows that other forms of inflammation do not terminate in abscess. For instance, encephalitis and softening, the result of plugging of a cerebral artery, or encephalitis around a hemorrhagic effusion, or around a gliomatous tumor or old cyst, shows no disposition to the formation of pus or abscess. The brain may soften, disintegrate, and a cyst may be formed, but there is no pus formed.

It is necessary, now, to ask if there be not good evidence to show that the brain may be the seat of suppurative inflammation and abscess without there being any cause to account for it? Is there not, in such cases, idiopathic inflammation which gives rise to idiopathic abscess? By idiopathic cerebral abscess, we suppose, is meant abscess which is not preceded or occasioned by injury or disease; its origin being unaccounted for. Lebert and others admit the occurrence of idiopathic cerebral abscess. Such cases are, however, in comparison with others, rare. It is beyond all doubt that a certain number of cases of cerebral abscess do occur in which no disease is discovered in any other part of the body, and there is no history of any recognized cause to account for the cerebral abscess.

Before, however, it be concluded that abscess has been formed idiopathically, it is necessary to remember that in the majority of cases there is a cause to account for the formation of such abscess, and that only in a very small minority have observers failed to find some admitted cause. In the face of such evidence, is there not good reason to think that, in this small minority of cases, the primary cause has been overlooked? And, when it is still further remembered that hours have been passed in searching for the primary disease or cause, and at last it has been found limited to a mesenteric gland, a gum-boil, or a whitlow—in fact the primary disease was so small, that it might have been very easily overlooked—it appears to us not difficult to understand how, even after very great care, the primary cause may have remained undiscovered. Bearing all this in mind, we recognize that in a few cases of cerebral abscess, the cause cannot be discovered; but even when the cause is undiscovered, we should not assume that the suppurative inflammation has commenced idiopathically in the brain.

Cerebral abscess proves fatal in many cases, not by a collection of pus in one or other part of the brain, but by extensive inflammatory softening around the abscess, involving vital parts of the brain; and it is from such softening that the abscess is enabled to make its way towards the ventricles or the surface of the brain. The softening around very old encysted abscess would appear not to be set up by pyogenic changes going on in its lining membrane, for there is not a large quantity of well-formed pus corpuscles in old encysted abscesses to show that such active changes have been going on in this membrane.

The softening would rather appear to be due to some circumstance interfering with the nutrition of the parts outside of the abscess, but in its neighborhood. The nutrition of such parts, owing to the presence of a foreign body, being very feeble, it is easy to understand how a blow on the head or a debilitated or cachectic state of the system may be sufficient to excite such feebly nourished parts to take on acute inflammatory softening.

Has abscess in the brain any tendency to spontaneous cure? Lebert thinks not; and when we remember that there is no well-established case on record, showing that an abscess has been spontaneously cured, we readily admit that the evidence very strongly favors the belief that cerebral abscesses do not tend to a spontaneous cure. It is, however, necessary to remember that the brain is a very vital organ, severely taxed in our every-day labors, and, if not sound, its functions, which are essential to life, may be brought to a stop. When there is an abscess in the brain, the organ being unsound, its functions are very liable to be perverted, and death follows; whereas, if the abscess were seated in an organ less essential to life, any perversion of its functional activity would not be attended with fatal results, and thus time would be

gained for the abscess to pass through the different stages essential for its cure. We may therefore ask ourselves whether it is that an abscess of the brain has no disposition to spontaneous cure, or whether it is that the patient does not live long enough for such a process to be accomplished? The development of a firm cyst wall would show that there is a possibility of spontaneous cure. The cyst wall exerts a protective influence, by localizing the mischief and protecting the sound from the diseased part. And experience has shown that time is only required for such protection to be very great, and for the barrier guarding the pus to become stronger and stronger.

We are next led to ask, is there anything in the condition of the pus discovered in old abscesses to show that these were in a process of cure? To our minds, there is. It is usual to find such pus in a very degenerate condition, viz. granular and fatty, which is favorable to absorption and concretion: such changes as occur in abscesses that have undergone spontaneous cure. This is no idle question. It is simply—Is cerebral abscess necessarily a fatal and incurable disease? Practically it is, but there is nothing in its morbid anatomy to lead us to conclude that it is necessarily incurable.

Diagnosis.—Cerebral abscess is inferred when there are symptoms of brain disturbance indicative of organic disease, and there are present those morbid conditions that are known to give rise to cerebral abscess, such as a discharge from the ear, nose, or chronic suppuration elsewhere, or when there is a history of a blow, or of some other acknowledged cause of the disease. No doubt that in some cases the inference proves correct, where there is evidence showing that the cerebral substance is undoubtedly diseased, and further evidence of suppuration going on in some part of the body; for here there are indications of acute brain disease, and we are led to suspect that this is due to abscess, since such causes are present as are known to produce it. With the brain, however, as with other organs, we are more often able to say that it is diseased than to say what is the precise nature of the pathological changes going on in its substance.

There may be evidence to show that a patient has chronic disease of the nose or ear, and cerebral symptoms may supervene suddenly; epileptiform seizures and other symptoms may be present, such as are seen in cases of cerebral abscess; the patient may die, and yet there may be no disease of the brain or of its membranes. In some cases, the membranes alone are diseased; in others, the brain substance is softened, without abscess. Disease of the bones of the skull—no matter whether it be fracture, syphilitic disease, or a growth—is liable to set up inflammation of the membranes of the brain, and the inflammation may spread, and give rise to suppurative inflammation of the brain substance. If the patient survive six or seven weeks, an abscess may be formed; if he die in two or three weeks after acute symptoms have set in, the brain may be found softened, but without abscess. Not unfrequently death takes place before there is time for the suppurative inflammation to form an abscess.

There may be a history of injury to the head, cerebral disease may appear to have followed as a consequence, and the post-mortem examination reveal disease in the brain, but not abscess.

Injury may be followed by the formation, not of an abscess, but of a tumor, malignant disease, or by softening in the brain; or further, the disease may not be in the brain at all, but on the surface. Experience has shown that an injury to the head may produce a large cyst in the cavity of the arachnoid, and the symptoms of the case may be similar to what are seen in cases of encysted abscess.

Cerebral symptoms associated with offensive discharge from the ear and nose, would lead one to suspect abscess in the brain, but in one of our cases there was tumor, and not abscess. The coexistence of tumor in the brain with the conditions that are known to produce abscess, makes the differential diagnosis extremely difficult. There are no pathognomonic symptoms of abscess or of tumor. It is only the different manner in which the symptoms are grouped, and the existence of those conditions that are known to produce one and not the other disease, which leads the practitioner to suspect that there may be tumor rather than abscess, or *vice versa*.

The symptoms of abscess may differ from those of tumor in the following respects. In abscess there is often marked cachexia and great emaciation. In tumor, the patients have often no marked cachexia, even look healthy, and the body is fairly nourished, certainly not emaciated. In abscess the duration of the cerebral symptoms is generally much shorter than in tumor. The symptoms in abscess are usually either latent or acute; in tumor they are often chronic. In the latter there may be local paralysis extending over several months, which is very rare in abscess. The intracranial nerves are much more frequently affected in tumor than in abscess. Occasionally, however, a person with tumor is seen to be much emaciated. These differences may enable the practitioner, in some cases, to diagnose

one condition from the other, but in neither case are these differences so constant that a certain diagnosis can be made.

An abscess may lie latent in the brain for many months, and then acute symptoms may suddenly set in, and the patient die in a few days. The same thing may take place with respect to cerebral tumor. Experience has shown that cancerous deposits also may exist in the brain without there being any decided cerebral symptoms.

Chronic encysted abscesses and tumors of the brain have many symptoms in common. A hydatid tumor, gliomatous tumor, a cyst, cancerous deposits in the brain, or any other substance acting as a foreign body, may produce pain in the head, epileptiform seizures, with or without paralysis, optic neuritis, vomiting, or gradual loss of muscular power.

We are often able to say, when there is acute persistent but variable paralysis, with pyrexia, that there is acute inflammatory softening of the brain; but whether that softening is going on around an abscess, a tumor, or a cyst, or whether excited by disease situated on the surface of the brain, we may be unable to give any exact opinion.

With respect to rigors in cases of cerebral abscess, we have already stated that they are very well marked in some instances, and may be not unlike those of ague. This symptom is not, however, peculiar to cerebral abscess. It occasionally occurs in other forms of brain disease, for instance, as gliomatous tumors or tubercle.

TREATMENT of abscess of the brain should be, by anticipation,—obviating the causes which lead to it; in chronic disease of the ear or nose, by maintaining a free exit for the discharge, no matter what the exciting cause. Rest is the most important part of the treatment, avoiding thereby both mental and mechanical excitement.

By a simple diet and quiet life, abscess may be dormant in the brain for an indefinite time.[1]

In cases where abscess follows injury to the head, surgical interference must be thought of. The principle in such cases is a mechanical one, namely, to reach the abscess and evacuate its contents, if that be thought advisable,—experience shows but little to commend it.[2]

---

[1] This is, however, to be observed, that encysted abscess of the brain is fatal from changes outside the cyst of an acute kind, such as might be presumed to be preventible to a great extent. In support of this opinion we may say that, in our experience, we have known abscess lie quiet for months after a blow on the head, and the patient and the medical attendant become confident that all was well; the symptoms of lesion having slowly gone off, and yet a fatal issue be produced after a few hours' suffering by neglecting the precaution of rest and regimen. Probably such rest and care should be continued, not for months only but for years. This we say from clinical observations of the changes in the cyst of old cerebral abscess.

[2] A series of statistical tables, giving the particulars of 76 cases of Abscess of the Brain, are omitted from this edition; for the reason that, valuable as they are in themselves, yet in proportion to the space they occupy, they are likely to be referred to in detail by very few readers. Their general history, and the lessons to be derived therefrom, are well stated in the preceding article.—H.]

B.—PARTIAL DISEASES OF THE NERVOUS SYSTEM.—
CONTINUED.

---

2. DISEASES OF THE SPINAL COLUMN.

MENINGITIS.
MYELITIS.
CONGESTION.
TETANUS.
LOCOMOTOR ATAXY.
IRRITATION.
GENERAL SPINAL PARALYSIS.
HYSTERICAL PARAPLEGIA.
REFLEX PARAPLEGIA.
INFANTILE PARALYSIS.
HEMORRHAGE.
NON-INFLAMMATORY SOFTENING.
INDURATION.
ATROPHY AND HYPERTROPHY.
TUMOR, ETC.
CONCUSSION.
COMPRESSION.
CARIES OF VERTEBRÆ.
SPINA BIFIDA.

DISEASES OF THE SPINAL CORD.

BY C. B. RADCLIFFE, M.D., F.R.C.P.

A. PRELIMINARY REMARKS.

BEFORE proceeding to cope with the intricate and difficult pathological topics which form the subject of the present article, it appears to be expedient to glance at some points in the physiology of the spinal cord, and also to try and ascertain the true significance of pain, spasm, and certain symptoms analogous to pain and spasm, which figure conspicuously in the histories of spinal maladies; for if these matters be not disposed of as preliminaries now, they will prove to be the cause of frequent and distracting digression afterwards.

I. A GLANCE AT SOME POINTS IN THE PHYSIOLOGY OF THE SPINAL CORD.

1. *Roots of spinal nerves.*—The result of recent researches has been to establish in the fullest manner the truth of Sir Charles Bell's great discovery, that the posterior roots of the spinal nerves are devoted to sensation only, and the anterior roots to motion only. In one article, at least, the creed of to-day is the same as that of yesterday: and it is some comfort to have it so, for in many other articles the creed of yesterday is not that of to-day.

2. *Posterior columns.*—If these columns be cut across, the result is, *not* numbness, as it would be if these columns were, as was once supposed, simply the continuation of the posterior roots of the spinal nerves, but hyperæsthesia and loss of co-ordinating power in the parts below the section, with a certain degree of local pain; and on inquiring further, it is found that this pain is due, not to any sensitiveness in the columns themselves, but to the irritation of the cut having travelled through the posterior roots of the spinal nerves, which posterior roots, as the researches of Lockhart Clarke show, pass through the posterior columns, more or less directly, to the central gray matter of the cord.

3. *Restiform bodies and small posterior pyramids.*—What has just been said of the posterior columns of the cord appears to apply equally to the restiform bodies, and to the small posterior pyramids of the medulla oblongata, which pyramids lie between the restiform bodies posteriorly. Hyperæsthesia and incoördination in the parts below the section, with some local pain, are still the result of cutting these parts across: and as the connections of these parts are above with the cerebellum, and below with the posterior columns of the cord, the natural inference is, that

the channel through which the cerebellum acts upon the body is formed of the restiform bodies and small posterior pyramids in the upper part of its course, and of the posterior columns of the cord in the lower part.

4. *Anterior columns.*—If one of these columns be cut across, it ceases to act on that side of the body in the parts below the section, and the paralyzed parts are benumbed to a certain degree, unless the cut be made in the part which lies immediately below the anterior pyramids of the medulla oblongata. In this part the anterior pyramid may be cut across without causing any very obvious paralysis or loss of sensation: in this part the results of dividing the anterior column at a lower level are only obtained when the cut is extended transversely, so as to divide the lateral column. It is plain, in fact, that in the uppermost part of their course, the anterior columns have not that intimate connection with the anterior roots of the spinal nerves, and that all-important part to play in voluntary movement, which they evidently have everywhere else: and it is also plain that the anterior columns, where they have to do with voluntary movement, have also something to do with sensation, for it is a fact that a certain degree of numbness is produced by the injuries which give rise to paralysis.

5. *Anterior pyramids.*—A transverse section of one of the anterior pyramids of the medulla oblongata in any part of its course annihilates all power of voluntary movement in the muscles below the section on the *opposite* side of the body, without affecting the sensation in any appreciable manner; and thus it is plain, not only that each pyramid contains very many, if not all, the conductors concerned in carrying the orders of the will to the muscles of the opposite side of the body, but also that the conductors which are collected in one pyramid decussate with those collected in the other pyramid at the lower and not at the upper boundaries of the pyramids. In a word, all the evidence, old and new, goes to show that these bodies are composed of conductors concerned in voluntary motion without any admixture of sensory conductors.

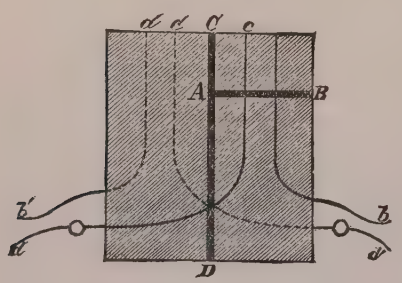
6. *Lateral columns.*—In the cervical region, for a short distance below the point at which the anterior pyramids of the medulla oblongata intercross, the lateral columns of the spinal cord have certainly very much to do in transmitting the orders of the will to the muscles; for, as has just been seen incidentally, the muscles below the section on the same side of the body are paralyzed by cutting one of them across in this part. In the lower part of the cervical region, and in the dorsal and lumbar regions, it is very different, and the difference is not very clearly determined. Here some trifling paralysis may be produced by dividing these columns transversely, but never more than this. Here, indeed, it would seem that this operation is followed by a certain degree of anæsthesia, and by the same result, as regards movement, as that which follows transverse division of the posterior columns—that is, not by paralysis, but by incoördination. A certain degree of anæsthesia appears to be a constant consequence of cutting across the lateral columns in any part of their course; and herein would seem to be an important distinction between the lateral and the posterior columns, for, as has been stated already, the result of cutting across the posterior columns is to produce hyperæsthesia, not anæsthesia.

7. *Olivary bodies.*—A section of the olivary bodies is followed, not by any marked degree of paralysis, or anæsthesia, but by a state of persistent spasm in many muscles on the *same* side of the body, in the neck especially,—a state which may sometimes continue for days, weeks, or even months. It is found, also, that this strange result is produced by irritating several parts of the base of the encephalon, the lateral and posterior parts of the medulla oblongata and pons Varolii especially, as well as by irritating the olivary bodies. These parts are not very clearly defined. "They seem," says Dr. Brown-Séquard, "to be quite different from those employed in the transmission of sensitive impressions, or of the orders of the will to the muscles, at least in the medulla oblongata and pons Varolii. They constitute a very large portion of these two organs, and, perhaps, as much as three-fourths of the one first named. They are placed chiefly in the lateral and posterior columns of these organs; and because many of their fibres do not decussate, the spasm produced by irritating them is on the *same* side of the body."

8. *Gray substance of the cord.*—Instead of being merely a nerve-centre—the special centre of Marshall Hall's excito-motor system of nerves—there is now reason to believe, with Dr. Brown-Séquard, that the gray substance of the spinal cord is an important conductor of sensory and motor impressions. Paralysis without loss of sensation on the same side of the body, loss of sensation without paralysis on the other side of the body, are the strange results of cutting across one lateral half of the gray substance of the spinal cord: anæsthesia on both sides of the body, paralysis on either side, are the equally strange results of making a longitudinal section midway between the two lateral halves: these are the two great facts which, when properly interpreted, furnish the reasons for believing, not only that there are sensorial and volitional conduc-

tors in the gray substance of the cord, but also that these two forms of conductors follow a different and definite course. Nor is it difficult to see how this may be. Let the course of the conductors in connection with the anterior and posterior roots of a pair of spinal nerves be what is represented in the following diagram,—*a b* being the motor conductor descending to the right, and *a′ b′* the corresponding conductor descending to the left; *c d* being the sensory conductor ascending from the left, and *c′ d′* the corresponding conductor ascending from the right,—and very little reflection will serve to supply the demonstration wanting. With the sensory and motor conductors arranged in this manner, it is plain that a cut across the lateral half of the gray substance—a lesion indicated in the diagram by the line A B—must de-

Fig. 58.



stroy the continuity of the motor conductor *a b*, and of the sensory conductor *c d*, and leave untouched the motor conductor *a′ b′*, and the sensory conductor *c′ d′*—must bring about, that is to say, what has been seen to happen in the first of the two experiments under consideration; namely, preservation of sensation with loss of motion on the side of the lesion, and preservation of motion with loss of sensation on the opposite side. Again, with the sensory and motor conductors arranged in this manner, it is plain that a section of the gray substance of the cord midway between the two lateral halves—a lesion indicated in the diagram by the line C A D—must leave the motor conductors *a b* and *a′ b′* untouched, and cut across the sensory conductors *c d* and *c′ d′* at their point of decussation—must bring about what happens in the second of these two experiments, viz., numbness on both sides of the body, and paralysis on neither side.

In saying that paralysis without loss of sensation, on the same side of the body, and loss of sensation without paralysis, on the other side of the body, is produced by cutting across a lateral half of the spinal cord, all is not said that has to be said. In such a case there is, in addition, increased temperature and sensibility on the side on which sensation is preserved, and diminished temperature on the side on which sensation is lost, especially if the section be made high up near the medulla oblongata. It would seem, in fact, that the injury has acted upon the vaso-motor nerves contained in the cord as well as upon the common motor and sensory nerves, causing paralysis of vaso-motor nerves on the side on which there is increased temperature and sensibility, and irritation of vaso-motor nerves on the side on which there is diminished temperature and anæsthesia. At any rate this mode of explanation is neither impossible nor improbable. The experiments of Professor Claude Bernard, Dr. Brown-Séquard, and others upon the cervical sympathetic, prove that when this nerve is paralyzed by dividing it, a state of hyperæmia, of which the most conspicuous signs are a bloodshot state of the conjunctiva and of the lining membrane of the nostril and ear, with a contracted pupil, and with increased temperature, is at once set up on the same side of the head: and also that when the end of the divided nerve below the section is irritated, the immediate result is dilatation of the pupil, with an almost complete blanching and cooling of the parts which were bloodshot and warm a moment before. The vessels in these parts evidently relax and receive more blood when their nerves are paralyzed, and contract and receive less blood where their nerves are irritated; and the increased temperature and sensibility which happen in the one case, and the diminished temperature and sensibility which happen in the other case, are nothing more than the natural consequences of the increased or diminished quantity of blood in the parts in each case respectively. All this is plain enough. Moreover, there are other facts which go to show that phenomena in every way analogous to those which result from paralysis or irritation of the cervical sympathetic are produced by paralyzing or irritating vaso-motor nerves in other parts. There is, therefore, no reason why it may not be inferred that the increased temperature and sensibility of one side of the body, and the diminished temperature of the other side, which happen when a lateral half of the spinal cord is cut across, are the result of vaso-motor nerves being paralyzed in the one case and irritated in the other case. Nay, such an assumption is well-high inevitable, for the structural connection between the spinal and sympathetic systems of nerves is such as to make it scarcely possible to believe that a lateral half of the cord can be cut across without paralyzing and irritating vaso-motor nerves.

9. *Motor and sensory tracks.*—The conductors concerned in voluntary motion,

and those belonging to common sensation, both intercross in the cord, but not at the same place. From the *right* side of the brain, voluntary impressions pass to the motor nerves of the *left* side of the body, their course thither being, first, down the *right* anterior pyramid, then across to the *left* lateral column, then for a short distance down the *left* lateral column, then down the *left* anterior column, and to some extent also down the *left* side of the gray substance and the *left* lateral column, and so out at the *left* anterior roots: from the *left* side of the brain, these impressions pursue a similar course, only passing to the right side of the body instead of the left. Entering at the *right* posterior roots of the spinal nerves, the impressions which give rise to common sensations pass to the left side of the brain, up the *left* side of the gray substance, and to some extent also up the *left* lateral column and the *left* anterior column, the crossing to the other side of the cord being at the level of the entrance of the conductors into the cord, or thereabouts: entering at the *left* posterior roots of the spinal nerves, the impressions in question take a similar course to the *right* side. Both sets of conductors intercross in the cord, but not at the same place. The conductors concerned in voluntary motion intercross at the decussation of the anterior pyramids. The intercrossing in this case is at this place, and at this place only: there is none above it, none below it. The conductors belonging to common sensation, on the other hand, intercross below the decussation of the anterior pyramids, and throughout the whole length of the cord. These are the main points to be remembered with reference to the tracks of these two forms of conductors in the cord.

The conductors which have to do with coördination of movement appear to be confined to the posterior columns of the cord, and to the parts which connect these columns with the cerebellum, the restiform bodies, and the small posterior pyramids. They are quite distinct from the conductors concerned in voluntary movement, and they also differ from these conductors in this, that those belonging to the two sides of the body do not intercross anywhere.

The vaso-motor conductors which enter into the composition of the cord appear to lie chiefly in the gray substance, for the dilatation of vessels resulting from paralysis of these nerves is brought about by dividing the gray substance rather than the white. Moreover, the fact that this dilatation of vessels is on the *same* side as that on which the gray substance is divided, must be taken as a reason for believing that the vaso-motor conductors belonging to the two sides of the body, like the conductors which have to do with the coördination of movement, do not intercross in the cord.

And so likewise with certain other conductors of a vaguer sort. These lie in and about the olivary bodies, and in the upper third of the lateral column; and there is, as it would seem, no intercrossing between those belonging to the two sides of the body, for the simple fact is this, that the persistent spasm which is brought about by irritation, which spasm is the only fact pointing to the existence of these conductors, is always on the same side as that to which the irritation is applied.

On the other hand, there appears to be nothing peculiar in the sensory conductors which are not concerned in common sensations—those which have to do with pain, tickling, temperature, and the rest. What has been said of the common sensory conductors would seem to apply to them in every respect, and indeed it may be doubted whether different conductors are required for the transmission of the different kinds of impressions.

10. *Increased reflex action.*—When the continuity of the cord is entirely interrupted by being cut, torn, compressed, or injured in any other way, voluntary movement and sensation are abolished in the parts below the injury; and at the same time the paralyzed muscles, especially in the lower extremities, become much more prone to reflex action. This increased proneness to reflex action is developed immediately, or all but immediately, and it may continue with little or no change for days, weeks, or even months. It makes its appearance before there is time for the development of inflammation or congestion; it continues after the time when any inflammation or congestion resulting from the injury which led to it may be supposed to have come to an end; and therefore it is difficult to look upon it as an indication of inflammation or congestion. Indeed the history of inflammation or congestion of the cord is opposed to this idea, for most certainly increased reflex action does not figure among the symptoms of unequivocal instances of these disorders. And this is all that need be said now, except this, that the history of increased reflex action would seem to be more intelligible on the view of muscular action which recommends itself to the writer, than on that which is commonly accepted.

11. *Increased temperature.*—In a paper on injuries of the spinal cord, published more than thirty years ago,[1] Sir Benjamin Brodie says: "M. Chopat has given an account of some experiments on animals, in which he found that the division

[1] Med.-Chir. Trans., vol. xx. 1837.

of the superior portion of the spinal cord produced a remarkable evolution of animal heat, so that it was raised much above the natural standard. I have made experiments similar to those of M. Chopat, and have met with similar results. I have also seen several cases in which an accidental injury of the spinal cord has produced the same effect. The most remarkable of them was that of a man who was admitted into St. George's Hospital, in whom there was a forcible separation of the fifth and sixth cervical vertebræ, attended with an effusion of blood within the theca vertebralis, and laceration of the lower part of the spinal cord. Respiration was performed by the diaphragm only, and of course in a very imperfect manner. The patient died at the end of twenty-two hours; and, for some time previously to his death, he breathed at very long intervals, the pulse being weak and the countenance livid. At last there were not more than five or six inspirations in a minute. Nevertheless, when the ball of a thermometer was placed between the scrotum and the thigh, the quicksilver rose to 111° of Fahrenheit's scale. Immediately after death the temperature was examined in the same manner, and found to be still the same." A Russian observer, Dr. Tscheschechin,[1] has also ascertained that considerable elevation of temperature, with quickened pulse and breathing, follows a section of the pons at its junction with the medulla oblongata, and that these symptoms go on increasing for two or three hours, until the state is that of high fever. Moreover, increase of temperature on one side of the body and decrease on the other has been seen to be one effect of dividing one-half of the gray substance of the cord. There is, indeed, reason to believe, not only that increased temperature is one effect of division of the cord, but that this change is in some way connected with paralysis of vasomotor nerves; for in speaking previously of the experiment last mentioned, it was shown that this paralysis may well be supposed to lead to this result.

12. *Hints for determining the level of the injury in certain forms of spinal paralysis, &c.*—If the injury be at the upper limit of the sacral region of the cord, the muscles of the bladder and anus will be paralyzed, and so will the muscles of the lower extremities, with the exception of those which are supplied by the anterior crural and obturator nerves (the psoas, iliacus, sartorius, pectineus, adductor longus, a. magnus, a. brevis, obturator externus, vastus externus, v. internus, rectus femoris, &c.), which nerves come off from the second, third, and fourth lumbar pairs of spinal nerves. If the injury be very low down in the sacral canal, the compressor urethræ and the accelerator urinæ, as well as the sphincter ani, will be paralyzed, but not the muscles of the legs; for the nerves of the three muscles, specified by name, come off almost from the extreme end of the cord, and below those which go to form the great sciatic. When the injury to the cord is higher up in the cord, in addition to the loss of voluntary power in the lower extremities and in the bladder and anus, the respiratory muscles will be more or less paralyzed. If the injury be at the upper limit of the lumbar region, the lateral muscular walls of the abdomen will be paralyzed, and so will all the muscles of the lower extremities, and one effect of the paralysis of the abdominal walls will be to compromise greatly the expiratory movements of respiration. If the injury be high enough to paralyze intercostal muscles, inspiration will be interfered with as well as expiration, and the degree of interference will be in proportion to the number of intercostal muscles implicated. If the injury be low down in the cervical region, all the intercostals will be paralyzed, and so will the muscles of the upper extremities, except those of the shoulders, which receive their nerves from higher portions of the cervical region. If the injury be at or above the middle of the cervical region—at or above the level of the fourth cervical pair of spinal nerves—death will at once result from the suspension of all inspiratory movements. In this latter case it is customary to ascribe the stoppage of breathing to paralysis of the nerve which supplies the diaphragm—that is, the phrenic; but this explanation does not go far enough. The injury which paralyzes the diaphragm paralyzes the scaleni, the inter-costales, and the serrati magni, which muscles elevate the ribs in ordinary respiration, and in so doing play a part which is scarcely less important than that played by the diaphragm; and not only so, but it paralyzes also the greater number of those accessory respiratory muscles which, acting upon and from the shoulders, come to the rescue when a great effort at inspiration is necessary, and produce additional expansion in the upper part of the chest. Not only is there a great difference between calm respiration and forced respiration, but there is a great difference also between the respiration of males and that of females. "In males," says Dr. Hutchinson, "the abdomen first bulges outwards, and the ribs and sternum nearest to the abdomen quickly follow this movement, until the motion, like a wave, is lost over the thoracic region. In females, the breathing commences with a gentle heaving of the upper part of the thorax, more or less apparent according to the fulness of the

[1] Reichert's and Du Bois-Reymond's Archiv f. Anat. u. Phys. 1866.

mammæ, and with some slight elevation of the shoulders; and this movement of expansion spreads from rib to rib in a downward direction, and any bulging of the abdomen from the descent of the diaphragm is distinctly after this heaving of the lateral wall of the chest, not before it." In females also this bulging of the abdomen is so inconsiderable that the number of respirations cannot be counted by the hand resting on that region as it can be in the male. In calm breathing, in fact, the diaphragm does more and the ribs do less in males than in females: and this difference is so real that, for the sake of distinction, calm breathing may be spoken of as diaphragmatic in males, and as costal in females. This difference is such, indeed, that respiratory movements which are healthy in women are morbid in men; and *vice versa*, that movements which are healthy in men are morbid in women. "In forced breathing," Dr. Hutchinson again says, "the greatest enlargement of the thoracic cavity in both sexes is made by the ribs and not by the diaphragm, as is generally believed;" and that this statement expresses what really happens, appears to be evident in the fact that in such breathing the hollow at the pit of the stomach, instead of being filled out and protruded, as it must be if the diaphragm descended in any marked degree, is actually drawn in and depressed. In forced breathing, indeed, the costal inspiration of women becomes more costal, and the diaphragmatic inspiration of men changes from this form to the costal. It is certain, however, that there may be forced diaphragmatic breathing as well as forced costal breathing, and that the one may be made to take the place of the other by an easy effort of the will, or by changes of position which interfere with the action of the diaphragm on the one hand, or of the ribs on the other. There is, indeed, no difficulty in understanding why diseases which interfere with the action of the diaphragm or ribs should make the breathing costal or diaphragmatic, as the case may be. As regards the expiratory movements of respiration there is little to say. In tranquil breathing, in males and in females alike, expiration is performed by the relaxation of the diaphragm allowing the abdominal viscera to press up into the position from which they had been depressed in inspiration by the contraction of this muscle, by the relaxation of the costal muscles allowing the ribs to spring back into the position from which they had been pulled up in inspiration by the contraction of these muscles, and by the resiliency of the air-passages themselves. In forced expiration the lateral and inferior muscular walls of the abdomen will help to empty the chest by pulling down the ribs and by contracting upon the abdominal viscera, so as to cause them to push up the diaphragm more effectually. It is easy, indeed, to see how a lesion of the spinal cord which paralyzes the lateral and inferior abdominal walls must interfere with the movements of expiration, and especially with such violent movements as coughing or sneezing. In a word, the whole case of the respiratory movements is one which makes it impossible to continue in the belief, that the one reason why the division of the cord at or above the origin of the phrenic nerve proves fatal, is because the diaphragm is paralyzed; for the plain fact is, that the injury which paralyzes the diaphragm paralyzes the muscles which elevate the ribs, both ordinary and extraordinary, and so puts an end to movements which are quite as important as those of the diaphragm, if not more so, in carrying on respiration. Of the other phenomena which may be present when the injury which interrupts the continuity of the cord as a conductor is in the neck, but not so high as to destroy life immediately, and which are not likely to be present when the injury is much below the cervical region, difficulty of swallowing, difficulty in vocalization, contraction of pupils, palpitation, and priapism appear to be the most important.

II. On the Practical Significance of Pain and Spasm, and of certain other Symptoms more or less akin to Pain and Spasm.

Have these symptoms to do with inflammation, or with a state which, though not unfrequently passing into inflammation, is in reality diametrically opposed to inflammation? This is the question to which I propose now to seek the answer, first, in relation to pain and the symptoms akin to pain, and, secondly, in relation to spasm and the symptoms akin to spasm.

1. *On the practical significance of pain and the symptoms akin to pain.*—There are some points in the history of common neuralgia—the beginning and ending of the paroxysm periodically at a given time, the association of the pain with rigors, the frequent ending of the pain in an obscure fit of feverishness, and others—which are calculated to suggest some relationship between this disorder and ague. It would seem, indeed, especially in that form of neuralgia which is met with in aguish districts, as if the neuralgia and the rigors were companion symptoms—as if there was some connection between the pain and a depressed state of the circulation such as is met with in the cold stage of ague. There is also some reason to believe that neuralgia

is antagonized rather than favored by inflammation and fever. It is no uncommon thing for the history of facial neuralgia or tic-douloureux to be this: first, neuralgia, without local tenderness and swelling, and redness, and with frequent chills and shivers, and a decidedly depressed state of the circulation; afterwards, cessation of neuralgia, cessation of chills and shivers, with local tenderness, redness, and swelling, and with some slight feverish reaction. What I have experienced in my own person, as well as what I have witnessed in others, enables me to speak with all confidence upon this point. It is also the rule, rather than the exception, for the *neuralgic* pain of toothache to come to an end when the face becomes swollen and inflamed; and it does not seem to be otherwise with the stabbing neuralgic pains which so generally precede the inflammatory eruption of herpes, for it is usual for these pains to subside concurrently with the development of the eruption. Nay, I know of several cases of sciatica, in which the relief to the neuralgic pain was coincident with the development of a tenderness which seemed to betoken neuritis at one or more points in the course of the painful nerve, and in which, after this change the patient was comparatively free from pain so long as the lame limb was kept still and let alone. With respect to neuralgia, in all its manifold forms, indeed one thing is certain, and this is, not only that neuritis is not necessary to its production, but also that this form of inflammation is at most a very exceptional complication.

Nor is a different conclusion to be drawn from the history of rheumatic and gouty pain.

In acute rheumatism it is generally found that the pains which had been torturing the patient for days, or weeks, or months previously, preventing him from being at ease in the daytime, and causing him to toss about in sleepless misery at night, come to an end when the feverish reaction and local inflammation of the fully-formed disorder make their appearance. After this, the joints are *tender* enough; but if the patient keep as still as he is very likely to do under the circumstances, he is comparatively or actually at ease so far as his old rheumatic pains are concerned. Or, if it be otherwise, the pains will generally be found to be in a part in which the signs of rheumatic inflammation are imperfectly established or absent, or else at a time when there is a decided remission in the feverish reaction—an event which happens more frequently in this disorder than is commonly supposed.

And certainly it is impossible to look upon the local inflammation of gout as essential to the racking pain of this disorder. "About two o'clock in the morning," says Sydenham, who knew full well from personal experience what he ought to say, "the patient is awakened by a severe pain in the great toe, or, more rarely, in the heel, ankle, or instep. The pain is like that of dislocation, and yet the parts feel as if cold water were being poured over them. Then follow chills and shiverings, and a little fever. The pain, which was at first moderate, becomes more intense; and with its intensity the chills and shivers increase." After tossing about in agony for four or five hours, often till near daybreak, the patient suddenly finds relief, and falls asleep. Before falling asleep, the only visible change in the tortured part is some swelling in the veins; on waking in the morning the part has become swollen, shining, red, tender beyond measure, and more or less painful, but painful only to a degree which is as nothing in comparison with the torture of the night past. It seems, indeed, as if the pain which now exists may in great measure be referred to the mere tension and stretching of the inflamed ligaments, for it may be relieved, or even removed, by judiciously applying support to the toe and sole of the foot. On the night following, and not unfrequently for the next three or four nights, the sharp pain may return, reappearing and disappearing suddenly, or almost suddenly, and resulting in the development of additional inflammatory swelling in the interval between falling asleep and waking in the morning. The pain in these relapses, like the pain in the first attack, is accompanied by chills and shivers, and by the most distressing irritability and excitability; but, until unequivocal signs of inflammation are developed in it, the painful part is not tender in the true sense of the word. The inflammation, moreover, is attended by no fever, or by very little; or if it be otherwise, as it is occasionally, the inflammation runs higher than usual, *and the characteristic pain is less urgent than usual.* Dr. Garrod points out this latter fact in his excellent work on Gout. From its history, then, it would seem as if the pain went hand in hand with the rigors which belong to the cold stage of gouty inflammation. It would seem as if the inflammation, as inflammation, had little to do with the pain; for if it were otherwise, it is scarcely to be supposed that the pain should be less urgent in the cases of gout in which the inflammation is most marked, and that the unequivocal signs of inflammation should make their appearance during sleep without waking the patient. Nay, it would even seem as if the pain were put an end to by the establishment of inflammation—as if, in

fact, the pains were antagonized rather than favored by the inflammatory condition. Moreover, the suddenness with which it begins and ends in the majority of cases must be looked upon as a reason for referring the pain to the category of neuralgia—a disorder with which, as I have already shown, inflammation has no necessary connection.

There is also reason to believe that pain holds the same relation to fever and inflammation in other kinds of fever besides the rheumatic, and in other kinds of inflammation besides the gouty.

The pain in the back, often very severe, which ushers in smallpox, disappears before the hot stage is fully established. It comes and goes hand in hand with the rigors, and it belongs to the cold stage as evidently as do the rigors. And this would seem to be the case also in other fevers; for it is the rule, and not the exception, for the pains which attend upon the onset of these disorders to pass away or to become greatly mitigated as soon as the cold stage gives place to the hot. Nay, it would seem as if pain gave place for the time to what may be called artificial feverishness. At any rate, I have more than once felt tic-douloureux in my face pass away as soon as I could set my blood in brisk motion by violent bodily exercise; and on two occasions I have put a stop to a sudden attack of lumbago while in the saddle, by a practice which is not unfrequently adopted in such a case in the hunting-field—that is, by leaning forwards, and beating the loins with the hands until the whole body was aglow, and the perspiration dropped from the forehead.

The acute pain of a dislocation or sprain—the pain to which Sydenham likens that of gout—does not, as a rule, remain after the parts have begun to be hot and tender and swollen; and as a rule, also, the pain of idiopathic inflammation goes before, and not along with, the redness and heat and swelling. In the idiopathic, as well as in the traumatic forms of inflammation, it would seem, indeed, as if the pain were related to the cold stage of the disorder, and not to the hot. Nor is a contrary conclusion to be drawn from the history of those cases in which the pain continues after the hot stage of the inflammation is fully established, for in these cases this persistent pain is evidently (in great measure at least) due to the stretching of parts made tender by the inflammation. Thus, for example, the pain which remains after the hot stage is fully established in orchitis and pleuritis, is at once removed or relieved by means which obviate this stretching,—in the former case by the free use of the knife, in the latter case by the application of a roller around the chest so as to prevent the movement of the ribs over the seat of inflammation.

Even in inflammation of the membranes of the brain, severe pain in the head cannot be looked upon as a symptom of this inflammation. About six years ago I had a youth in the Westminster Hospital with well-marked symptoms of acute cerebral meningitis. When I first saw him, he complained of frequent rigors and of a constant agonizing pain in the head, and at this time his face was pale and perspiring, his ears and his head generally were below the natural temperature, his pupils somewhat dilated, and his pulse contracted and feeble. Eight hours afterwards, when I saw him the second time, his face was flushed, his head burning hot, his pupils contracted, his eyes ferrety, his skin hot and dry, his pulse strong and full, and fierce delirium had taken the place of the pain. And this, so far as my experience goes, is the regular history of pain in this disorder. It is pain ceasing, not pain beginning, as the symptoms of active determination of blood to the brain make their appearance. It is pain in association with an anæmic rather than with a hyperæmic condition.

For these among many reasons it is that pain (with the exception of that form of pain which is dependent on tenderness, and which is accidental only) does not appear to be a symptom of inflammation or fever. In inflammation or fever the pain would seem to be connected with the cold stage preceding the hot stage, and not with the hot stage itself—with a state of capillary contraction and deficiency of blood, and not with a state of capillary relaxation and excess of blood—with a state of vaso-motor irritation, and not with a state of vaso-motor paralysis: in other cases, the pain would seem to have to do with a state of circulation which is in reality closely akin to that which exists in the cold stage of inflammation and fever. Pain, however, must not be regarded as a symptom of inflammation or fever because it happens to be associated with the so-called cold stage of these disorders. In point of fact, this so-called cold stage of inflammation or fever is a state which is diametrically opposed to the so-called hot stage. In this cold stage, the vaso-motor nerves (and not these nerves only) are in a state of irritation, and, as the result of this irritation, the capillaries are contracted and comparatively bloodless; in the hot stage, on the contrary, the vaso-motor nerves are paralyzed, and, as the result of this paralysis, the capillaries are relaxed and bloodshot. Instead of being stages in the same process, the so-called cold stage and the so-called hot stage are conditions diametrically opposed to each other. Instead of being stages in the same process, it would

rather seem that the hot stage has a remedial relation to the cold stage—that, within certain limits, the hot stage is the salutary refluence of a tide of life which has ebbed too low in the hot stage. It is not difficult to see that there is an intimate connection between the so-called cold stage and the so-called hot stage, and that the first may easily change into the second. It is not difficult to see that there must be this relation between these stages; for if, as there is good reason to believe, irritation of vaso-motor nerves may bring about the cold stage by causing contraction of vessels, it is easy to understand that the paralysis of vaso-motor nerves, which follows when this irritation is carried beyond a certain point, may lead to the hot stage by causing relaxation of vessels. At any rate, be this as it may, the plain fact would seem to be that pain, with the exception of that form of pain which is dependent on tenderness, is a symptom belonging to the so-called cold stage of inflammation and fever, or to a state of circulation closely akin to it, and not to the hot stage of inflammation and fever, or to a state of circulation akin to it. Nay, it may even be supposed, and not without some show of reason, that pain *must* be associated with contracted and empty capillaries; for, the sympathies of the nervous system being what they are, it is not easy to believe that the vaso-motor nerves do not participate in the irritation of the sensory nerves, for which pain is the expression in words.

And if this be so—and this is the practical conclusion to which these remarks tend—it follows that pain is likely to be relieved by measures which are calculated to rouse the circulation and increase the quantity of blood in the capillaries of the painful part, and not by those which have a contrary action.

With regard to tingling and other symptoms which are more or less akin to pain, there is little to say. Indeed all I can say is that the history of these symptoms, so far as is known to me, would seem to agree rather than to disagree with that of pain, in connecting them with a state of irritation, and not with a state of actual inflammation.

2. *Of the significance of spasm and the symptoms akin to spasm.*—The violent and general epileptic form of convulsion which attends upon death by hemorrhage or suffocation is associated with a defective and not with an excessive supply of arterial blood to one or other of the great nerve-centres. Nor is it otherwise with ordinary epileptic or epileptiform convulsion. The deathly paleness of the countenance which precedes the convulsion is, indeed, a plain proof that the fit commences in a state of circulation which is the very opposite to that of active determination of blood to the head, and the strong pulse which is usually perceptible in the arteries as the fit progresses is no contradiction to this conclusion. This strong pulse is usually regarded as a sign of arterial excitement—as a proof that more arterial blood is being injected into the arteries at this time, and that, on this account, certain nervous centres are excited to an unwonted degree of activity: but the simple fact is, that the strong pulse which is present under these circumstances derives its strength, not from arterial blood, but from venous. Black blood is being pumped into the arteries at the time; and because black blood moves less readily through the capillaries than red blood, the arteries become distended and the pulse endowed with a counterfeit power. The strong pulse in question is caused by the suffocation which is a part of the fit: it is a pulse of black blood and not of red, as may easily be proved by making an opening into the artery: it is nothing more, in fact, than the natural pulse of suffocation. Hence, the strong pulse of the epileptic or epileptiform paroxysm is no proof that this form of convulsion is connected with an excited condition of the circulation; on the contrary, when rightly read, it points only to the opposite conclusion.

It would seem also that convulsion is not associated with an over-active condition of the circulation, even in those cases in which at first sight it might appear to be so. In the fevers of infancy and early childhood, especially in the exanthematous forms of these disorders, convulsion not unfrequently takes the place occupied by rigor in the fevers of youth and riper years. It occurs in the initial cold stage, or else in the last moments of life, not in the intermediate hot stage. Again in inflammation of the membranes of the brain, convulsion, when it occurs, is connected with the cold stage before the hot stage, or with the cold stage after the hot stage, and never with the hot stage itself. Nay, I am disposed to think that there is something altogether uncongenial between convulsion and a state of febrile reaction in the circulation, for it is a fact not unfrequently verified that fits of common epilepsy are often suspended during the continuance of such reaction.

As indeed I have endeavored to show at length elsewhere,[1] the physiology and pathology of muscular action, so far as I can read them, serve only to connect all

[1] Epilepsy, &c.: Lectures delivered at the Royal Coll. of Phys. in London. Post 8vo. Churchill, 1862.—"Dynamics of Nerve and Muscle," and "Electrophysiologica," in "Nature," Jan. 4, 11, and 16, 1872. Post 8vo. Macmillan & Co., 1872.

the varied forms of tremor, convulsion, and spasm, with diminished and not with increased activity of the circulation; and thus the practical significance of spasm and the symptoms akin to spasm would appear to be the same as that of pain and the symptoms akin to pain—namely this, that the measures calculated to afford relief are likely to be those which will rouse the circulation to greater activity and increase the quantity of blood in the capillaries, and not those which have a contrary action.

B. On Diseases of the Spinal Cord.

Under the head of diseases of the spinal cord there is no lack of subjects. As of primary importance may be mentioned spinal meningitis, myelitis, spinal congestion, tetanus, locomotor ataxy, and spinal irritation; as of secondary importance, reflex paraplegia, infantile paralysis, hysterical paralysis, hemorrhage, white softening, induration, atrophy, hypertrophy, tumor, concussion, compression, vertebral caries, spina bifida, &c. I shall take each of these subjects in the order in which it has been enumerated, and, as far as I can, apportion the limited space at my command (very limited for such a purpose) so that there may be room for saying most where most is wanted.

I. Spinal Meningitis.

Inflammation of the membranes of the spinal cord is usually associated with inflammation of the substance of the cord (myelitis) or with inflammation of the membranes of the brain, but uncomplicated cases do occur now and then, and with care it is not difficult to discriminate between the symptoms which are essential to spinal meningitis and those which are only accidental.

1. Symptoms.—In order to arrive at a knowledge of the symptoms of spinal meningitis, I will relate as a text one of five cases verified by post-mortem examination which have come under my own notice, and then proceed to see wherein it agrees with or differs from other cases of the kind. I choose an acute case rather than a chronic one, for it is only in the acute form of the disease that the symptoms are to be defined with certainty.

Case.—A lightly-made, delicate-looking youth, nineteen years of age, a cigar-maker by trade, was admitted into one of my wards in the Westminster Hospital on the 27th December, 1864.

(*a*) When I saw him first—this was on the day after his admission—he complained chiefly of pain in the back and great general weakness and weariness, and expressed his belief that he had got rheumatic fever. He was then sitting by the fireside, and looking very ill. On telling him that he had better lie down, he got up and walked towards his bed, or rather he attempted to do so, for the first step brought on a severe pain in the back and legs, with a feeling of faintness and want of breath, and he would have fallen if assistance had not been at hand. Very soon after lying down he passed about a quart of water without any difficulty.

(*b*) The account he gives of himself is this. A week ago, after being very tired by a long walk, he was seized by shiverings and sharp pain between the shoulders. During the next three days he was feverish and without appetite, but still able to go about and do his work. All this while he had very little pain, and his nights were not disturbed. On the night of the fourth day from the commencement of the illness, he was awakened by violent pain along the whole course of the spine, in the groins, and in the right leg. Next day the pain occurred several times in paroxysms, and was accompanied by a good deal of starting and jerking in the legs; and so also on the two days following. On the day before admission to the hospital, some difficulty in opening the jaw was experienced, and the paroxysms of pain, and jerking, and starting had become more frequent and urgent. All this while the bowels and bladder acted properly.

Dec. 28.—There is no material change since yesterday—not for the worse, certainly.

Dec. 29.—Last night, after three or four hours' sleep, the patient awoke with very severe pain along the spine and down both legs, and since that time the pain has recurred several times. These attacks are separated by intervals of comparative or complete ease, and instead of the jerks and starts, which went hand in hand with it previously, the pain is now accompanied by stiffness in the muscles of the back and legs. At the present moment (about 2 P. M.) the head is drawn back on the pillow, and considerable pain and stiffness in the neck is caused by moving it. Before making such movement the patient was free from pain and stiffness in this region. Asking him to try and sit up, he attempted to do so, but was stopped at once by a severe paroxysm of pain along the whole length of the spine and down the legs, and by the muscles in the painful parts becoming stiff. The action of the muscles produced in this way arched the body backwards almost as much as in ordinary cases of tetanus, and at the same time pursed up the mouth and eyes, and gave a set expression to the features generally, so that

the patient for the time had the appearance of a person considerably older than himself. The pain went off in a few minutes, and soon afterwards the stiffened muscles relaxed. The effort to move one of the legs spontaneously gave rise to a sharp pain in the thigh and loins, and the limb became somewhat stiffened in a semi-flexed position, and this state of things did not pass off for several minutes: and passive movement produced the same result. There was no numbness: on the contrary, the condition of the skin as to sensation everywhere, as judged by pricking and pinching and by differences of temperature, was plainly that of slight over-sensitiveness. Pressure along the spinal column failed to detect tenderness anywhere, and the result of applying a sponge wrung out of hot water was equally negative.

In the course of the examination it was evident that any movement of the body, or neck, or legs, active or passive, gave rise to pain and stiffness in the muscles moved; and also that there was little or no pain or stiffness so long as the patient kept quite still. It was evident, in fact, that the muscles were relaxed, except perhaps in the neck, in the intervals between the paroxysms. The poor sufferer was evidently in a great strait, dreading all movement, because he knew full well what the effect of movement would be, and at the same time continually prompted by an intolerable feeling of unrest and fidgetiness to wish to have his position changed in a way which he could not or dared not compass by his own efforts: and it is difficult to avoid the conclusion that the stiffness is, in the main, an instinctive act to prevent the movement which gives rise to the pain, rather than spasm like that which is met with in tetanus. The arms are affected as well as the legs, but not to the same degree. They are weak—so weak that it is not easy to find strength to carry the food to the mouth, the left arm being somewhat the weaker of the two. The left arm also cannot be moved, either actively or passively, without giving rise to pain and rigidity, to pain shooting up between the shoulders, to rigidity flexing the limb somewhat at the elbow, and bending the thumb slightly into the palm: not so the right arm. There is no numbness in either arm, and no very decided over-sensitiveness. Mastication is difficult, and deglutition still more so, apparently from the muscles set in movement becoming stiff in moving. The breathing is shallow and slow; the pulse quick (130) and very wanting in strength; the skin profusely perspiring after a paroxysm, and hot and moist at other times. Thirst is much complained of. The bladder is full, and it cannot now be emptied voluntarily. The urine is acid. The penis is flaccid, and has been so ever since the commencement of the illness. The bowels have not acted. The pupils are equal and natural, and there is no headache or other "head symptom."

Dec. 30.—A tolerably good night has been passed, and this afternoon the patient thinks himself a little better.

Dec. 31.—There has been a bad night, and much ground has evidently been lost since the last visit. In a paroxysm which is just over, want of breath was experienced rather than pain. Sensation is still somewhat exaggerated everywhere. Urine cannot be passed without the catheter, but the bowels have responded to-day to a dose of castor-oil and spirits of turpentine which was administered yesterday. During my visit I had an opportunity of seeing the patient *after* a paroxysm as well as in it, and I quite satisfied myself that the muscular stiffness of the paroxysm soon passed off, and that in the interval between the paroxysms the muscles were relaxed, except perhaps at the back of the neck—with this possible exception, because all along the head remained drawn back to some degree upon the pillow.

Jan. 1, 1865.—The night has been perfectly sleepless, with now and then some trifling light-headedness. The paroxysms of pain, stiffness, and difficulty of breathing are not so frequent (three hours have passed since the last), but the respiration is certainly shallower and less sufficient, and the pulse more rapid and unsteady. There is the same want of power over the bladder. When I left the ward, it was plain enough that the patient was sinking: when I returned two hours later all was over, death having happened in a fit of choking and suffocation caused by attempting to swallow a spoonful of beef-tea with a morsel of bread sopped in it. *In the agony, the patient not only sat up in bed, but got out of bed and stood for a moment with his hands bearing upon the shoulders of the nurse who had been feeding him.* The body was carefully examined after death by Dr. Bazire, and the following notes were taken at the time from his dictation:—

"Time, twenty-four hours after death. Weather frosty. Cadaveric rigidity well marked. The muscles of the back dark and highly congested. On cutting through the posterior arches of the vertebræ the vertebral vessels are seen to be gorged with dark fluid blood. There is no effusion of blood outside the meninges in the interior of the canal. The meninges are highly congested throughout the whole length of the canal, but to a considerably greater degree in the region between the scapulæ. In this latter region, in addition to the thickening, opacity, and intense

red color of the dura mater elsewhere. there are streaks in its substance of black coagulated blood. The arachnoid is intensely red, and the pia mater extremely congested in the same region. Beyond it, the dark red color of the dura mater gradually passes into a lighter shade, and becomes a bright pink near the cauda equina in one direction, and near the medulla oblongata in the other. The arachnoid is whitish again near the cauda equina. There is no effusion of serosity, blood, or pus, either between the meninges or on the surface of the cord; indeed, there seems to be a smaller quantity than usual of cerebro-spinal fluid. The substance of the cord itself looks normal in consistence, color, and size. The central vessel of the cord is highly congested, and on section of the cord there exudes from the centre fluid black blood in minute drops. The cerebral meninges are normal. The cerebral sinuses are highly congested, and the same appearances of congestion (due probably to the mode of death) are met with in the substance of the brain. The organ itself is normal."

The symptoms of acute spinal meningitis are plainly exhibited in this case, and there need be no difficulty in distinguishing those which are of primary importance from those which are secondary.

As symptoms of primary importance may be enumerated these:—fits of pain produced by movement along the spine and in the extremities: fits of muscular stiffness in the painful parts along with the pain; intervals of comparative or complete freedom from pain and muscular stiffness so long as movement can be avoided; absence of paralysis; some exaltation of sensibility; loss of power over the bladder; partial loss of power over the bowel; absence of spinal tenderness.

Fits of pain along the spine and in the extremities, produced by movement.—This pain, as I think, must be regarded as the most prominent symptom in acute spinal meningitis. It may be confined to the region of the spine, but more generally it shoots into the extremities, into the legs especially. As a rule, it does not shoot beltwise round the trunk. It is brought on by any movement of the trunk, and, in great measure at least, it may be prevented by avoiding such movement. It is often brought on also by moving one of the extremities, the pain in this case beginning in the limb, and extending thence to the spine. It seems to depend, in part at least, upon the same cause as the pain of pleurisy, viz. the dragging of an inflamed and therefore exquisitely tender serous membrane, and its character is certainly more like the pain of pleurisy than like that of rheumatism (to which latter it has been likened), for it occurs in the same sharp, sudden, breath-stopping catches.

Fits of muscular stiffness in the painful parts along with the pain.—It is usual to regard this stiffness as analogous to the spasm of tetanus: it is necessary, as I believe, to look upon it as expressing an instinctive act of muscular contraction, of which the object is to prevent pain by arresting certain movements which produce pain. The spine and extremities cannot be moved without causing pain: the stiffness prevents the pain by preventing the movement; this would appear to be the true view. This explanation, originally given by M. Dance as applying to the muscular stiffness in a case of acute spinal meningitis observed by him and recorded by M. Ollivier, applies perfectly to the muscular stiffness of the case which has been related as the text, and it applies, as I believe, with the same exactness to all cases of the kind. Indeed, I believe there can be no greater mistake than to confound the stiffness in question with the spasm of tetanus. This will be seen more particularly when speaking of tetanus: and here I will only say that tetanus in its most violent form is constantly present where there are no signs of spinal meningitis, and that, in the few cases in which such signs chance to be met with, it may be supposed that the inflammation is a consequence rather than a cause of the irritation which gives rise to the tetanic spasm—a consequence of the irritation in the vaso-motor nerves having proceeded until it has issued in paralysis of the vaso-motor nerves. Nay, after what has been said in the preliminary remarks, it is not impossible that the spinal meningitis which is occasionally associated with tetanus may have served to counteract the spasm rather than to cause it. At any rate, it is certain that spasm of the spinal muscles is not so marked a phenomenon in acute spinal meningitis as in tetanus, and that it is not to be regarded "comme indiquant positivement la phlegmasie des membranes de la moelle;" and it is, to say the least, highly probable that the muscular stiffness which simulates true tetanic spasm is in great measure an instinctive act of muscular contraction to prevent a movement which produces pain.

Intervals of complete or comparative freedom from pain and muscular stiffness so long as movement can be avoided.—These intervals are sometimes of considerable length, even for days. According to my own experience, indeed, the rule would seem to be that as long as the patient can keep still, so long is he, comparatively at least, free from pain and stiffness—a rule which is very different from that which obtains in tetanus.

Absence of paralysis.—The patient is weak, very weak, and he seems to be paralyzed, but in reality he fears to move

because movement brings back the pain. "Les mouvements, qui sont en quelque sorte enchaînés par la douleur, ont moins de force, mais ils ne sont point paralysés." (Ollivier, p. 595.) Let this fear be forgotten, and it is possible not only to sit up, but to get out of bed and stand, as happened in the final agony of the patient whose case I have given. This power of movement has been noticed in several cases, of which one is related by Ollivier, and another referred to; and I believe it would be witnessed in all cases of *uncomplicated* acute spinal meningitis in which the fear of suffering pain from movements was not the one absorbing feeling.

Some exaltation of sensibility. — In the case which I have given there was some exaltation of sensibility as to touch, pain, and differences of temperature, but to no very marked degree; and this would appear to be the rule in cases of the kind. It would seem, indeed, that numbness is a purely accidental symptom, which is never present unless the substance of the cord is implicated in the meningeal inflammation.

Loss of power over the bladder. — In acute spinal meningitis, when the symptoms are fully developed, this particular symptom is scarcely ever absent, if ever. Before this time it may be absent, as it was in the case on which I am commenting; but this absence must certainly be looked upon as the exception rather than the rule. Not unfrequently the inability to empty the bladder is preceded by a state of irritability which makes it necessary to pass water almost incessantly.

Partial loss of power over the bowel. — On this point M. Ollivier makes a remark which is certainly true: "Je ferai remarquer que l'abolition des fonctions de la vessie persiste toujours au même degré depuis le commencement jusqu'à la fin, tandis qu'il n'en est pas de même pour l'intestin, puisqu'il y a assez souvent des garderobes naturelles dans les derniers temps de la maladie." (Vol. ii. p. 601.)

Absence of spinal tenderness.—This absence is certainly a common, if not a constant, feature of acute spinal meningitis. In some chronic cases, no doubt, there may be some local spinal tenderness, but on inquiry these prove to be cases in which the phenomena of spinal irritation are mixed up with those of spinal inflammation—in which the inflammatory affection is complicated with that condition of which, as will appear in due time, local spinal tenderness is the distinctive feature.

These are the points which may be regarded as of primary importance in comparison with those which have still to be considered, namely — absence of marked spasmodic symptoms, difficulty of mastication and deglutition, difficulty of breathing, no increased reflex excitability, no priapism, fits of perspiration, no active inflammatory fever, no marked "head-symptoms."

Absence of marked spasmodic symptoms. —The rigidity which attends upon the paroxysms of pain has been seen to be in the main an instinctive act of muscular contraction to prevent a movement which produces pain, and there appear to be no other symptoms of a spasmodic character which occupy a conspicuous place in the history of spinal meningitis. Or if there be any such symptoms, these are in all probability confined, as were the jerks and starts in the case under consideration, to that early period of the disorder in which it may be supposed that actual meningeal inflammation was not developed—to the so-called cold stage of the disorder probably.

Difficulty of mastication and deglutition. —This difficulty is often absent, and when present it is at most a trifling trouble comparatively. There is no true trismus as in tetanus; there is at most only stiffness which prevents the jaws from opening easily and moving freely. This stiffness, moreover, is late in making its appearance, whereas in tetanus trismus is one of the very first symptoms. In a word, difficulty of mastication and swallowing would seem to occur only in those cases of spinal meningitis in which the higher portions of the cord are implicated.

Difficulty of breathing.—This difficulty is always present in some degree, and especially during a paroxysm of pain and stiffness. In some cases, indeed, the movement of the chest may be actually suspended at this latter time, and death may happen from this cause, as indeed was the case in a patient whom I saw not long ago with Dr. Julius of Richmond.

No increased reflex excitability. — This is not, perhaps, what might be expected theoretically: but, be the explanation what it may, the fact would seem to be that reflex irritability is not increased in acute spinal meningitis in the way in which it is ordinarily increased in tetanus. So far as I have been able to ascertain, there would seem to be no material change of reflex excitability in the meningeal inflammation.

No priapism. — The cases in which erection of the penis would seem to be a symptom appear to be those in which the substance of the cord is affected rather than the membrane—cases, too, in which the seat of the disease is in the cervical and upper dorsal region rather than in the lumbar region. At any rate, it would seem to be the rule for the penis to be flaccid in uncomplicated cases of acute spinal meningitis.

Fits of perspiration. — As in tetanus these follow a paroxysm almost invari-

ably, especially in the latter stages of its disease. Of this there appears to be sufficient evidence.

No active inflammatory fever. — Thirst is a frequent symptom throughout, and there may be at first some heat of skin, but in the most acute cases there is little or no active sympathetic fever. On the contrary, there is usually, even in the cases which have most claim to be considered as acute, a decided want of febrile reaction from the beginning to the end.

No marked head-symptoms. — In very many cases inflammation of the spinal meninges is only a part of a more general disorder in which the cerebral meninges are also implicated, and, therefore, "head-symptoms" of one kind or other will often enough be mixed up with the spinal symptoms; but in cases like the one under consideration, where the spinal meninges were alone inflamed, "head-symptoms" do not figure at all, or figure only as phenomena of very secondary importance. Upon this point there is no lack of evidence. Where spinal meningitis is chronic in its course, its symptoms are often so mixed up with the Protean symptoms of spinal irritation (of which more in due time) as only to be detected with great difficulty. It may be suspected that the meninges are affected by inflammation rather than by simple irritation if fits of pain and stiffness are produced by movement in the spine and extremities, and if there be at the same time no spinal tenderness, no paralysis, and no tingling or numbness; and this is all that can be said except this, that this suspicion will gather strength if there be chronic disease in the bones and ligaments of the spine. But it may be questioned whether long-continued contraction of the muscles of one or more of the extremities or of the cervical muscles can be reckoned among these symptoms, for such contraction is certainly common enough in cases where the only condition of disorder in the spinal cord or its membranes is one which, from the sudden way in which it begins and ends, and for other reasons as well, would seem to be one of simple irritation.

2. Post-mortem Appearances.—As Ollivier pointed out, the traces of spinal meningitis after death are met with usually, not in the arachnoid membrane, which is non-vascular, but in the subjacent vascular tissue. The arachnoid is so thin and transparent as to allow the vascular injection produced by the inflammation in the deeper structures to appear through it, and that is all. This injection is generally less evident on the surface of the cord than on that of the dura mater, because in the former place it is hidden by the effusion of turbid, sero-purulent, or purulent fluid in the space between the arachnoid and pia mater—in the space naturally occupied by the rachidian fluid—is hidden by an effusion which, before the arachnoid is opened, often causes the cord to have a swollen, opaque, yellowish-white, or yellowish appearance. Any fluid effusion is usually in this space, but sometimes there may be fluid, in this case often sanguinolent, in the space outside the dura mater, especially if there be disease in the bones or ligaments of the spine. Sometimes the rachidian space is obliterated here and there by inflammatory adhesions; sometimes the surface of the arachnoid is roughened or otherwise altered by calcareous or other deposits in patches; sometimes the opposed surfaces of the arachnoid are more or less adherent: but generally these surfaces are smooth and free, and the inflammatory products are met with below this membrane, and not above it. Very often, also, the proper signs of spinal meningitis are mixed up with those of cerebral meningitis or myelitis, or with those of disease in the bones or ligaments of the spine.

3. Causes.—The causes of spinal meningitis are often very obscure. In some cases it is rheumatism, or syphilis, or the suppression of some menstrual, hemorrhoidal, or other habitual discharge, or the spreading of cerebral meningitis downwards, or of disease in the bones and ligaments of the spine inwards, which would seem to figure as a cause; in other cases it is a casual injury to the back, or a chill caught by lying on the back on the cold and damp ground, or some particular disease, as tetanus, chorea, or hydrophobia, to which blame appears to belong. In fact, the causes are legion, and it is impossible to connect spinal meningitis with any particular cause or set of causes.

4. Diagnosis.—One or two points of diagnosis have been mentioned incidentally when dealing with the symptoms of spinal meningitis, and with these it is best to be content at present, for before this matter can be gone into advantageously materials must be had which can only be forthcoming when the phenomena of myelitis, spinal congestion, tetanus, and other spinal maladies have been passed in review.

5. Prognosis.—Acute spinal meningitis is, without a doubt, a very formidable and fatal disease. There are, indeed, few well-authenticated instances of recovery on record, and by some it is doubted whether there be any. Life may be cut short in four or five days, or it may be prolonged to twenty or thirty days, but not often—not often indeed—beyond six or seven days. In the subacute and chronic forms of the disease, the prognosis

is of course less gloomy, but even here it is far from cheering.

6. Treatment.—In all cases of spinal meningitis, rest in the recumbent position, more or less strictly enforced according to the urgency or leniency of the symptoms, is indispensable, the best position, perhaps, being not strictly on the back, but rather upon the side, and with the limbs a little lower than the back, so as to favor the draining away of blood from the congested parts, and, at the same time, to facilitate the use of the local applications to the spine which may be necessary. Upon this point there can be little or no difference of opinion; upon all other points, in all probability, few will think alike. For my own part, I should be disposed to place most confidence in iodide of potassium and opium, with the local application of ice to the back in acute cases, and to bichloride of mercury, with counter-irritation in one form or other to the spine, in chronic cases. At the same time, I am inclined to think that the present fashion has set very unwarrantably against the old practice of giving calomel and opium, so as to affect the gums slightly and speedily, and of using local, if not general, bleeding in acute inflammatory disease. There can, I think, be little doubt as to the marked influence for good of calomel and opium in acute inflammation of serous membranes; and it would require very little persuasion to induce me to prefer this mode of treatment to that of iodide of potassium in acute spinal meningitis; and, further, I can readily believe that in such a case recovery would be promoted by judicious abstraction of blood. I have twice seen symptoms, so closely resembling those of acute spinal meningitis as not to be distinguishable from them, disappear coincidently with the occurrence of local hemorrhage, once from piles, once in the form of menstruation; and I can well believe that a similar result might be furthered by the application of leeches around the anus or to the cervix uteri—to these parts rather than to the back, because their vessels would seem to communicate more directly with the deep spinal vessels. It is very probable, however, that the time will soon pass in which depletion in any form, or depressing remedies of any kind are required, and that the indications will rather be towards brandy, or ammonia, or turpentine, or ether, than towards the remedies which have been mentioned, for all acute diseases of the spinal cord would seem to have a rapidly devitalizing influence upon the system. In acute cases the catheter will be necessary to empty the bladder; in chronic cases, aching and stiffness of the limbs may point to friction and shampooing as likely means of relief. In every case there is sure to be some peculiarity to which attention must be directed if the plan of treatment be all that it ought to be; and, in short, every case must be treated on its own merits.

II. Myelitis.

Myelitis, or inflammation affecting the substance without involving the membranes of the cord, is a well-defined and not very uncommon disease. It may occur in an acute or in a chronic form: it may be general or partial: and, to say the least, its features are quite as well marked and distinctive as those of spinal meningitis.

1. Symptoms.—As an instance of acute myelitis, and as a text for what has to be said under this head, I take the notes of the case of a hospital patient under my care some time ago.

Case.—Charles K., a draper's assistant, twenty-six years of age, unmarried, a patient admitted into the National Hospital for the Paralyzed and Epileptic on the 9th of January, 1864.

(*a*) The chief symptoms complained of are paralysis and anæsthesia below the waist, a disagreeable feeling of tightness around the waist, inability to pass water, involuntary stools, and pain in the left side of the chest. Above the waist, the power of movement and the power of sensation are natural; below the waist, all the voluntary muscles are entirely paralyzed, and the sensibility to pain, to tickling, to differences of temperature, as well as to touch, are completely lost. Pressure along the spine is felt above the point to which the anæsthesia reaches, but not below it, and where felt the patient bears it without wincing. In other words, there is no tenderness on pressure in that part of the spine which preserves its sensibility. The feeling of warmth produced by passing a sponge soaked in moderately hot water along the spine is felt above the point to which the anæsthesia reaches, but not below it; and, where felt, the feeling of heat is natural, except at the line of junction between the sensitive and insensitive parts, and there the feeling produced is that of burning. Moreover, the warm sponge produces the same feeling of burning all around the body in the course of this line of junction, and thus it is plain that this local over-sensitiveness to heat is not confined to the spine. No reflex movements are produced by tickling the soles of the feet. The alæ nasi work very much, the lips are somewhat dusky, the lower intercostal muscles are motionless, and the accessory inspiratory muscles are in full work; the air-passages (especially on the left side) are loaded with

phlegm, the pulse is hurried and weak, the skin is moist and somewhat cooler than natural, and the voice is so low as to be scarcely audible. A cough of the feeblest sort is almost incessant, but the expiratory power at command is altogether insufficient to bring about the expectoration which is so much needed. All appetite is gone, but food can be taken, and there is no thirst, or none to speak of. The urine, which is acid, and of the specific gravity of 1015, has to be drawn off by the catheter. There is no priapism. A stool has just passed without the patient being aware of it until his nose took account of the accident.

(*b*) A week ago, on awakening from a short nap, the patient found that his toes had gone to sleep, and that he had to "take long breaths." Instead of passing off, the feeling of tingling spread rapidly from the toes to the feet, from the feet to the legs, from the legs to the thighs, until it reached the seat, becoming less and less endurable as it spread, and being at last accompanied by a feeling of tightness around the waist and around the left instep, and by a state of restlessness which made it scarcely possible to sit still for a moment. After suffering in this way for a couple of hours, an attempt to pass water, which failed altogether, was followed by an almost intolerable uneasiness at the end of the penis, and by a sudden weakness in the legs which made it necessary to remain on the bed upon which he had fallen. Up to this time there had been no difficulty in standing, or walking, or even in going up and down stairs. A friend of the patient's now present says: —"I saw him on the evening of the day on which he was attacked, a couple of hours or so after he had been obliged to take to his bed. I thought he was suffering from severe rheumatic pains. For some hours those pains were excruciating. I had never before seen any one suffer so much. He tossed about in dreadful agony; he roared out with pain often, and, when not roaring, he groaned." Having thus passed seven or eight miserable hours, he fell asleep, and slept until breakfast-time next day. Upon waking in the morning he could neither move his legs nor empty his bladder; he had lost all feeling below the waist, and all the miserable feelings which had kept him in a state of continual unrest before he fell asleep were gone. On inquiring whether these feelings were of the character of pain, he says, "No, not exactly; worse than pain, one continued numb stinging feeling, as if the parts were asleep;" so that the friend's words which have just been given must be taken as meaning not exactly what they seem to mean in this particular. For the six days preceding his admission to the hospital a state of imperfect priapism was apt to come on of itself, or to be brought on by introducing a catheter to draw off the water, and this is the only point remaining to be noticed here, for in other respects the condition seems to have remained stationary, except, perhaps, that a little ground was lost every day.

The patient seems to have come of a healthy family, and, though never very strong, to have himself always enjoyed tolerably good health. He was confined to the house for a few days about two months ago by "influenza," and this is the only illness of any kind he remembers to have had. He says, "I was fatigued by a long walk on the day I was taken ill, and for a month and more I had felt more tired in my back and legs than usual in an evening, and more rheumatic —less up to the mark;" and also, "My back always ached at the end of the day's work, and so did my legs, and I was always glad to go to bed soon, for in bed I was comfortable:" and besides these statements there appears to be nothing at all calculated to throw light upon the history of his present malady.

Jan. 10.—Early this morning, after a sleepless night, a severe rigor commenced in the right arm, and then extended first to the back, and afterwards to the whole body. This rigor continued a full quarter of an hour, and was followed by profuse perspiration. During its continuance the paralyzed parts were very cold: after it had ceased the warmth returned, and brought with it a considerable mitigation of the cough and trouble of breathing. Indeed, after the establishment of reaction, difficulty of breathing ceased to be an urgent symptom, except for a moment or two after waking from an occasional and very brief doze. The anæsthesia in the trunk has mounted full an inch higher since yesterday, but it has not extended to either of the upper extremities. Priapism occurs frequently. The pulse is 150; the respirations are 36 in the minute.

Jan. 11.—There has been no sleep in the night. The engorged condition of the lungs has gained headway, and the harassing suffocative cough has returned. Hiccough is frequent and distressing. Once during the day the passage of the catheter was obscurely felt, this being the first sign of feeling in this part since the commencement of the illness. The urine is decidedly acid. The electro-contractility and electro-sensibility of the paralyzed muscles are annihilated.

Jan. 12.—For the last twenty-four hours the increased difficulty of breathing attending sleep has caused the patient to wake immediately if he for a moment forgot himself. "I can't breathe except I keep awake," he said in a voice scarcely

audible; and also, "I hope I have not long to live." The passage of the catheter is still obscurely felt, and the escape of flatus and feces is perhaps not so entirely unfelt as it has been since the commencement of the illness. In other parts the anæsthesia, like the paralysis, remains as complete as ever. The urine is still acid, distinctly so. For the last twenty-four hours there has been no priapism, and scarcely any cough. At present hiccough is almost constant, the pulse is fluttering, the hands are cold and clammy, and, in short, the signs of the near approach of death are not to be mistaken.

Jan. 13.—The patient lingered through the night, and died about daybreak; his mind unhappily remaining too clear to the very last.

The notes of the post-mortem examination are as follow:—

Jan. 14, 4.30 P. M.—Rigor mortis is fully established everywhere. The dependent parts present considerable signs of suggilation, especially along the course of the spine, and there is incipient breaking of the skin on both the nates. The arachnoid covering of the cord everywhere is clear, smooth, and without any traces of inflammation. The outside of the lumbar enlargement is curiously nodulated. On making a longitudinal section, the whole substance of the cord, from the brachial enlargement to its inferior extremity, is found to be of a yellowish-red color, softened in a remarkable manner, and in the lumbar region almost like cream in consistence. Several small patches of extravasated blood are scattered in the softened structure, these patches being undefined in outline, more numerous in the lumbar than in the dorsal region of the cord, and situated chiefly in the posterior columns. The red discoloration which has been mentioned is most marked in the neighborhood of these patches. The examination did not extend further, the friends of the patient consenting to it only on condition that it should be thus partial.

Jan. 15.—On examining some portions of the diseased cord under the microscope, the natural structure is found to be altogether broken down, and mixed up with blood-corpuscles, exudation granules, and (in fewer numbers) pus-corpuscles.

With a view to arrive at a knowledge of the general features of myelitis, I select as the principal points for comment in this particular case the following: Paraplegic anæsthesia, ushered in by tingling or some similar sensation in the parts which eventually became anæsthetic; paraplegia ushered in by uncontrollable restlessness; a disagreeable feeling of tightness around the waist and elsewhere; absence of pain in the spine or extremities—of pain produced by movement especially; absence of trismus and other spasmodic or convulsive symptoms; retention of urine; involuntary stools; absence of pain on pressure (spinal tenderness) in any part of the spine; increased sensibility to differences of temperature, by which moderately warm or iced water gave rise to a feeling of burning instead of the natural feeling over the vertebra which marks the upper limit of the myelitis; annihilation of reflex excitability in the paraplegic parts; priapism; acidity of urine; comparative voicelessness; impeded respiration; engorgement of lungs and other viscera; tendency to bed-sores; loss of electro-contractility and electro-sensibility in the paralyzed muscles; absence of head-symptoms; absence of fever.

Paraplegic anæsthesia, ushered in by tingling or some similar sensation in the parts which eventually became anæsthetic.—In this case the anæsthesia was developed suddenly during the first night's sleep; it was deep-seated as well as superficial; it implicated the sensibility to pain, tickling, and differences of temperature, as well as that of touch; it had a paraplegic distribution: and this would seem to be the rule in cases of acute myelitis. In chronic cases it is developed more gradually, and it may not extend to all the various forms of sensibility; moreover, it may in some instances be quasi-hemiplegic instead of paraplegic; but the rule in acute cases appears to be what it is found to be in this. The anæsthesia seems to be usually ushered in by tingling or by some analogous sensation, disagreeable enough, but not amounting to actual pain. In this particular case the preliminary sensation was not pain, but an unbearable "numb stinging," as if the parts were asleep, with a feeling of tightness around the waist, and around one of the insteps. In acute cases it is right to speak of anæsthesia as ushered in by tingling or some similar sensation, but scarcely so in chronic cases. In chronic cases, indeed, these anomalous sensations may never exactly come to an end, because in these cases the destruction of sensibility may never get beyond numbness—may never reach nearer to anæsthesia; that is to say, than dysæsthesia.

Paraplegia ushered in by uncontrollable restlessness.—The paralysis was thus ushered in in the case under consideration, and in six similar cases which have come specially under my notice, by restlessness, and *not* by any more marked tremulous, convulsive, or spasmodic symptom. Neither does it appear that a different rule obtains in other cases, acute, subacute, or chronic. In the great majority of cases, no doubt, the paralysis has a paraplegic form, but in a few cases it is not

so. In the great majority of cases, the paralysis is accompanied by numbness, but not absolutely in all. Sometimes, for example, as in the case in which the paralyzing lesion is limited to a portion of one lateral half of the spinal cord,—the case about which enough was said in the preliminary remarks,—there is paralysis without numbness on one side, and numbness without paralysis on the other side. Several cases of this kind are on record, and the number of them which I have myself met with is sufficient to convince me that they are scarcely to be looked upon as out of order and exceptional. Sometimes, also, as in the case where the paralyzing lesion is confined to a portion of one of the anterior columns, the paralysis may be divorced from numbness, and not only so, but it may be hemiplegic in its distribution; and in such a case it may, in fact, be no easy matter to say whether it is dependent upon a cerebral or upon a spinal cause. In some cases, also, the paralyzing lesion may be so localized as to affect only, or chiefly, an arm on one side and a leg on the other side. Usually, however, the paralysis is distinguished by being associated with numbness, and by being paraplegic in its distribution.

A disagreeable feeling of tightness around the waist and elsewhere.—A feeling of circular constriction around the trunk, or around some part of an extremity, around the trunk especially, is so common as to deserve to be considered as an almost constant symptom in myelitis. I do not recall a case, acute or chronic, in which it was entirely absent at all times.

Absence of pain in the spine and extremities—of pain produced by movement more especially.—In chronic cases of myelitis, Dr. Brown-Séquard speaks of "a constant pain in the part of the spine corresponding to the upper limit of the inflammation of the cord" as a characteristic symptom; but I question very much whether this statement is in accordance with well-sifted clinical facts. Pain, either in the spine or elsewhere, is not mentioned, for example, in the nineteen cases, acute or chronic, given by Ollivier, except in three; and of these three the myelitis was complicated with meningitis in two, and in the one remaining the symptoms justify the presumption (and there was no post-mortem examination to set it aside) that the same complication existed. At any rate, it is certain that there is not in uncomplicated myelitis that severe pain in the back and limbs which is brought on or aggravated by movement in spinal meningitis.

Absence of spasmodic symptoms.—Ollivier speaks of continuous contraction of the limbs as being met with "assez ordinairement," in chronic myelitis; but the cases cited by this excellent observer do not substantiate this statement. Thus, out of nineteen cases of myelitis, complicated and uncomplicated, acute and chronic, there are three only in which these contractions were present, and not one of the three can be cited correctly as a case of myelitis. In one of the three (No. 87) the sensibility was intact, and the disease of the cord confined almost exclusively to the anterior column; in another (No. 93) there was obtuse sensibility, and the disease was chiefly in the gray matter; and in the third (No. 94) sensibility remained, and there was no post-mortem examination to show what the disease in the cord really was. In each one of these cases, also, there were "head-symptoms" which do not figure in uncomplicated myelitis. Again, prolonged contraction of the extremities is a not unfrequent symptom in cases in which there is neither myelitis nor spinal meningitis—cases which come properly under the head of "spinal irritation," and about which more will have to be said in another section of this article. In these cases the contraction, instead of pointing to inflammation of the cord or its membranes, is really no more than one of a series of so-called hysterical phenomena. It is a sign of functional disorder only, and that it is so is evident (these among other proofs) in the sudden and complete way in which it passes off, as well as in the fact that it does not leave behind it any permanent organic traces. It depends, as it would seem, upon a state of irritation in some part of that track in which irritation gives rise to prolonged spasm—a state issuing, it may be, now and then in inflammation, but in itself, so far as the condition of the bloodvessels is concerned, diametrically opposed to inflammation. Nay, even in those exceptional cases of myelitis in which there is increased reflex excitability in the paralyzed limbs, it is difficult to connect these spasmodic symptoms with the inflammation. Dr. Brown-Séquard says: "When the dorso-lumbar enlargement is inflamed, reflex movements can hardly be excited in the lower limbs, and frequently it is impossible to excite any. On the contrary, energetic reflex movement can always be excited when the disease is in the middle of the dorsal region, or higher up." And again, when speaking of the reflex convulsions which may happen in the cases where the inflammation is in the middle of the dorsal region or higher up, he says, "Convulsions do not take place at the beginning of the inflammation, but some time after, and they recur by fits for months and years after." And this is precisely what happens. In a word, the truth would seem to be that these reflex spasmodic movements must be referred, *not* to inflammation in the lumbar enlargement of the cord, nor yet to inflammation

higher up in the cord; for in this case, to repeat what has just been said, "the convulsions do not take place at the beginning of the inflammation, but some time *after*, and they recur by fits for months and years *after*." They happen, as it would seem, *after* the inflammatory disorganization has interrupted the continuity of the cord, and produced a state of things analogous to that of a guinea-pig, or other animal, whose spinal cord has been cut across experimentally—a state of things of which increased reflex excitability in the paralyzed parts is one of the consequences. Nor is a different conclusion to be drawn from the occasional presence in the paralyzed muscles of a state which is analogous to or identical with the "late rigidity" of Todd. This "late rigidity" is very different from "early rigidity." In "early rigidity" the electro-motility of the muscles is increased, and the muscles relax during sleep, and to a less degree under the influence of warmth. The muscular contraction is evidently of the nature of spasm. In "late rigidity," on the contrary, the muscles are wasted, their electro-motility annihilated, and sleep or warmth do not tell in causing relaxation. This form of muscular contraction, indeed, if not identical with rigor mortis, is, as it would seem, more akin to this state than to spasm. In the case of myelitis which serves as my text, there was none of the painful muscular rigidity produced by movement which is so prominent a symptom in spinal meningitis. There was, indeed, no spasmodic symptom of any kind, with the exception of the rigor which ushered in the extension of the disease on the day after the admission of the patient to the hospital. And this absence of spasmodic symptoms would seem to be the rule in all cases of myelitis, acute or chronic. In children, it is true, myelitis may be ushered in by convulsion—in which case the convulsion manifestly represents the rigor which may usher in myelitis in adults, and as manifestly belongs to the precursory stage of irritation, and not to the state of actual inflammation—but even in children, unless there be some meningeal complication along with the myelitis, this preliminary convulsion would seem to be a rare phenomenon.

Want of control over the bladder.—This appears to be the earliest as well as the most constant of the symptoms of myelitis. It usually depends upon paralysis of the accelerator urinæ and compressor urethræ, but now and then it would seem to be connected, for a while at least, with a state of spasm in the latter of these muscles, in which case the dribbling away of the water or the introduction of a catheter will sometimes produce marked reflex spasms in the legs. I remember one case—a case in which the myelitis seemed to have interrupted the continuity of the cord high up in the back—where an attempt to use the catheter often gave rise to strong reflex spasms in both legs, and to a state of spasm in the urethra strong enough to prevent the passage of the instrument.

Want of control over the rectum.—In myelitis, paralysis of the sphincter ani is usually associated with paralysis of the accelerator urinæ and compressor urethræ. Now and then also, the sphincter ani, instead of being paralyzed, may be in a state of reflex spasm: thus, in the case to which I have just referred, the administration of an enema was sometimes rendered impossible by the spasm set up in the sphincter ani and in the femoral muscles by the pipe.

Absence of local spinal tenderness.—As in spinal meningitis, so in myelitis, absence of tenderness on pressure in any part of the spine would seem to be the rule, and not the exception. Ollivier, speaking of pain in the back in myelitis, says, "Elle n'est jamais rendue plus aiguë par la pression," and my own experience in the matter is, without question, to the same effect.

Altered sensibility to heat and cold, by which a feeling of burning is felt when a sponge soaked in moderately warm water or a piece of ice is applied to the spine immediately above the seat of inflammation.—Several years ago it was pointed out by Mr. Copeland that, when a sponge soaked in water a little above the temperature of the blood was passed along the spine from above downwards, it gave rise to the natural feeling of heat until it reached the inflamed part, and that then this feeling changed to that of burning: and more recently Dr. Brown-Séquard has shown that a similar result is arrived at by passing a piece of ice down the spine, the natural feeling of cold being felt until the inflamed part is reached, and then an unnatural feeling of burning. In many cases, no doubt all this would seem to be quite true, but not in all, perhaps not in the majority; and therefore it is impossible to look upon the feeling of burning thus produced as more than an occasional occurrence in myelitis.

Annihilation of reflex excitability.—What has to be said under this head has been anticipated when speaking of the absence of spasmodic symptoms in myelitis. It has indeed been seen to be the rule for all reflex movements to be annihilated or greatly weakened in the paralyzed parts, and that the apparent exceptions to this rule are to be explained, not by referring the increased reflex movement to myelitis, but by supposing the inflammatory disorganization to have interrupted the continuity of the cord and produced a state of things analogous to that of a

guinea-pig whose spinal cord has been cut across for experimental purposes.

Diminution of electro-motility and electro-sensibility in the paralyzed muscles.—Except in those few, very few, cases in which the reflex excitability is increased, the electro-motility and electro-sensibility of the paralyzed muscles are invariably diminished in myelitis. Where the reflex excitability is increased the electro-motility may also be increased, and so also may the electro-sensibility, but more generally the increase in the former property is without a corresponding increase in the latter. The paralyzed muscles are wasted in almost all cases, and relaxed also, except in those few cases in which the paralysis has lasted for a very long time and become associated with that state of "late rigidity" which, sooner or later, is often found to seize upon paralyzed muscles. Marshall Hall noticed the impairment of irritability in spinal paralysis, and was of opinion that an opposite state of things existed in cerebral paralysis. As was pointed out by Todd, however, this supposed distinction between spinal and cerebral paralysis does not hold good, the simple fact being that in the great majority of cases of cerebral paralysis the irritability of the paralyzed muscles, instead of being increased, is either not materially altered or else more or less diminished—most generally diminished in a very marked degree. In a word, the investigations of this very accomplished physician show most clearly that in cerebral paralysis the irritability of the paralyzed muscles is only increased in those comparatively few cases in which the paralysis is associated with "early rigidity."

Priapism.—It is difficult to attach any diagnostic value to this symptom. As in acute spinal meningitis, so in acute myelitis, it is sometimes present and sometimes absent, less frequently present in the latter affection perhaps than in the former.

Acidity of the urine.—Dr. Brown-Séquard says: "One of the most decisive symptoms in myelitis is alkalinity of the urine. There is no patient attacked with myelitis in the dorsal region of the cord whose urine is not unfrequently alkaline. At times, especially after certain kinds of food, the urine is acid, but the alkalinity soon returns." And no doubt the urine is very generally alkaline in myelitis, especially in those cases in which the paralysis of the bladder has led to secondary disease of this organ; at the same time, as in the case under consideration, the urine is too often acid to make it possible to insist upon alkalinity of the urine as a necessary feature in myelitis.

Dyspnœa.—Difficulty of breathing was a very urgent symptom in the case which serves as my text, and so it must be in every case where respiratory muscles are so gravely implicated in the paralysis, and where the lungs are so much engorged. Indeed, the usual way in which myelitis proves fatal is by compromising the sufficiency of the respiration. Now and then, especially when chronic inflammation affects the higher regions of the cord, the difficulty of breathing may occur in paroxysms not unlike those of asthma, but usually the difficulty shows itself rather as simple shortness of breath,—shows itself in a way which supplies another proof of the absence of the spasmodic element in the history of myelitis.

Want of power in the circulation.—There is little or no sympathetic fever in the most acute form of myelitis; and in the ordinary chronic forms, the feeble pulse, the œdematous condition of the paralyzed extremities, the disposition to passive engorgement in the lungs and elsewhere, and other symptoms of like meaning, show very plainly that the state of the circulation is eminently asthenic. It would even seem as if there were something in the very fact of myelitis which has a positive influence in subtracting power from the circulation—which exercises a devitalizing influence upon the system generally.

A tendency to bed-sores, wasting, and other signs of defective nutrition in the paralyzed parts.—Sooner or later, generally at a very early date, a marked disposition to bed-sores in places where paralyzed parts are subjected to pressure is apt to show itself in myelitis, and so also are other signs of defective nutrition in the same parts, such as œdema, dryness and scurfiness of the skin, and a wasted and flabby state of the muscles. So marked, indeed, is this impairment of nutritive power in these paralyzed parts, that it is only by very great care that bed-sores and the other lesions which have been mentioned can be prevented.

Absence of head-symptoms.—In cases where acute myelitis attacks the higher portions of the cord, there may be, and there in all probability will be, various "head-symptoms"—vertigo, singing in the ears, grinding of the teeth, delirium, convulsion, coma, or others—but these cases, to say the least, are not common. Whether acute or chronic, indeed, myelitis is much more apt to attack the lower portions of the cord than the upper, in this respect differing from spinal meningitis; and when it attacks the upper portions of the cord, and its symptoms present cerebral complications, the chances are that the case is not simple myelitis, but myelitis with more or less spinal meningitis in addition.

When the cord is affected generally, the symptoms of myelitis will not differ

greatly from those which are present in the case which has been given; when the inflammation is more localized, the symptoms will vary accordingly. If, for example, the inflammation be limited, as it usually is, to the lumbar enlargement of the cord, the level of the paralysis and anæsthesia will be proportionally low down; and if the extreme end of the cord only be affected, it is possible that the legs may escape altogether, and the bladder and anus be alone at fault. As indeed the level of the inflammation in the cord falls or rises, so must the level of the paralysis and anæsthesia fall or rise also. Exaggerated reflex movements in the inferior extremities will also (in all probability) be associated with the paralysis and anæsthesia, if the lower part of the cord be sound and the inflammation confined to a portion of the cord higher up. Again, the symptoms which are present when the inflammation is limited to a part only of the thickness of the cord will be different in many respects from those which are met with when the whole thickness is affected. If, for example, a portion (the upper half inch of their course excepted) of the anterior columns be affected solely, there would be paralysis without anæsthesia; or if the posterior columns were alone affected, there might be incoördination of movement and some hyperæsthesia instead of paralysis and anæsthesia. In short, the variations of symptoms, which occur where myelitis is restricted to particular parts of the cord, can only be properly intelligible to him who has clear notions respecting those physiological matters which were glanced at in the preliminary remarks, — which were then glanced at chiefly in order to avoid perplexing physiological digression and discussion in the present place among others. I will, therefore, assume that what was said in the preliminary glance at some points in the physiology of the spinal cord, will serve to explain sufficiently the variations of symptoms which may be expected to exist when the integrity of particular parts of the spinal cord is destroyed by myelitis or in any other way: and, for the rest, I will only say that myelitis may be chronic and subacute as well as acute in its course, and that these several varieties interblend insensibly the one with the other.

2. Post-mortem Appearances. — Myelitis may result either in softening or in hardening of the spinal cord. Most frequently the cord is broken down, reduced to a yellowish or reddish cream-like consistence; the color, derived from the admixture of pus or blood-corpuscles, being more yellow or more red according as the one or the other of these corpuscles predominates. This softening may affect the whole thickness of the cord, or certain parts more than others, the gray matter especially; it may extend from one end of the cord to the other, or it may be confined to certain regions, in which latter case the part most likely to be affected is the lumbar enlargement; and it would often seem to have its starting-point in the central gray matter, which is the most vascular part of the cord. In the first stage of myelitis this central gray matter has a rosy or vinous tinge, which is not natural to it; it is plainly more vascular than it ought to be; and, in short, it has undergone the very same change which is met with in the gray matter of the brain in encephalitis. Sometimes the spinal cord is considerably swollen, and sometimes the surface may have a nodulated appearance in certain parts, from the membranes having yielded at these points to the blood which may have escaped, or to the pus or other fluid which may have collected, underneath. Not unfrequently small collections of blood are met with in the softened nerve tissue, especially in the position of the central vessel, so that the first impression upon opening the cord may be that of hemorrhage rather than that of myelitis. One remarkable feature of inflammatory softening, says Dr. Todd, is that "it exhales a marked odor of sulphuretted hydrogen, and so indicates a rapid advance of putrefaction;" and again, "It is a fact deserving of attention that the substance of the spinal cord softens very rapidly after death, the lapse of half an hour, during which the nervous substance has been exposed to the air, often producing a manifest alteration." Indeed, there are reasons for believing that the amount of disorganization met with in the cord after death does not necessarily represent the exact amount which existed during life, and that a cord which is found to be broken up after death almost utterly, may have retained during life sufficient integrity to allow of the transmission of certain sensitive and motor impressions. On this view the return of slight sensation in the urethra and rectum shortly before death, and the preservation of the power of moving and feeling in the arms, which were noticed in the case which serves as my text, are not altogether unintelligible.

Induration, the other result of myelitis, is looked upon by some as a stage always preceding softening, but it would rather seem, to mark, as Ollivier supposed, a less acute form of inflammation. In it the fibrinous products of the inflammation seem to have been more organizable. The cord thus indurated varies greatly in appearance; it may be almost as pale, bloodless, crisp, and hard as cartilage; it may be more or less red and vascular;

and in either case, when examined under the microscope, its proper tissues are found to be broken up and destroyed almost as effectually as they are when the cord is softened. A cord which is indurated has usually a shrunken appearance, but it may be swollen considerably. There is no doubt an induration of the cord, as well as a softening, which cannot be referred to myelitis, and which must not be confounded with that which is the result of inflammation; but I must not stay to point out the differences, nor yet to do more than say that in myelitis there will in all probability be found, in addition to the signs which have been indicated, engorgement of the lungs, kidneys, and other viscera, possibly more marked vascular changes, with bed-sores, œdema, dry and scurfy skin, wasted muscles, and other signs of defective nutrition in the paralyzed parts.

3. Causes.—Nothing very much to the point can be said under this head, and the only remark I feel called upon to make is this, that as in spinal meningitis a rheumatic habit has been found to figure more or less conspicuously among the causes of the malady, so here a like position would seem to be due to a strumous habit. I would also confess to a growing impression that myelitis may not unfrequently be connected more with excess of sexual indulgence than with any other single cause, but I cannot say that this impression has yet taken the form of a definite conviction.

4. Diagnosis.—In dealing with the symptoms of myelitis it has been shown that these are very different from those of spinal meningitis—so different as to make it difficult to confound them, if only moderate care be taken in realizing them. In spinal meningitis the most prominent symptom is pain in the back and extremities, produced or aggravated by movement; in myelitis pain of any kind has scarcely a title to be reckoned among the symptoms, pain produced by movement certainly not. In spinal meningitis the sensibility is somewhat exalted, in myelitis it is abolished. In spinal meningitis there is muscular weakness, and the muscular movements are fettered by pain, but there is no true paralysis: in myelitis, paralysis is the symptom of symptoms. In spinal meningitis there is a state simulating trismus and tetanus, a state of muscular rigidity half voluntary as to its character, of which the object is to prevent certain movements which give rise to pain; in myelitis the muscles are limber, and there is usually an utter absence of any symptom akin to tremor, convulsion or spasm.

Nor need the symptoms of common paraplegia (resulting from chronic myelitis) be confounded with those of locomotor ataxy. In common paraplegia there is paraplegia more or less marked of the lower extremities, and the nutrition and irritability of the paralyzed muscles are, as a rule, unmistakably impaired; not so in locomotor ataxy. In common paraplegia the paralysis extends to the bladder and sphincter ani, and the sexual power is greatly weakened, if not altogether abolished; not so, or not to anything like the same degree, in locomotor ataxy. In common paraplegia the characteristic neuralgic pains of locomotor ataxy are wanting, and numbness is nothing like so prominent a symptom as in the ataxic disorder. In common paraplegia, where walking is possible, the gait—instead of being precipitate and staggering, the legs starting hither and thither in a very disorderly manner, and the heels coming down with a stamp at each step, as in locomotor ataxy—is hampered and slow, each leg being brought forward with evident difficulty, even with the help of an upward hitch of the body on the same side, and the part of the foot first coming in contact with the ground being, as a rule, not the heel, as in ataxy, but the toes. In common paraplegia, impairment of sight or hearing, or strabismus, or ptosis, or injection of the conjunctivæ, or contraction of the pupils, frequent if not constant symptoms in locomotor ataxy, form no part of the history. In fact, in these respects, and in others of minor importance which might be mentioned, the histories of common paraplegia and locomotor ataxia are so different that it is not easy to see how, with only a moderate amount of care, the two can be confounded.

Now and then, it is true, instances occur in which it is not so easy to distinguish this gait of common paraplegia from that of locomotor ataxy—cases in which the weakened muscles contract somewhat spasmodically when put in action, but, as a rule, the gait in common paraplegia and in locomotor ataxy is sufficiently characteristic to make it difficult to confound these two affections.

In cases where the myelitis is confined to the posterior columns of the cord, the symptoms will be those of locomotor ataxy rather than those which have been ascribed to myelitis; for, so far as the production of symptoms is concerned, it is of no moment whether the disease disorganizing the posterior columns be inflammatory or non-inflammatory, acute or chronic; and in other cases of local myelitis symptoms are sure to be present which cannot fail to lead to a correct diagnosis, if what was said in the preliminary remarks upon the physiology of different parts of the spinal cord be borne

in mind in interpreting them. Indeed, with what is known of the physiology of the spinal cord, there need not be much difficulty in determining the whereabouts of local mischief in the cord.

That myelitis cannot well be confounded with other spinal disorders—spinal congestion, tetanus, spinal irritation, and the rest—will be seen readily enough when a clear idea of these disorders has been realized, and only then; and this being the case, it is best to waive these questions in diagnosis until the fitting opportunities for dealing with them present themselves.

5. Prognosis.—Acute myelitis affecting any considerable extent of the spinal cord is, without doubt, a very grave disorder. It may be fatal in fifteen or twenty hours, and it is seldom that life is prolonged beyond the end of the second week. Instances of recovery are on record, it is true, but these are very few in number, and of them there is, perhaps, no single one in which the correctness of the diagnosis may not be impugned. Even chronic myelitis is a very grave disease; for though life may be prolonged, especially where the disease is confined to the lower part of the cord, the mischief once done seems to be in a great measure irreparable. At the same time it is only right to say that of late years the results of treatment have been much more satisfactory, and that it is possible now to hope where there was little room for hoping formerly.

6. Treatment.—There appears to be little room for what is called active treatment even in acute myelitis. The inflammation is evidently of a very low type, and, reasoning from what is known of its beneficial action in erysipelas and in some other low forms of inflammation, it seems to me that sesquichloride of iron would be likely to be of more real service than iodide of potassium. Indeed, I should be disposed, until I know of a better plan, to trust chiefly to full doses of this preparation of iron, to food and wine, and to the position recommended by Dr. Brown-Séquard for draining away blood from the spine—a position in which the patient is made to lie upon his abdomen or side, with his hands and feet in a somewhat dependent position.

With regard to the good or bad effects of belladonna, or ergot, or strychnia, it is not very easy to arrive at a satisfactory conclusion. I agree with Dr. Brown-Séquard in thinking that belladonna and ergot may have the effect of counteracting a hyperæmic condition by causing contraction in the vessels, and that the vessels of the spinal cord may, perhaps, respond most readily to their action, but not as to the indications for employing these remedies. Pain and spasm are, to Dr. Brown-Séquard, signs of hyperæmia: to me, except the pain produced by movement, they are signs of irritation only—of a state which is connected, not with hyperæmia, but with anæmia, a state of contraction of the vessels which may pass into relaxation, but which need not necessarily do so; and, therefore, to me pain and spasm, instead of being indications for the employment of belladona or ergot, are in very deed contra-indications. Nor can I agree in thinking that strychnia acts by increasing the amount of blood in the spinal cord and in its membranes, and that on this account it is contra-indicated in hyperæmic conditions of these parts. Strychnia, without doubt, produces tetanic spasms and other unequivocal signs of spinal irritation, but it is begging the question altogether to suppose that the strychnia increases the amount of blood in the cord and its membranes, that this increase of blood augments the vital activity of the cord, and that the spasms and other signs of irritation attest this augmentation of vital activity. Indeed, so far from this being a necessary conclusion, all the evidence presented in the preliminary remarks, as it seems to me, points in the opposite direction, and connects the state of irritation of which the spasms are the signs, not with a hyperæmic condition, but with an anæmic; and most assuredly I know of nothing in the history of myelitis or spinal meningitis which is calculated to invalidate this conclusion. Moreover, the investigations of Dr. Harley upon the action of strychnia upon the blood go to show that this action is really equivalent to loss of blood in that it directly interferes with the proper arterialization of the blood. In a word, I cannot find any fundamental difference between the action of belladonna, ergot, and strychnia upon the bloodvessels, neither can I understand why strychnia, properly used, might not be of as much service as belladonna or ergot in lessening a hyperæmic condition of the cord. For my own part, however, I confess to a feeling which makes me hesitate to employ either belladonna, or ergot, or strychnia in myelitis, or in any analogous condition, until I know more of their action, or until I have more unequivocal empirical evidence of the good resulting from their use.

In chronic cases the one grand indication of treatment, as it seems to me, is to improve the nutrition of the cord, and the medicines best calculated to carry out this indication are cod-liver oil, sesquichloride of iron, phosphorus in one form or other, arsenic, and possibly bichloride of mercury, which latter preparation, when properly used, I believe to be tonic and antiseptic in a high degree, and in

many respects much more analogous in its action to arsenic than to any of the proto-compounds of mercury in common use.

The local means for promoting the recovery of the paralyzed muscles are certainly of not less importance than the general means, possibly of much greater importance, and these local means are very various. The efficacy of frictions and shampooings appears to be indisputable. The efficacy of proper movements can only be doubted by those who are unacquainted with the results arrived at by the "movement cure," and by systematic movements of one kind or another, with or without the help of mechanical apparatus. The efficacy of faradization has been abundantly proved, and there is good reason to believe that this is not the only mode of using electricity which will be of great service; that in fact statical positive electricity, or the interrupted galvanic current, or the application of the galvanic current in such a way that the paralyzed nerve is acted upon chiefly by the positive pole—a mode of using electricity about which I have spoken elsewhere, and which I have used extensively during the last five or six years—will often be of great service in proper cases. Indeed I should think that the treatment was wanting in very essential particulars if these local means, one and all, were not associated with the general means of treatment, and employed systematically and perseveringly; and especially I should regard it as a great blunder if these local means were deferred so as to allow the paralyzed muscles to lose what when lost is not easily recovered—that is, their irritability and healthy organization.

There are also other local measures which are of great service in the treatment of paralysis, and one of these to which I am disposed to attach especial importance is to protect the paralyzed parts from cold. In many cases, as is well known, these paralyzed parts are cool, and in not a few instances, where the paralysis is incomplete or associated with early rigidity, this paralysis and rigidity is greatest when these parts are coldest. For example, it is no uncommon thing for a partially hemiplegic patient whose paralyzed fingers are contracted, stiff, and altogether useless when acted upon by cold, to be able to open his hand and use his fingers with comparative freedom when the hand is warm in bed, or placed in a warm bath, or held a while before the fire. At any rate, I have long been satisfied that the well wrapping up of the paralyzed parts in woollen, or silken, or india-rubber coverings is an important help in treatment.

It would also seem that good of the same kind, much good, may be got from an exhausting apparatus made on the principle of Junot's boot. The effect of such an apparatus, properly used, is to make the paralyzed parts warmer at the time, and to enable them to preserve this warmth for a considerable time—to produce a change in the circulation, which must have a good effect upon the nutrition and irritability of the paralyzed muscles.

It is also more than probable that electricity may be of service in improving the condition of the circulation in the paralyzed parts, for an increased feeling of warmth in the paralyzed parts is the result of faradizing these parts, or of electrifying them with statical electricity; indeed I have been more than once disposed to think that the beneficial effects of electricity in the resuscitation of paralyzed parts are as much brought about indirectly by changes produced in the circulation as by changes wrought directly in the nerves and muscles.

As regards the necessity for tenotomy and the use of orthopædic apparatus in certain cases, it is difficult to speak to any good purpose. I shall have to refer to these subjects when speaking of infantile paralysis, and here I will only say, that in many cases, in children especially, the cure will be greatly facilitated by tenotomy and orthopædic apparatus, and that it is not always easy to decide between the cases in which these measures are desirable and those in which they are not desirable. [For *Polio-myelitis* (inflammation of the gray substance of the cord) see Infantile Paralysis.—H.]

III. Spinal Congestion.

Spinal congestion, or plethora spinalis, is not less definite in its history than myelitis or spinal meningitis, neither is it of less practical interest. In the sequel, indeed, it may appear, not only that spinal congestion is fully entitled to the place which has been assigned to it in the catalogue of diseases, but also that it really comprehends more than one spinal disorder which is now known under a different name.

1. Symptoms.—As an instance of well-marked spinal congestion, I take the notes of a case under my care not long ago.

Case.—Mary L., aged 28, but looking very much older, married but never pregnant, was admitted into the Westminster Hospital on the 12th of June, 1866.

(*a*) With the exception of being able to turn her head on the pillow and to move the fingers and toes a little, all power of voluntary movement appears to be wanting. The symptoms chiefly complained of are tingling in the tips of the fingers

and toes, a dull burning aching along the back and in the limbs, and a feeling of being "tired to death." If altered in anywise, the sensibility to touch, pain, tickling, and differences of temperature, is somewhat more acute than natural. The spine is nowhere tender on pressure, but the dull burning aching in this region is increased by the application of a sponge soaked in hot water. The soles of the feet may be tickled without giving rise to undue reflex movements. The bladder and bowels act properly. The mind is not at all affected. The state generally is evidently one of great exhaustion and prostration without fever, the pulse being quick, unsteady, and very compressible, the respiration shallow, and curiously interrupted by sighs.

(*b*) Three weeks ago, menstruation, which had only just begun, was suddenly checked by an alarm of fire. This was shortly before bedtime. The next morning, after a very sleepless and miserable night, the state had become very much what it now is. Up to this time the patient had never been obliged to remain in bed a single day on account of illness. She had often been weak and ailing, and she had suffered a great deal at the menstrual periods from pain and weakness in the back and legs, and that is all. She also appears to have sprung from a tolerably healthy stock.

(*c*) Within the first fortnight after admission to the hospital, the tingling in the tips of the fingers and toes came to an end, and so did the aching in the back and limbs. A week later the arms as well as the hands could be moved a little. At the end of six weeks the legs remained almost as helpless as at first, but the arms and trunk had so far recovered power as to allow of a change from the lying to the sitting posture without any great difficulty. At the end of twelve weeks it was possible to get out of bed, and, with the help of a stick, to move to the table in the centre of the ward. On the 3d December, five months after admission, the patient left the hospital convalescent. All this while the appetite was tolerably good, and the bladder and bowels acted properly. Now and then, in the progress towards recovery, especially about the menstrual periods, there were short relapses in which the tingling in the tips of the fingers and toes, and the aching in the back and limbs, came back, and the paralytic weakness of the muscles was almost as great as at first, —in which the ground already gained seemed all but lost. Now and then, also, the nights were disturbed by a distressing state of shortness of breath, not amounting to asthma. Before the legs recovered power their muscles were somewhat wasted, but not considerably so; indeed, neither here nor elsewhere was the paralysis accompanied by any marked wasting of the muscles, or by any appreciable impairment of electro-sensibility or electro-contractility. Moreover, any movement, whether active or passive, had always the effect of relieving rather than of increasing the aching in the back and limbs, when this symptom was present. The treatment pursued was chiefly rest, good living, hypophosphite of soda, nux vomica now and then in small doses, cod-liver oil, and faradization.

Assuming, as I well may, this to be a case of well-marked spinal congestion, I take as points of comparison between it and other cases of the kind, general and partial, these :—suddenness of access; incomplete paralysis in a paraplegic form; no numbness; tingling in the tips of the fingers and toes; no exaggeration of reflex excitability in the paralyzed limbs; no want of control over the bladder and bowel; no spinal tenderness; aching in the back increased by warmth; pains in the back and limbs not increased by movement; no marked impairment of the electro-contractility and electro-sensibility, and no material wasting, of the paralyzed muscles; no feverishness; breathlessness; no bed-sores; proneness to relapses.

Suddenness of onset.—To be well, or comparatively well, on going to bed, and to be paralyzed in the morning, as in the case which I have given, is no uncommon thing in spinal congestion. It is indeed the rule rather than the exception for the illness to be spoken of as a "stroke" by the sufferer.

Incomplete paralysis in a paraplegic form.—Paralysis, often all but complete, but never quite so, and taking the paraplegic form, must be looked upon as the rule in spinal congestion. The paralysis is decidedly paraplegic in the end, and it may be so from the beginning, but not unfrequently one leg or one arm is affected before the other, and occasionally the leg and arm of the same side may for a short time be affected, as in hemiplegia, before the disease extends to the leg and arm of the other side. Not unfrequently there remains a difference in the degree of paralysis on the two sides, one leg or arm being more affected than its fellow. In cases where the congestion of the cord is general the arms as well as the legs are paralyzed, the former perhaps as much as the latter; but in the common run of cases, where the congestion is confined chiefly to the lumbar region of the spine, the legs are exclusively or chiefly affected.

No anæsthesia.—Numbness is a symptom of myelitis, but not of spinal congestion. In the latter disorder, indeed, instead of numbness there is occasionally a state of things which may be spoken of as hyperæsthesia: thus, in a case very

like the one I have given, which came under my notice in private practice about three years ago, the weight of a single bed-sheet was distressingly heavy to the patient, and long-continued aching of the paralyzed arms and legs was produced by handling them ever so lightly.

Tingling in the tips of the fingers or toes of the paralyzed limbs. — This symptom is almost always present at one time or other, coming and going and staying a longer or shorter time, often, as it would seem, very capriciously. One is glad to get rid of it, for while it remains it is difficult altogether to put aside the fear lest the state of the cord should pass out of simple spinal congestion into the graver disease of myelitis.

No exaggeration of reflex excitability in the paralyzed limbs.—Increased disposition to reflex movement is usually regarded as one of the symptoms in spinal congestion. It is supposed that the greater afflux of blood to the spinal cord must bring with it greater reflex excitability. I believe, however, that this supposition is not at all borne out by the facts. I believe, indeed, that the moderate reflex excitability in the case under consideration is not at all exceptional, and that it is the rule in all cases of spinal congestion for this manifestation of muscular contractility to be, if altered at all, diminished rather than increased.

No paralysis of the bladder or sphincter ani.—In myelitis, paralysis of the bladder or sphincter ani, more or less complete, is a prominent symptom : in spinal congestion, on the contrary, these symptoms are absent, except in those mixed cases where there is reason to believe that some degree of myelitis is also present. In the case which I have given there was not the least want of control over the bladder or bowel from the beginning to the end.

No tenderness on pressure along the spine. —Absence of spinal tenderness I believe to be the invariable rule, not only in spinal congestion, but also in myelitis and spinal meningitis. I believe, indeed, that spinal tenderness is a sign of the presence of that functional disorder of the cord which is usually called spinal irritation, and that it does not accompany the graver diseases of the cord which have been named when they are uncomplicated with spinal irritation. Upon this subject I shall have more to say presently.

Dull aching along the spine increased by warmth.—I have noticed this symptom in three cases of well-marked general spinal congestion which have come under my own observation, and in many cases of partial congestion ; and I am disposed to think that this will prove to be one of the points of difference between spinal congestion and spinal irritation. I have also noticed the same symptom in myelitis and spinal meningitis, and therefore I cannot regard it as having any special connection with spinal congestion. In fact, so far as my experience goes, I can say that this symptom is likely to be met with in congestive or inflammatory diseases of the cord, but not in spinal irritation simply ; and that in this latter case, the local application of warmth to the spine is more likely to relieve pain than to cause it.

Pains in the back and limbs not increased by movement.—This symptom has some claim to be regarded as constant. The aching would seem to go and come with the congestion ; and the fact, for fact it seems to be, that it is not increased by movement, may help to distinguish spinal congestion from spinal meningitis, for in the latter affection movement of the limbs, whether passive or active, is attended with pain in the parts moved and in the back.

No marked impairment of electro-contractility and electro-sensibility in, and no wasting of, the paralyzed muscles.—In myelitis the paralyzed muscles are prone to waste and to lose their electro-contractility and electro-sensibility, and herein, therefore, would seem to be a marked difference between this disorder and spinal congestion ; for, so far as I know, the contrary state of things invariably holds good in spinal congestion.

No feverishness.—This is no special feature ; indeed, fever would seem to have little to do with any affection of the cord, not even excepting meningitis in its most active form.

No bed-sores.—A marked disposition to bed-sores would seem to be the rule in myelitis, but not so in spinal congestion or spinal meningitis. Upon this point, more than upon many others, there is tolerable unanimity of opinion.

Shortness of breath.—Where the spinal congestion is at all general, this state of things may be readily accounted for by the paralytic weakness of muscles concerned in respiration. In the case which serves as my text, the occasional shortness of breath is noticed as not amounting to asthma ; and this is a point of some interest, for it may be supposed that the difficulty of breathing would have taken this form—would have had something of a decidedly spasmodic character—if the congested condition of the cord involved, as it is supposed to do, an exaggeration of reflex excitability.

Proneness to relapse.—Whether this may prove to be a constant feature in spinal congestion remains to be seen. That it is not an uncommon one is, to say the least, highly probable.

Spinal congestion varies greatly in its degree and in the extent of cord implicated. Limited to the lumbar region, and carried to a degree which produces

not paralysis, but weakness more or less approaching to paralysis in the legs, it is common enough; indeed, many women seem to suffer from it before every menstrual period; and between this partial and incomplete form and the general and complete form, of which the case which has been given is an instance, there are all possible grades of transition. It would seem to be most common in women, but it is not peculiar to the female sex or to any age. The onset of the disorder is generally sudden, in relapses as well as in original attacks; and the cases do not at all divide themselves into acute and chronic as do the cases of many other disorders.

2. Post-mortem Appearances.—These appearances are very vague and unsatisfactory, at most being simply some engorgement of the veins of the spinal cord and membranes, with some excess of the spinal fluid, both of which phenomena, as will be easily understood, are not very unlikely to escape detection unless the post-mortem examination be conducted with unusual care. With the exception of this engorgement and serous effusion, the only morbid sign which has been noticed (and this by no means constantly) is slight infiltration with blood of the cellular tissue exterior to the dura mater. In all uncomplicated cases, the structure of the cord and of its membranes is in nowise altered.

3. Causes.—As in the case which I have given, the suppression of the catamenia would seem to figure most conspicuously among the causes of spinal congestion, and next to this the cessation of hemorrhage from piles. Beyond this it is difficult to single out any one cause which has a just claim to be considered as at all special: and, for the rest, nothing further need be said except this,—that spinal congestion is not unfrequently a consequence of pulmonary or abdominal congestion or inflammation—a consequence, perhaps, which has often more to do in compromising the safety of the patient than the primary disorder itself.

4. Diagnosis.—Paraplegic paralysis is a symptom common to spinal congestion and myelitis, with this difference, that it is less complete in the former affection than in the latter. The paralysis is associated with anæsthesia in myelitis; not so in spinal congestion. The control over the bladder and bowels is lost in myelitis; not so in spinal congestion. The paralyzed muscles are prone to waste and lose their electro-contractility and electro-sensibility in myelitis; not so in spinal congestion. The absence of anæsthesia would seem, indeed, to connect spinal congestion more closely with spinal meningitis than with myelitis, and so also would the pain in the back and aching in the limbs; but the pain and aching in spinal congestion cannot well be confounded with the pain which is met with in spinal meningitis, for the pain in this latter affection is produced by movement and accompanied by rigidity, whereas the pain in the former affection is not produced and accompanied in this manner. Hysterical paralysis, so called, agrees with the paralysis depending upon spinal congestion in some respects, but not in others. It agrees in that the paralyzed muscles are neither prone to waste nor to lose their electro-contractility; it disagrees in that numbness is a prominent symptom, more prominent even than the paralysis, and that the electro-sensibility of the paralyzed muscles is either annihilated or very much diminished.

5. Prognosis.—Recovery is the rule, no doubt, in cases of spinal congestion, but there is no difficulty in finding cases in which the disease has been fatal, and quickly fatal too. In the partial form, affecting the lumbar portion of the cord only, spinal congestion may come and go quickly without any great damage being done; but in the cases in which the cord is more extensively and more profoundly affected, as in the case which has been cited, recovery may occupy a considerable time. Thus, of the cases recorded by Ollivier, No. 55 remained in hospital nearly five months, No. 56 two months, No. 57 three months, and No. 58 "assez longtemps." Recovery is slow, it may be, because time is required for the absorption of the excess of the spinal fluid to which the state of spinal engorgement had given rise.

6. Treatment.—What has been said respecting myelitis must be supposed to apply here equally. Indeed, the only special remark which appears to be called for in this place is this,—that in cases where, as very generally happens, the spinal congestion can be referred to suppression of a menstrual or hemorrhoidal discharge, the primary indication would appear to be the setting up of an equivalent discharge by applying leeches to the os uteri or to the anus.

IV. Tetanus.

Tetanus is unhappily no rare or unfamiliar malady. The name, from τείνω, I stretch, refers to that rigid and cramped condition of the muscles which is the most characteristic symptom, and which, in sober earnest, is suggestive of rigor mortis, not only *in posse* but actually *in esse;* for there

are some cases in which, without any interval of relaxation, tetanic rigidity at once passes into cadaveric rigidity. Hydrophobia alone excepted, tetanus is at once the most appalling and the most perilous of all spasmodic diseases.

1. Symptoms.—As an instance of well-marked tetanus, I take the notes of a case which I happened to see from the beginning to the end ten years ago.

Case 1.—Patrick M——, a fair, slightly-built, delicate-looking man, unmarried, aged 27, the coachman of a gentleman then under my care. On the 21st of April, 1861, meeting him as I was leaving the house of his master, he took the opportunity of saying that he was not well enough to bring round the carriage, and of asking me what he had better do. What he complained of chiefly were a stiff neck and sore throat, with a feeling of weakness and illness. The stiff neck and sore throat made their appearance for the first time this day; the feeling of illness and weakness has been present for the last three days. The mouth cannot be opened so as to allow a fair look at the tongue, and a meal, it appears, has just been left unfinished, not for want of appetite, but simply on account of the difficulty experienced in masticating and swallowing the morsels. There is no feverishness.

P. M. ascribes his second indisposition to having been out with the carriage several hours in the wet and wind three nights ago, and he says further that he is liable to colds. Before speaking to me, he had taken some opening medicine which a chemist had prescribed and prepared for him, and he thinks that this dose may account for the fact of feeling so ill and weak at the present moment. Some simple treatment was recommended, and I took my leave, not at all divining what was so soon to follow.

April 22.—Receiving information that this poor fellow was very ill, I went round to see him at his lodgings. I found him strangely altered. His teeth were firmly and inseparably clenched, and he looked literally like an old man—so like, that his mother, who lived with him, said that she could have thought his father had come back to life if only his hair had been gray. His voice had also become so low and indistinct as to make it difficult to catch what he said. The medicine given by the chemist yesterday, it appears, has purged him violently several times in the night, and more than once while at stool he has been seized with acute pain in the pit of the stomach, which took away his breath, and made him think he was going to die. It was in the night, while at stool, that the jaws became closed. I wished him to go to the hospital, and he was willing to do so, but his mother would not consent. Eggs beaten up with brandy were ordered to be given repeatedly, and every three hours a draught containing five grains of quinine and half a drachm of Hoffmann's anodyne. I now noticed on one of the fingers, which was tied up in a piece of rag, a small wound, healing and apparently healthy, the result of a tear by a rusty nail about a fortnight ago.

On a second visit, later in the day, I found that repeated attempts had been made in the interval to give the food and medicine, but with very trifling success. There was no great difficulty in getting the food or medicine into the mouth, for almost all the teeth on the right side were gone, but the attempt to swallow brought on spasm in the throat, and on more than one occasion the spasm forced the greater part of what was taken back through the nostrils. And this difficulty was all the more distressing, because a feeling of hunger prompted the patient of his own accord to make frequent attempts to swallow. The chief complaint now was of a dragging pain at the pit of the stomach, piercing through to the back. In answer to a question whether he could sit up in bed, he said, "I think I am too stiff to do so," and then he tried to sit up, and succeeded after making two or three abortive attempts. While sitting up, I found that he could scarcely move his head, and that the muscles of the neck and back were very stiff and hard. I had only just noticed these phenomena when the noise caused by the upsetting of a chair brought on a fit of spasm, in which the patient was suddenly thrown backwards upon the bed with considerable force, and left resting upon his head and heels, in a state of complete opisthotonos—a state so complete as to make it possible for me to pass my hand under the loins without touching either the body or the bed. This severe spasm lasted not less than a couple of minutes, and the only muscles which did not seem to be implicated in it were the abdominal, those of the arms and hands, and those of the eyeball. In this spasm the complexion became dusky and livid, and the features altered in a frightful manner, the angles of the mouth being drawn upwards and outwards so as to give the expression known as the *risus sardonicus*, the set teeth being slightly uncovered, the nostrils spread, the eyes staring and prominent, the brow knit, the hair bristling—the complexion and features became changed, that is to say, as they are changed in sudden suffocation. All this while, too, the skin generally was dusky and hot and drenched in perspiration. For some time after this spasm had passed off the patient remained moaning, and unable to speak audibly, and then he said, "that pain will kill me if it comes back." I noticed, also, that there re-

mained after this spasm a state of tetaniform rigidity and contraction, by which no inconsiderable degree of opisthotonos was still kept up. The eggs and brandy and the medicine were ordered to be given by enema.

April 23.—Two attempts were made to administer the enemata ordered over night without success, the irritation of the pipe in each instance bringing on a fit of spasm; indeed, all that it has been possible to give since my last visit have been a few sips of wine and water. There has been no sleep whatever during the night. During the last eighteen hours several fits of spasm like the one described have occurred, and the permanent rigidity and contraction remaining between the fits have increased. The abdominal muscles, which were not at all implicated yesterday, are now as hard and stiff as those of the neck, back, and legs. The pulse is quick (about 140), weak, and somewhat irregular: the breathing is shallow, hurried, and frequently checked by gasps and catches, even when it is not interrupted by the fits of spasm.

No material change has taken place since the morning. On one occasion in the course of the day an egg beaten up with some brandy has been swallowed, but all other attempts to administer food or medicine, whether by the mouth or by the rectum, have been rendered abortive by the fits of spasm to which they gave rise.

April 24.—Again the night has passed without sleep, and to-day the constant tetaniform contraction has become almost universal. In fact, the only muscles which are not obviously affected are those of the hands, and tongue, and eyeball. The fits of spasm, also, are now more frequent and severe, being not more than fifteen or twenty minutes apart, and lasting until death from suffocation seems even more than imminent; they are brought on by the most trivial causes—an attempt to swallow, a draught of air, the simple straightening of the bedclothes—or they come on without any apparent cause. There is no improvement in the breathing and pulse, but if anything a change for the worse. During the fits the skin is hot, dusky, and drenched in perspiration: in the intervals it has an ominous coolness and clamminess. The mouth is full of viscid frothy saliva, and there is much thirst. While I was present a small quantity of dark urine was passed slowly and with some difficulty, and this appears to be the only time the bladder has acted for at least twenty-four hours. The pupils are large, especially in the paroxysms.

Shortly before I went again, at the end of the day, there had been a momentary snatch of sleep, which had been abruptly brought to an end by an attack of opisthotonos, in which the tongue or cheek had been bitten, and now the frothy viscid saliva which filled the mouth to overflowing was deeply crimsoned with blood—a ghastly addition to a countenance already overcharged with horrors. During the last six hours the paroxysms have been less frequent and severe, but the vital powers are evidently fast ebbing away. "I cannot get my breath," was the answer slowly and almost inarticulately given to the question, "Have you much pain?"

Death happened about midnight, an hour after I had taken my leave, after a paroxysm of opisthotonos of no special violence, brought on, as it would seem, by an attempt to wipe away the bloody saliva from the lips. When I left the mind was perfectly clear and collected, and at no time, either before or after, was it otherwise.

For the rest it only remains to add (for the objections made to a post-mortem examination were insuperable) that the countenance appears to have retained after death the aged expression it had before death, and that the corpse when "laid out" was found to have stiffened without losing altogether the opisthotonic attitude. The mother of the patient is my only authority upon these points, for unfortunately it did not occur to me to make inquiries respecting them before the funeral had taken place.

In order to realize the points of resemblance and difference between this case and other cases of the kind, the course I propose to pursue is to take one after the other, as the points demanding attention, these—permanent muscular contraction, beginning by causing trismus, ending by causing opisthotonos, and implicating when at its height almost all the voluntary muscles except those of the hands, the eyeball, and the tongue: pain at the pit of the stomach, piercing through to the back; difficulty of swallowing, from the occurrence of spasm: fits of painful spasm in the permanently contracted muscles; risus sardonicus, and an aged expression of countenance; apnœa in the fits of spasm, and more or less dyspnœa at other times; increased temperature, without true fever; increased reflex excitability; absence of sleep; absence of numbness or tingling; absence of "head symptoms;" no marked want of control over the bladder and bowels; comparative voicelessness; the mouth clogged with viscid frothy saliva; a bitten tongue or cheek; dilatation of pupils; absence of priapism; presence of a wound; death by apnœa; early if not immediate rigor mortis.

Permanent muscular rigidity, causing, first, trismus, then opisthotonos, and implicating, when at its height, almost all the voluntary muscles except those of the hands, the eyeball, and the tongue.—Muscular rigidity, continuing without any marked relaxation from the time of its first appearance, is the most characteristic symptom of tetanus. It would seem to be the rule for this state of stiffness to begin in the muscles of the jaws, causing trismus, and to extend from thence as a centre, first to the muscles of the face and neck, then to those of the back, causing opisthotonos, then to those of the lower extremities, and lastly, to those of the upper extremities, the progress in both extremities being from above downwards; but there are exceptions to this rule, for a few cases are on record in which the muscles of the neck have been affected before those of the jaws, and others, also only few in number, where the muscles near a wound, as of a stump after amputation, have been the first to become rigid. Even in the most extreme cases, the hands and the tongue are found to remain limber, and it is but very rarely, except perhaps in children with "head symptoms" in addition to the ordinary phenomena of tetanus, that a squint or a fixed stare shows that the deep muscles of the orbit are affected. Fits of spasm, of which more will have to be said presently, may seize upon the tongue, as they do very frequently upon the muscles of the throat in attempts to swallow, but there is no proof that either the tongue or the muscles of the throat are ever in a state of permanent contraction. Neither is there any certain proof that the heart or other involuntary muscles are in any degree permanently contracted. The affected muscles are very hard, curiously so, feeling very much as they do in rigor mortis, and they are not unfrequently somewhat tender when pressed or squeezed. In the great majority of cases, without question, the first effect of tetanic rigidity is to close the jaws and cause trismus, and the next to bend the body backwards and produce opisthotonos. Opisthotonos, indeed, is almost as characteristic and constant a phenomenon as trismus. Now and then, it is true, instead of the body being bent backwards it may be bent forwards (emprosthotonos), or sideways (pleurosthotonos), but these cases are quite exceptional, and *opisthotonos* may in reality be looked upon as the position which the body always takes or tends to take in tetanus.

Pain at the pit of the stomach piercing through to the back.—This is reckoned by the late Dr. Chambers as the pathognomonic symptom of tetanus, and in fact it is scarcely ever absent, not even at the very beginning. This pain is especially severe in the fits of spasm, and then it is often agonizing, but it is present also, if not in a severe, at least in a mitigated form, in the intervals between these fits, scarcely ever ceasing altogether, even for a moment, when once it has made its appearance. It depends, there is little reason to doubt, upon the diaphragm being implicated in the tetanic condition. Once it was looked upon as a certain death-warrant, but this opinion, as Mr. Curling has shown, is untenable.

Difficulty of swallowing from the occurrence of spasm.—This spasm, which is provoked by the attempt to swallow, may be in the pharynx or gullet, or in the cardiac aperture of the diaphragm, one or all, making swallowing impossible, and often leading to the violent ejection of fluids through the nose or from the mouth. The distress consequent upon it may sometimes cause a horror of liquids not unlike that which exists in hydrophobia, and it always constitutes a grave difficulty, for it not only incapacitates the patient from feeding in the usual way, but it prevents him from being fed by means of the stomach-pump.

Fits of painful spasm in the permanently contracted muscles.—These fits become more frequent as well as more violent and painful as the disease progresses, recurring when at the worst every ten or fifteen minutes, and lasting from one to two and a half minutes. So violent has been the muscular contraction in some of these fits, that the teeth and thigh-bones have been broken, and great muscles like psoas and recti femorales torn across. These fits of spasm are almost invariably very painful, the being that of cramp, but now and then the pain has been absent: thus, Sir Gilbert Blane mentions, on the authority of a surgeon in the navy, a case of severe tetanus, fatal in four days, in which the fits of spasm only gave rise to a sort of pleasurable tingling; and Mr. Curling instances an analogous case. Most generally the pain in the fit of spasm is felt chiefly at the pit of the stomach, and very often the pain in this region may be so agonizing and stifling as to make the patient insensible to pain elsewhere. Sometimes the pain in the neighborhood of a wound, as in the stump after amputation, is that which is most complained of.

Risus sardonicus and an aged expression of countenance.—The sneering expression, caused by the angles of the mouth being drawn backwards and upwards, and known as the risus sardonicus, in association with spread nostrils, staring and prominent eyes, knitting of the brows, and bristling of the hair, is so often present as to be properly reckoned as pathognomonic of tetanus. In the fits of spasm the lips are often drawn apart so as to expose the set teeth, but sometimes they

are kept tightly pressed together by the spasmodic action of the orbicularis oris. The aged expression which was present in the case I have given, is exceptional, but it has been met with in other cases. Thus, Mr. Curling refers to a case of idiopathic tetanus, related by Dr. W. Farr, in which the patient, who was only twenty-six years of age, looked at least sixty; and he says further that he himself has "observed the same circumstance in an equally remarkable degree."

Dyspnœa with fits of comparative apnœa.—When tetanus is fully developed, an apprehension of suffocation is often present even in the intervals between the fits of spasm, and in these fits the suffused eyes, the livid countenance, and the agonizing struggle for breath show plainly enough that this is no sense a groundless fear. How this difficulty is brought about is not easy to say, and probably the way is not always the same. Sometimes spasmodic closure of the glottis would seem to be a prominent cause; sometimes the thorax is, as it were, held in a vice by the spasm of all its muscles generally; most commonly, perhaps, these two causes act together. From my own small experience I should be disposed to attach more importance to the last cause than to the first, and I question whether much relief would be obtained in any case by carrying out Marshall Hall's suggestion of opening the windpipe in cases of tetanus.

Increased reflex excitability.—In P. M., as the disease advanced, the fits of spasm were brought on by the most trivial causes—a draught of air, a sudden noise, an attempt to swallow, an attempt to administer an injection, the arrangement of the bed-clothes, the lightest touch even—and hence it may be inferred that increased reflex excitability was an element in this case. Nor is this case at all exceptional in this respect. As the disease advances, in fact, the controlling influence of the nervous system is removed, and this is all, for what are counted as signs of increased reflex excitability are in reality no more than signs of nervous exhaustion, such as manifest themselves whenever the vital powers are sufficiently lowered by loss of blood, or in any other way.

Greatly increased temperature, without true fever.—In the fits of spasm, and in a lesser degree in the intervals between the fits, the skin is very hot and damp, this heat rising in some cases as high as 110·75° Fahr., the sweat having now and then a peculiar pungent smell. Usually the skin is literally drenched in perspiration and covered with sudamina. Usually the pulse is quick and weak; and if in the fits of spasm it acquires more force, the state of semi-suffocation then present shows very plainly that this change in its character is, as I have shown elsewhere, due, not to the injection of more red blood into the artery, but to the greater resistance which imperfectly aërated blood has to encounter in getting out of the artery.

The increased heat of skin in tetanus at first sight appears to show that fever is a part of tetanus, but further inquiry points to a very different conclusion. As death approaches, the temperature, instead of falling, as it might be expected to do, may actually rise higher, and, what is stranger still, the rise may not be at its maximum until the patient has been dead for some time. Dr. Wunderlich[1] gives three cases which establish this fact—which he was the first to observe—beyond all contradiction.

The first of these cases is one of idiopathic or rheumatic tetanus, the patient being a butcher, aged 29. The disorder, which presented nothing remarkable in its symptoms, run its course in five days. Shortly preceding death there was some delirium, with marked abatement in the spasms, death happening in the exhaustion following a bout of spasm of no special severity. Putrefaction was unusually rapid. The brain was healthy, the cord was injected, and its texture (neuroglia) considerably broken down. The temperature of the ward at the time of death was 77° Fahr. What is of interest in the state of the breathing, the pulse, and the temperature, is as follows:—

[1] Archiv der Heilkunde, Bd. ii., iii., and v. (1861, 1862, and 1863).

Date.	Respirations.	Pulse.	Temperature, Degrees Fahr.
24th July, 1861	24	96	102
25th " "	22	82	102
26th " " 9 A.M.	20	96	104·45
" " " 6 P.M.	32	112	103·55
" " " 9.20 P.M.	36	180	110·1
" " " 9.35 P.M., *death* . .	...	...	112·55
After death 2′ . .	...	...	112·77
" 5′ . .	...	...	113
" 20′ . .	...	...	113·22
" 35′ . .	...	...	113·55
" 55′ . .	...	...	113·67
" 60′ . .	...	...	113·55
" 70′ . .	...	...	113·22
" 90′ . .	...	...	113
" 100′ . .	...	...	111·8
" 6 hours . .	...	...	106·25
" 9 " . .	...	...	104
" 12 " . .	...	...	102
" 13½ " . .	...	...	101

The second case is one of traumatic tetanus in a man aged 20, fatal tenth day. Up to twenty-four hours before death the tetanic symptoms were fully marked, and the mind quite clear; at this time, and especially in the six hours immediately preceding death, unrest, talkativeness, jactitations, and slight delirium were more prominent symptoms than the spasms. The appearances after death were like those found in the first case. In the last three days the mercury went up slowly and steadily from 100° to 105°·8, at which point it stood three hours before death; at death and afterwards the notes made of the temperature are these :—

At death	107°·6 Fahr.
10′ after death .	107·8
15′ " . .	108·
20′ " . .	107·8
38′ " . .	106·45
58′ " . .	105·8
1 hour 5′ " . .	105·3
" 20′ " . .	104·45
" 35′ " . .	103·55
2 hours " . .	101·75
4 " " . .	99·3

The third case is that of a man, aged 57, a bookbinder by trade, with idiopathic or rheumatic tetanus. Tetanic symptoms set in in the usual way on the 20th June, 1863, and were fully developed two days afterwards, when also symptoms of pneumonia were detected. Death happened toward the end of the day following, as much or more from the pneumonia as from the tetanus; and after death the only very marked appearances were those of pneumonia. In this case the temperature, which was never higher than 104°·55, was—

3½ hours before death	102°·85 Fahr.
At death	not stated
10′ after death . .	103·32
21′ " . .	103·55

Other cases are also on record which show that this strange rise in temperature up to death and after it, is not peculiar to tetanus. Dr. Wunderlich gives three such cases :—(1) A case apparently of lead-poisoning in a plumber, ending fatally in 40 hours: the symptoms being sudden insensibility, and, later on, tetanic and epileptiform convulsions. In this case the temperature at death was 107°·7, and there was some slight increase afterwards; (2) a case of cerebro-spinal meningitis, with unrest, delirium, and retraction of head for four days, and then sopor, in which the temperature was 107°·26 at death and 107°·37 after death; and (3) a case of rheumatic fever, with cerebral meningitis, shortly ending in coma on the sixth day, in which the temperature ranged from 109°·62 to 110°·75 in the five hours before death, and stood at 111°·87 thirty minutes after death. Dr. Erb[1] also gives three cases with fuller detail, namely, these :—

A young man, aged 22, with tubercular inflammation of the base of the brain, who died without convulsions, after having been in a state of profuse perspiration and unconsciousness for twenty-four hours, with rapid respiration (60 to 44), and a pulse quite uncountable, towards the end. In this case the temperature in the twenty-four hours before death, at death, and afterwards, was as follows :—

24 hours before death . . .	from 102°·65 to 104·9 Fahr.
At death	104·9
13′ after death	105.12
25′ "	104·67
55′ "	104

A woman, aged 22, six months gone in pregnancy, who died with symptoms of

[1] Deutsches Archiv für Klin. Medicin. vol. i. 1866.

coma, without convulsions, the coma having set in suddenly an hour and a half before death, and in whom signs of purulent meningitis were detected after death. During the comatose state the breathing was very labored, and the pulse full and frequent. The temperature, of which the notes are as follows, was only taken after death :—

At death	Temp. not stated.
6′ after death	103·45 Fahr.
10′ "	104
15′ "	104·67
20′ "	104·9
25′ "	104·9
35′ "	105·12
45′ "	105·12
1 hour 40′ after death .	104
2 hours 40′ " . .	101·22

A woman, aged 22, who after having suffered from diabetes mellitus in its ordinary form for three and a half years, passed into a state of sopor, after having had headache and some delirium for twenty-four hours. Death happened in about forty-eight hours from the commencement of the head symptoms. During the twenty-four hours of sopor preceding death, the degree of heat ranged from 102°·65 to 106°; at death and afterwards the notes made of the temperature are these :—

	°
At death	106 Fahr.
5′ after death . . .	106·25
15′ "	106
25′ "	105

The body has been found to become very hot before death, and to remain very hot after death, in cholera, in yellow fever, and in several other cases, of which instances are given by Dr. Erb and by several other writers in Germany, and by Drs. Ringer,[1] Weber,[2] Murchison,[3] Sanderson,[4] and others in this country; the cause of death in the majority of these cases being some sudden affection of the brain, coma in others; but there are but few cases in which the heat of the body has been found to rise after death. Indeed, I know of no such cases besides these I have quoted, except one, which came under my notice a short time ago—a case of a man, aged 60, who died from sunstroke in twenty-six hours, the symptoms being sudden coma, with great oppression of the pulse and breathing, without convulsion. In this case the temperature was—

	°
12 hours before death . . .	103·25 Fahr.
3 " " . . .	104
At death . . .	not ascertained.
7 hours after death . . .	105·5

[1] Med. Times and Gazette, vol. ii. 1867.
[2] Clinical Soc. Trans. vol. i. 1868.
[3] Ibid.
[4] Ibid.

If, then, the temperature rises in this manner under these circumstances, it is more than difficult to connect the increased heat of tetanus with increased activity of the circulation or with anything like fever in the ordinary form of the word. The temperature rises as the time of death approaches, when the state of the circulation must every moment be becoming more and more the reverse of increased activity: the temperature continues to rise even after actual death, when the blood has come to a standstill. These are the facts; and, these being the facts, it may be that the increased heat in tetanus may be connected, not with increased activity of the circulation, not with true fever, but rather with the contrary state of things. Nor is it more easy to connect the increased heat of tetanus with the spasms. A part of the increase may be accounted for in this manner, but only a small part. Indeed, the simple fact that in one of the cases which has been instanced a marked abatement in the severity of the spasms was accompanied by an actual rise in the column of mercury, and that the column continued to rise after death, when all spasm is at an end, is in itself a sufficient proof that it is not in muscular action that the explanation of the increased temperature of tetanus is to be found. Moreover, the fact that the temperature rises in the same way before and after death, in cases where neither convulsion nor spasm was among the symptoms during life, must lead to the same conclusion. How to explain the increased heat of tetanus is another matter. Increased heat, as was shown in the primary remarks, is an effect of injuries by which the cord or medulla oblongata is torn or cut across. Increased heat, as is seen in some of the cases which have just been alluded to, is an accompaniment of certain diseases which annihilate, more or less completely, cerebral action. It seems as if one condition of this change in temperature was the paralyzing of a regulating cerebral influence; and beyond this it is difficult to see further, except it be that this paralysis, reaching to the vasomotor nerves, allows the minute vessels to dilate and receive more blood, and that the increased quantity of blood, even though this blood may be stagnant, may lead to increased molecular changes, of which increased heat is an effect. What is necessary, however, is not to find the cause of the increased heat in tetanus, but simply to point out the fact that increased heat in this case does not imply increased activity of circulation—that true fever, in the ordinary sense of the word, is not a part of tetanus. And this, as it seems to me, is a legitimate inference from the evidence which has been cited, and the comments which have been made.

Absence of sleep.—In the acute cases,

sleep, as a rule, is banished altogether, and even in the subacute cases this blessing is only realized in unrefreshing broken snatches. Want of sleep, indeed, is one of the not least distressing features of this disease. "The muscles," says Mr. Curling, "are observed to be relaxed during sleep, a striking example of which occurred to Mr. Mayo in a boy who recovered from the disease. On visiting his patient before the symptoms were subdued, Mr. Mayo found him asleep, and remarked that he lay perfectly relaxed. The abdominal muscles were soft and yielding, and had not the least tension. The boy was awakened, and at the instant the full tension of the muscles returned. Not being further disturbed, he fell asleep in a few minutes, when the muscles again became relaxed, and again, on his being awakened, resumed the state of spasm. I have, on several occasions, witnessed the same phenomena." Except the biting of the tongue, on waking from a brief nap, be a reason for believing that the muscles of the jaws had been relaxed during sleep, so as to allow the tongue to get between the teeth, there was no proof that the muscles were relaxed during sleep in the case I have given; but in other cases I have had proof sufficient of this relaxation.

Absence of numbness and tingling.—Of this there can be no doubt—that numbness and tingling form no part of the history of tetanus.

Absence of "head symptoms."—The mind is clear from the beginning to the end of the disease almost invariably, and not unfrequently it is a matter for wonder how well the patient bears up under his atrocious sufferings—a marked difference this between tetanus and hydrophobia. And in the few instances in which delirium or coma has made its appearance a short time before death, it is not improbable, as more than one writer has observed, that this derangement is often more the result of the remedies employed than of the disease.

No marked want of control over the bladder or bowel.—In tetanus there is, as a rule, none of the difficulty with the bladder which is almost invariably met with in acute spinal meningitis. The bladder may act seldom, but it is not incapable of acting. Constipation is a common but not a constant symptom, and when it is present it may be a question whether, like the "head symptoms," it is not as much due to the medicines used as to the disease. Now and then, however, there may be great difficulty in voiding the contents of the bladder and bowels, and in some of these cases the resistance to the introduction of a catheter or enema-pipe has shown that a part of this difficulty is owing to spasm of the compressor urethræ or sphincter ani.

Comparative voicelessness.—This phenomenon is readily accounted for as a result of the spasmodic interference with the action of the chest and of the tight shutting of the jaws. Indeed, it could not well be otherwise in the fully developed disease.

The mouth clogged with viscid frothy saliva.—This is a common if not a constant symptom, though not so marked in degree as in hydrophobia, and there is no difficulty in accounting for it in either case, for the inability to drink and swallow will explain at one and the same time why the saliva is viscid and why it accumulates in the mouth.

A bitten tongue or cheek.—This accident is of rare occurrence, and its rarity may be taken as an incidental proof of sleeplessness as a symptom of tetanus, for it is to be supposed that the opening of the jaws, from the relaxation of their muscles during sleep, would allow the tongue or cheek to get between the teeth—to get into that position in which the spasm which attends the moment of waking would be sure to crush it.

Dilatation of pupil.—This condition was always present in the case which serves as my text, especially in the fits of spasm, and this has been the rule in several cases of tetanus in which I have examined the pupil. Mr. Curling, on the contrary, found the pupil contracted in the majority of his cases.

Absence of priapism.—Mr. Morgan states that priapism occurs occasionally; but this observation is not confirmed by other writers on the subject. I have never seen it, and I am very much disposed to think that the case or cases in which Mr. Morgan saw it were cases, not of tetanus, but of acute spinal meningitis, in which disorder priapism is an occasional symptom.

Presence of a wound.—The great majority of cases of acute tetanus appear to be in some way depending upon a wound or injury of one kind or another in one place or another. I shall have occasion to refer to this relationship elsewhere: and at present I would only notice, in passing, the presence of a wound which, to all appearance, presented no indications of an inflammatory or otherwise unhealthy character.

Death by apnœa.—Apnœa is one way, and perhaps the common way, in which death is brought about in tetanus. Not unfrequently, however, the patient sinks from asthenia, having been to a great degree free from fits of suffocative spasm for some time before death. Spasm of the heart has also been mentioned as a method of dying in tetanus, and the heart has not unfrequently been found to be curiously hard and contracted after death; but an examination of the facts tends very much to discountenance this idea, and to show

that death is either by apnœa or asthenia, singly or together.

The immediate occurrence of rigor mortis. —Sommer and others have noticed that rigor mortis may occur without any appreciable interval of muscular relaxation after death from convulsions, and Dr. Brown-Séquard has confirmed this observation and given a definiteness to it which it had not before. He has indeed done more than this, for he has not only confirmed the fact that rigor mortis may occur without any appreciable interval of muscular relaxation, but he has established the law that rigor mortis is long in coming on and long in passing off where death was not preceded by any long-continued violent action of the muscles, and that it is quick in coming on and quick in passing off in direct proportion to the amount of long-continued violent action which preceded death. In many animals killed by strychnine, for example, in which death was brought about, not by one violent spasm, but by many, he has found rigor mortis set up before the heart had ceased to beat. Nay, he even refers to the case of a man under his own observation in which rigor mortis occurred before the heart had ceased to beat. I have never witnessed this phenomenon either in animals or in man; but I have more than once failed to find any line of separation between tetanic stiffness and cadaveric rigidity in animals killed by strychnine, or by the shocks of a Ruhmkorff coil: and I am therefore quite prepared to understand that in P. M——'s case, where there were many convulsions before death, rigor mortis may have occurred without any appreciable interval of muscular relaxation, and in this way fixed in the corpse the age expression of the countenance, and the opisthotonic attitude.

Two distinct varieties of tetanus are usually recognized, and properly so—the *traumatic*, in which a hurt of some kind or other is believed to be the primary cause; and the *idiopathic*, in which the only obvious cause would seem to be exposure to cold and damp. In each variety the symptoms are much the same, any difference of moment being only one of degree. In the acute form, the spasms come on suddenly, occur frequently, and grow in violence with each recurrence: in the less acute forms the spasms are more slowly developed in the first instance, the paroxysms are comparatively far between, and they do not recur with increasing rapidity and violence. The traumatic, as a rule, is more acute than the idiopathic variety. Trismus nascentium is considered by many as a distinct variety of tetanus, but this appears to be a distinction without a real difference. It is tetanus in newly-born infants,—traumatic, because the wound of the navel seems to have a good deal to do with its production, and at the same time idiopathic, for it is certain that cold and damp, and foul air and other general causes also figure conspicuously as sources. It is, indeed, to this form of tetanus that a remark of Sir Thomas Watson applies especially, which is applicable to all forms, namely this, that "although tetanus may be excited by a wound independently from exposure to cold, or by cold, without any bodily injury, there is good reason for thinking that, in many instances, one of these causes alone would fail to produce it, while both together call it forth."

2. Post-mortem Appearances. — There are no morbid changes in the nervous system peculiar to tetanus. "Serous effusion with increased vascularity," says Mr. Curling, "is generally observed in the membranes investing the medulla spinalis, and also a turgid state of the blood-vessels about the origin of the nerves," and the same changes may also be met with in the cranium, but in a less degree, and less frequently. It is also a fact of considerable moment in relation to this point, that Majendie, Ollivier, and Orfila failed to detect any perceptible lesion in the spinal cords of animals dying from the tetanus produced by strychnia. Out of seventy fatal cases collected by Mr. Curling, there were only two in which changes in the nervous system unequivocally the result of inflammatory action were discovered after death, and these two were cases where there had been a blow or wound to the back, where the symptoms had plainly to do with the inflammation of the cord or its membranes rather than with tetanus, and where the signs of inflammation found after death may, to say the least, be referred to the injury quite as easily as to the tetanus. Mr. Curling also points out, as a fact not to be overlooked, that the turgid state of the vessels of the pia mater, together with the effusion of serum which is met with in the spinal cord and brain after death from tetanus, is also met with in those persons who may have been poisoned by opium, hydrocyanic acid, and other powerful agents often employed in the treatment of tetanus, as well as after death from delirium tremens, hydrophobia, epilepsy, and other diseases; and as bearing upon these exceptional cases, in which unequivocal signs of inflammation in the cord or brain have been met with after death from tetanus, he says, "Whether inflammation be the result of injury or arises spontaneously, it is worthy of notice that the spasms, though continued and severe, do not occur in such violent paroxysms as in traumatic tetanus." Neither can the preternaturally injected state of

the minute vessels supplying the sympathetic ganglia, especially the cervical and semilunar, met with by Mr. Swan and others in some cases of tetanus, be looked upon as at all constant phenomena after death from tetanus.

Nor do recent microscopic investigations into the condition of the spinal cord in tetanus bring to light any clearer signs of inflammatory change. Mr. Lockhart Clarke[1] finds the vessels injected, and the substance of the cord in a state varying from simple softening to complete solution, the softened or dissolved portions forming irregular "areas of disintegration," filled with the *débris* of bloodvessels and nerves, or with a fluid finely granular or perfectly pellucid. These areas of disintegration were chiefly in the gray substance around the canal, but they were also in the white substance. They were, in fact, in no one part exclusively or particularly. Here and there were extravasations of blood, and "other exudations," but pus corpuscles are not mentioned. "In the walls of the bloodvessels," Mr. Clarke says, "there was no morbid deposit, nor any appreciable alteration of structure, except where they shared in the disintegration of the part to which they belonged; but the arteries were frequently dilated at short intervals, and in many places were seen to be surrounded, sometimes to a depth equal to double their diameter, by granular and other exudations, beyond and amongst which the nerve-tissue, to a greater or less extent, had suffered disintegration." "The appearances met with," says Mr. Clarke, are "exactly similar in kind to the lesions or disintegrations which I find in various cases of ordinary paralysis, in which there is little or no spasmodic movement." The cord is broken up, in fact, as at a certain time it is broken up by ordinary putrefaction, and, the dilated vessels and certain exudations of blood and serum excepted, this is all that is noticed. The case points to disintegration, not to inflammation; and what Mr. Clarke finds in six cases is substantially the same as that which Dr. Dickenson[2] finds in the one case examined by him. Indeed the only peculiarity in this latter case is, in the presence, in addition, of an excessive quantity of a translucent, structureless, and finely granular, carmine-absorbing material, evidently the sero-fibrinous plasma of the blood, which has escaped from the minute arteries into various parts of the substance of the cord where the nerve tissue has broken down, or which lies in pools here and there between the cord and its membranes. It is a state of œdema rather than anything else, certainly not a state of inflammation.

Traces of inflammation in the wound, especially in the injured nerves, may be met with after death from tetanus, and more frequently than in the spinal cord or other great nervous centres; but these again, instead of being constant, are not even common appearances. In the great majority of cases, indeed, the wound, if there be one, is perfectly healthy and healing. Neither are there any other post-mortem facts which can be looked upon as essential to tetanus, for those which remain to be mentioned, as ruptured muscles, broken or dislocated bones, engorged lungs, injection or contraction of the pharynx and palate, worms in the alimentary canal, and others, are plainly accidental and exceptional.

3. Causes.—The two great causes of tetanus are, as has been mentioned already, cold and damp, and bodily injury of some sort. Exposure to cold and damp tells most in this manner when acting upon a body previously relaxed by heat and perspiring, and this is all that can be said, except that this exposure is more likely to issue in tetanus in a foul atmosphere than in a fresh one. As regards the hurt which may give rise to tetanus, it is difficult to know what to say. In the Peninsular war, as Sir James McGregor states, tetanus supervened on every description and in every stage of the wounds, from the slightest to the most formidable, in the healthy and sloughing, the incised and lacerated, the most simple and the most complicated; and this statement expresses the opinion of all surgeons, military and others. Indeed, all that can be said is, that punctured wounds seem to be more likely to issue in tetanus than incised, and wounds in the extremities more than wounds in the head, breast, and neck. And certainly an inflammatory condition of the wound cannot be regarded as essential. In a great number of cases, in the majority perhaps, the primary wound was completely healed and almost forgotten when the symptoms of tetanus made their appearance; and Dr. Rush, who had extensive opportunities for observation in the military hospitals of the United States, and who was unquestionably a most competent observer, remarks that there was invariably an absence of inflammation in the wounds causing the disease. John Hunter also says: "The wounds producing tetanus are either considerable or slight. . . . . When I have seen it from the first, it was after the inflammatory stage, and when good suppuration was come on; in some cases when it had nearly healed, and the patient was con-

[1] Med.-Chir. Transactions, vol. xlviii. 1865.
[2] Ibid. vol. li. 1868.

sidered healthy. Some have had locked jaw after healing was completed. In such I have supposed the inflammation to be the predisponent cause, rendering the nervous system irritable as soon as it was removed. When tetanus comes on in horses, as after docking, it is after the wound has suppurated and begun to heal." There is, indeed, abundant evidence to show that an inflammatory condition of the wound is not necessary to the production of tetanus, and some evidence even which is calculated to lead to a contrary conclusion, by showing that where an inflammatory condition of the wound has been present, this condition has passed off before the tetanic symptoms made their appearance—the inflammation, to repeat the words of John Hunter just used, "rendering the nervous system irritable *as soon as it was removed*," not rendering it irritable as long as it was present. The interval between the hurt and the development of the tetanic symptoms varies considerably. In eighty-one of the cases collected by Mr. Curling, the symptoms made their appearance between the fourth and fourteenth days, both inclusive, and in nineteen on the tenth day. Four cases are also given in which the symptoms came on more speedily, one (somewhat doubtful) almost instantaneously, another in one hour, a third in two hours, and the fourth in eleven hours, and, at the other extreme, one in which they were deferred as late as the tenth week. In traumatic tetanus the sooner the symptoms show themselves the more acute and dangerous is the malady. In idiopathic tetanus the symptoms, as a rule, commence sooner than in traumatic tetanus, often in a few hours; but the idiopathic, notwithstanding, is generally of a more chronic kind than the traumatic, and far less dangerous.

Tetanus is not a malady peculiar to any country, or climate, or people, but it is more common in hot countries than in cold. It would appear, also, that negroes are more likely to be attacked than whites. Great atmospheric changes, especially from heat to cold and damp, as to a cold and dewy night after a sultry day, are evidently most favorable to the development of tetanus, and so in a less degree are foul air, despondency, terror, physical exhaustion. It must be confessed, however, that cases of idiopathic tetanus, as compared with those which are traumatic, or partly idiopathic and partly traumatic, are, to say the least, extremely rare in this country.

4. Diagnosis. — The differences between tetanus and acute spinal meningitis are sufficiently marked to prevent any confusion as to diagnosis if only a moderate degree of attention be paid to the subject. In tetanus the jaw is firmly set from the first, and, in addition to the fits of spasm, there is permanent muscular rigidity between the fits: in spinal meningitis, if the jaw be set at all, it is rather at the close of the disease, and then only in an inconsiderable degree, and spasms or muscular rigidity are neither constant nor conspicuous phenomena. In spinal meningitis, indeed, it is plain that the muscular rigidity and seeming spasms are in great measure voluntary or semi-voluntary acts to prevent the pain in the back and limbs which is produced by movement, and that the muscles are relaxed almost as long as the patient can keep perfectly still. In a word, the true involuntary fits of spasm and the permanent muscular rigidity which are constant and characteristic phenomena in tetanus, are not present in acute spinal meningitis.

Nor can hydrophobia be very well confounded with tetanus. In tetanus the features are drawn into the risus sardonicus, the eyes are natural, and the whole countenance is expressive of pain and suffering,—nothing more: in hydrophobia there is an impress of excitement and distress and horror and unrest upon the features which has no counterpart in the tetanic countenance. In tetanus the body is for the most part rigidly fixed in one position by tonic spasm; in hydrophobia the spasmodic movements are clonic, and the body is in a state of perpetual unrest until the stage of final exhaustion. In hydrophobia, noisy attempts are continually made to spit and hawk away the viscid phlegm which clogs the mouth and throat—the noises being sometimes not altogether unlike the bark of a dog—and any effort to relieve the tormenting thirst, or even the bare thought of such an effort, brings on the fit of fear and convulsive agitation which has given rise to the name hydrophobia: in tetanus there are no symptoms which can be considered as strictly comparable to these. In tetanus, finally, the mind is clear to the last, whereas in hydrophobia there is almost from the first a peculiar and often very wild delirium.

The tetanic symptoms produced by strychnia and some other poisons may be more easily confounded with traumatic tetanus, but even here it is possible, with care, to make a correct diagnosis. It is possible, as Dr. Christison pointed out, for strychnia to be given in repeated doses so regulated as to produce a train of symptoms scarcely, if at all, distinguishable from traumatic tetanus; but not so if, as is usually the case, an amount sufficient to produce death be given in one dose. In this latter case, indeed, the differences of the symptoms are sufficiently marked. In the toxic tetanus the symp-

toms run a rapidly fatal course, death happening in a quarter of an hour, half an hour, and usually within the hour: in traumatic tetanus, with very few exceptions, life is prolonged for two or three days at least. In the toxic tetanus the arms are stretched stiffly out, the hands clenched, and the legs separated widely from each other and rigidly extended: in traumatic tetanus the hands are usually free from spasm, and the arms nearly so, and even the legs are scarcely ever affected to the degree which is seen in toxic tetanus. In the tetanus caused by strychnia, Mr. Poland says, "The patient can open his mouth to swallow; there is no locked jaw:" in traumatic tetanus, locked jaw is the first and most constant manifestation of the spasm.

The jaw may be locked for a long time, and various muscles in other parts may be affected with continuous spasm, in cases in which hysteria is supposed to figure largely as a cause—cases in which there is the condition called spinal irritation: but these cases, as will appear in due time, even when most like, are in reality so unlike tetanus as scarcely to deserve even this passing mention.

5. Prognosis.—In the cases "in which the access is slow, the spasms by no means violent, the paroxysms slight and recurring at long intervals, and where the patient can obtain sleep, whether traumatic or not, we may generally anticipate a favorable result;" and, again, "the longer the interval before the appearance of the symptoms, the more chronic the disease, and the greater the probability of recovery." So speaks Mr. Curling of the chronic cases of tetanus in contradistinction to the acute; and in illustration of the probability of recovery, he adds: "In thirteen cases, symptoms of tetanus occurred about three weeks after the wound, and four only were fatal; and in seven cases in which they did not make their appearance till after a month, only two ended fatally." In the cases, on the other hand, in which the spasms supervene rapidly upon the injury, and recur with increasing violence at decreasing intervals, and in which sleep is banished, a vast majority die—die, as Hippocrates noticed ages ago, within four days. Death may happen in a fit of suffocation in which sometimes there is obviously spasm of the glottis, but more frequently it would seem to be brought about by asthenia after a fit of spasm. The time occupied in recovery varies greatly,—one, two, three, four, five, six, seven, eight weeks, or even longer. A certain degree of weakness and stiffness may also remain in the muscles long after recovery. In one case rigidity of the muscles of the jaw remained for six months; in another it returned whenever the patient caught cold up to nine months; and in a third, at the end of three years, it is stated that the "features retained the indelible impression of the disease." These cases are given by Mr. Curling.

6. Treatment.—After passing in review the principal remedies that have been tried in tetanus—opium, blood-letting, the cold bath and cold affusion, ice to the spine, the warm bath, bark, wine and spirits, mercury, purgatives, foxglove, tobacco, musk, prussic acid, carbonate of iron, oil of turpentine, strychnia, woorali, ether and chloroform inhalations, amputation, division of nerves, tourniquets—Sir Thomas Watson says: "In all cases, there being no special indication to the contrary, I should be more disposed to administer wine in large quantities, and nutriment, than any particular drug;" and this statement, I take it, expresses a very general feeling in this country. For my own part, I should certainly be more disposed to trust to alcohol than to any drug; but, in saying this, I do not say that I should place no confidence in drugs. I should certainly place no confidence in any sedative or narcotic given by the stomach in sedative or narcotic doses; but, on empirical as well as on theoretical grounds, I should say that opium can scarcely be dispensed with, and that chloroform or ether inhalations will be of infinite service in relieving pain and spasm, and that too without compromising the chances of recovery, if care be taken to pour in wine and to supply nourishment at the same time so as to prevent the patient from waking up almost immediately after the inhalation.

If the *rationale* of spasm be that which is hinted at in the preliminary remarks, the great indication of treatment must be, not to depress the circulation, but to rouse it into greater activity; and one reason why the treatment of tetanus has been so eminently unsatisfactory may be that this indication has not been fully realized and carried out. In tetanus much wine may be given without producing anything like intoxication, or without relaxing the spasms in any degree. The system in this disease is altogether insensible to the action of wine in ordinary doses. As to this there can be no doubt. Whether a different result would have been arrived at if alcohol had been given more boldly, ardent spirits in place of wine, ardent spirits undiluted rather than diluted, is yet an open question, but I am disposed to think that the spasms might have been conquered without compromising the safety of the patient if this had been done. There are now not a few cases on record which show that the bite of a rattlesnake or cobra or other deadly

serpent may be prevented from killing by at once giving ardent spirits in sufficient quantity, and I am disposed to think that these facts have an important bearing upon the treatment of tetanus. There are, undoubtedly, great differences between the condition in tetanus and the condition in these poisoned bites, but there are also certain resemblances which must not be lost sight of. There is the same insensibility to the action of alcohol in ordinary doses; there is an exhaustion to be counteracted, which is more rapidly fatal in the poisoned bite than in tetanus, but which in acute tetanus is sufficiently rapid to create the gravest fears, and to justify the most heroic measures; there may even be a poison at work in both cases as well as a wound, a poison introduced into the wound in one case, a poison generated in the wound in the other case. There are resemblances between the two cases, indeed, which, though not very close, may be close enough to justify the hope that a practice which has been found to answer in the bite of a poisonous serpent may also be found to answer in acute tetanus.

In speaking thus, it is not intended to imply that ardent spirits are the only way of fulfilling what has been said to be the primary indication of treatment in tetanus. Eau de luce has been found to be of great service in the bites of serpents, and it might be of service in tetanus. Ether, also, might be of use, or turpentine, or camphor, or ammonia. But to my mind these and other medicines of a like nature are more likely to disorder the stomach and system generally, and in other respects are less manageable and less certain in their action, than ardent spirit.

As regards local measures it is less difficult to arrive at a conclusion. In many cases, no doubt, there is an eccentric irritation, starting from the wound or some other point, and much good would be done if this could be removed. It is probable, also, that this end might be gained in more ways than one, and that one very direct way is by the subcutaneous injection of various substances—morphia, atropine, woorali, conia (which seems to be strictly analogous in its action to woorali), Calabar bean, &c. The results of these injections in causing the relaxation of spasm in conection with the minor forms of spinal irritation are very encouraging. One thing, however, ought to be borne in mind, and that is, that these injections should be used so as not to produce a general depressing or paralyzing effect upon the nervous system. All that ought to be aimed at is to obviate local irritation merely; and, to my mind, to go beyond this point is both wrong in principle and dangerous in practice.

[Dr. Joseph Hartshorne called attention many years ago to the value of vigorous counter-irritation along the spine, in tetanus. He employed a solution of cantharides in oil of turpentine for this purpose.—H.]

For the rest, it is, of course, desirable that the patient should be carefully guarded from cold, and from anything which would excite or disturb him, as too much light or noise, or too meddlesome nursing. In a word, quiet and warmth are not only desirable; they are indispensable.

V. Locomotor Ataxy.

Until very recently the disease which forms the subject of the present article was confounded with paraplegic diseases. The difficulty in locomotion, which is the most characteristic symptom, was supposed to be owing to simple paralytic weakness of the legs. It was not perceived that the legs, in the earlier stages of the disease at least, had lost little, if any, of their power to act separately—so little, indeed, that it might require all the force of a strong man to bend or straighten them against the will of the patient—and that what they had lost was that power of co-ordination by which the two limbs are enabled to act together, as they have to do in standing and moving about. The credit of having first drawn this distinction, and at the same time shown that this want of co-ordinating power is so associated with a definite group of other symptoms as to deserve to be regarded as a distinct disease, is due to Dr. Duchenne (of Boulogne). Before this time, no doubt, the characteristics of such a disease had been more or less clearly realized. They had been described, in fact, under the old name of *tabes dorsalis*, especially in the sketch of this disease given by Dr. Romberg. They had been detected by the late Dr. Todd, and not only so, but associated with that particular lesion with which they are now known to be connected, namely, with chronic disease of the posterior columns of the spinal cord. "Two kinds of paralysis of motion," wrote Dr. Todd, "may be noticed in the lower extremities,—the one consisting simply in the impairment or loss of voluntary motion; the other distinguished by a diminution or total loss of the power of co-ordinating movements. In the latter form, while considerable voluntary power remains, the patient finds great difficulty in walking, and his gait is so tottering and uncertain, that his centre of gravity is easily displaced. The cases are generally of the most chronic kind, and many of them go on from day to day without any increase of the disease, or improvement

of their condition. In two examples of this variety of paralysis I ventured to predict disease of the posterior columns, the diagnosis being founded upon the views of the functions of the columns which I advocate; and this was found to exist on a post-mortem inspection; and in looking through the accounts of recorded cases, in which the posterior columns were the seat of lesion, all seem to have commenced by evincing more or less disturbance of the locomotive powers." (Cyclopædia of Anatomy and Phys., vol. iii. p. 721, S.)

Dr. Todd published these remarks in 1845; Dr. Duchenne's first memoir appeared in 1857. Dr. Todd must, therefore, have the credit of having anticipated Dr. Duchenne; but still the lion's share of honor must be assigned to the latter, for the plain fact is that Dr. Duchenne has developed in a series of formal memoirs what Dr. Todd has only indicated in these few sentences. In a word, it must be allowed that Dr. Duchenne deserves almost the entire credit of being the first to detect the exact features of the disease now known as progressive locomotor ataxy, and to call the attention of others to the subject.

The name of progressive locomotor ataxy (*ataxie locomotrice progressive*), from α, privative, and τάξις (order), is that which was chosen by Dr. Duchenne. It is not a very fortunate one, but it has been adopted, and must be retained, until a better one is found. It is certainly to be preferred to *tabes dorsalis*, for this name is commonly supposed to imply past incontinence on the part of the patient. How far it is right to perpetuate the cheerless affix *progressive* is, however, very questionable. At present, no doubt, the prognosis is full of gloom. From bad to worse is the common course of things, but, at the same time, there are cases—and their number is increasing every day—in which the symptoms have been long stationary, and others in which there has been unequivocal amendment. But even if the element of hope were wanting, it is surely desirable not to bring this unhappy fact into undue prominence. It is surely not necessary to continue to use an epithet of which the effect must be to frighten the patient and discourage the practitioner, and this too without compensating advantages of any kind. As it seems to me, indeed, everything is gained by the name locomotor ataxy which is gained by the name progressive locomotor ataxy; and nothing is lost but what can well be spared; and therefore in what I have to say I shall drop the term "progressive," and speak simply of "locomotor ataxy."

1. Symptoms.—As a text for what I have to say upon the symptoms of locomotor ataxy, I take from my note-book a case which was some time ago (April, 1865) under my care in the National Hospital for the Paralyzed and Epileptic.

Case.—A sailor, thirty-four years of age, by name J. C——, well proportioned, unusually well developed as to muscle everywhere, very lean, and much bronzed by long exposure to sun and sea.

(*a*) This man is capable of walking without a stick, but his gait is peculiar—staggering, precipitate, the legs starting about vaguely and spasmodically, and the heels coming down heavily at each step. With his eyes shut, or in the dark, he reels over at once and falls to the ground, if left to himself. Sitting or lying down, he can lift either leg steadily into any position, and fix it there so firmly that it is out of my power to bend or straighten it against his will. In order to do this, however, he must see what he has to do, for if his eyes are shut, his limb at once becomes uncertain and unsteady in its movements, and comparatively powerless. His right leg is a little weaker than the left, but not in any well-marked degree. He finds it very difficult to come downstairs, or to turn round, or to quicken his pace much, and he is speedily fatigued by the acts of standing or walking. On being told to shut his eyes, and touch his nose with the forefinger of each hand in turn, he does so with tolerable accuracy, especially with the forefinger of the left hand. On being told to stretch out his arms, and keep them out, he does so, but only so long as he is allowed to see what he is doing; for, on holding a book up before his eyes, the arms, shoulders, neck, and head—the upper part of his body generally—at once became afflicted with convulsive agitation. When the book was taken away, these movements speedily came to an end, but not before they had issued in a fit of crying and sobbing which was not a little distressing to witness. This fit took the patient quite by surprise, and it could not be accounted for by the examination having been conducted roughly, or carried on for an undue length of time; indeed, the holding of the book before the eyes, which was the immediate cause of the fit, did not occupy more than a minute at the most.

The muscles of the lower limbs stand out firm and hard when made to contract by the will, and the contraction seems to be not at all wanting in force. Indeed, as has been already stated, it is out of my power to bend or extend the limb against the will of the patient. There is no tremulousness in the legs or elsewhere, and there are no marked reflex movements when the soles of the feet are tickled.

What are complained of chiefly are severe pangs of pain, stabbing, boring, in flashes like lightning, flitting from one spot to another in a very erratic manner, recur-

ring in paroxysms varying in length from a few minutes to twelve, twenty-four, or forty-eight hours, and generally remaining at the same spot during the same paroxysm. These pangs are most frequently felt in the two feet, especially along the outer side of the metatarsal bone of the little toe; and they also are not unfrequently met with at the back of the thigh, in the nates, and in the upper arm about the lower part of the belly of the biceps. They are scarcely ever absent, especially at night; at night, too, there is often a sensation of great coldness, with some degree of constriction at the seat of pain.

Tactile sensibility, measured by the compasses, is found to be much impaired in both feet, especially in the soles, in the calves of both legs, and to some degree also at the back of the thighs, in the nates, and in the palms of the hands. The ground is felt very obscurely, but, so far as it is felt, the sensations are accurate—that is to say, it does not seem as if there were elastic cushions, pebbles, or other imaginary bodies upon the floor, or as if the feet had nothing under them but free air, as is sometimes the case. Very rough pinching is scarcely at all felt in the benumbed parts, but elsewhere the sensibility to painful impressions is keen enough.

There is also evident impairment of the proper sensibility of the muscles, joints, and bones in the limbs, and especially in the legs. Thus, the patient never knows clearly where his feet are without looking at them, and now and then he has been so uncertain in this respect, that a foot has slipped out of bed without his being the wiser; and thus, again, his finger has not the power of discriminating between a sovereign and a shilling by the weight merely.

The sight of each eye is defective, and glasses afford no relief. The pupils are equal in size, and respond fairly to the light. The conjunctivæ are very much injected. There is no arcus senilis. There is no squinting or ptosis.

The hearing is so dull as to make it necessary to speak in a very loud tone in order to be heard, and one ear seems to be as deaf as the other. There are also constant singing and humming noises in the head—"I still hear the wind in the shrouds," he says.

The memory is bad, the spirits are despondent, and of late (this statement is volunteered by the patient) there has been a frequent disposition to commit suicide.

The pulse is feeble, and about 70 in the minute. The appetite is good. The bowels are somewhat constipated, and a long time is spent over a stool. The urine is voided slowly, and with difficulty, although there is no stricture, and now and then it escapes during sleep. Sexually, the state may be spoken of as approaching to spermatorrhœa.

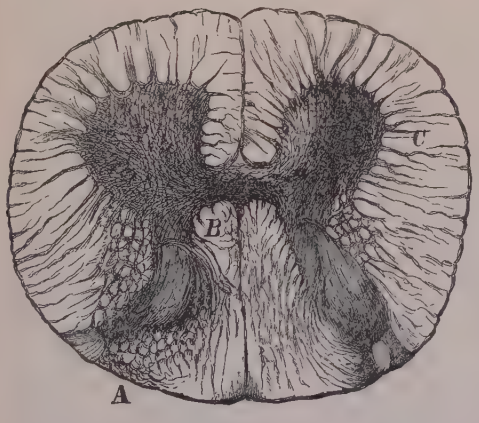
(*b*) Five years ago J. C—— began to suffer from pains in the legs and back, and to be unsteady in his gait; about the same time, also, his sight and hearing began to fail; and from that time to this he has continued to get gradually worse and worse. Four years ago he had sunstroke in the West Indies, of which the immediate symptoms were violent agitation and shaking, without loss of consciousness, and for which he was taken into a hospital and bled. But this accident was twelve months *after* his present malady had commenced, and therefore it is not possible to look upon it in the light of a cause. There never was either squinting or ptosis. He was at sea seventeen years in all, chiefly in hot climates, as the West Indies and the West Coast of Africa, and he remained on board three years after he had begun to suffer from unsteadiness of gait, and from the other symptoms which have been mentioned. Once during the time he was at sea he had chancres, without constitutional symptoms; and repeatedly he had diarrhœa; but, with these exceptions, his health on all occasions appears to have been pretty good. He says that he was always very careless, often sleeping, almost without clothes, on the bare deck, or on the ground, and that he was always "too much given to drink and women." For the last two years the sexual inclinations have been much damped, but before this time, from what he says, he appears to have been little better than a very satyr. Two years ago, when obliged to abandon his calling as a sailor, he was for a while treated in the hospital at Quebec for rheumatism. Afterwards he found his way to this country, and became an out-patient first at one hospital and then at another. During this time he appears to have been frequently blistered along the spine, and on one occasion to have been salivated. For the rest, I have only to add that his father died early in life of consumption; that his mother died young of some unknown chronic disease; and that a brother, the only child besides himself, is now dying of the disease which proved fatal to his father.

This case has not yet ended in a post-mortem examination; and of many other cases which have come under my notice, not one as yet is *complete* in this sense. All, therefore, that I can do is to say that in other cases of the kind the posterior columns of the spinal cord, and the posterior roots of the spinal nerves, are found to be diseased in the lumbo-dorsal region, and that the morbid appearances consist sometimes in a kind of gray degeneration, and sometimes in a gelatiniform and translucent condition, in a diminution of consistency, or in a state of induration

called sclerosis. These changes are confined to the posterior columns of the cord; or if they extend further, it is not to the antero-lateral column, but only to the neighboring portion of the posterior cornu of the central gray matter. In the majority of cases the disease is confined to the lumbo-dorsal portion of the cord, and it is only in quite exceptional instances that it extends upwards, so as to implicate the cervical portion. In the majority of cases, the diseased structure is more vascular than the healthy structure of which it has taken the place, the vessels being more or less deeply imbedded in oil-globules of various sizes, and when examined further, it is found to be made up of atrophied and degenerated nerve-tissue, of the connective tissue in excess, and of amorphous granular matter. Now and then, also, traces of degeneration have been found at the roots or in the course of the optic nerves, or of one or other of the nerves of the muscles of the eye.

[The term *sclerosis* is now, by common consent, applied to the morbid change observed in Locomotor Ataxy, as well as in several other spinal affections. (See Induration, in a later part of this volume.) Charcot and Pierret have endeavored to show that, in Ataxy, the sclerotic alteration begins in the external *bands* (funiculi cuneati) of the posterior columns, near to the posterior roots of the spinal nerves. The lumbar portion of the cord is the first seat of the disease, which afterwards extends to the dorsal, and sometimes to the cervical region. In the upper part of the cord, the columns of Goll (funiculi graciles), which lie next to the posterior fissure, become involved. The gray posterior horns, including Clarke's pillars, at their bases, are also affected. Lockhart Clarke has raised the question whether the diseased action does not begin in the posterior cornua.

Fig. 59.



Pierret's case of Locomotor Ataxy: transverse section of lumbar portion of spinal cord. A. Posterior roots. B. Internal radicular fasciculi. On the right, the sclerosis has extended to the anterior cornu, C, which has suffered diminution in every diameter. Also, the external group of motor cells has disappeared; there being left a dense, opaque, fibroid tissue, containing numerous myelocytes. (Charcot.)

The conclusions of Erb[1] very well represent the present state of knowledge upon this subject.

"It is in the highest degree probable that in tabes we have to deal with a chronic inflammatory process; that it merely represents, therefore, one form of chronic myelitis.

"It is possible, and perhaps probable, that this chronic myelitis may take its origin in two different ways: at one time from a primary irritation and degeneration of the nerve-elements themselves (parenchymatous sclerosis), at another from a primary irritation and proliferation of the interstitial tissue (interstitial sclerosis)—thus giving a double method of origination of tabes, as was believed by Remak, Sr.

"It is possible, and perhaps probable, that the sclerosis begins in the external bands of the posterior columns, and spreads from thence further, and that the sclerosis of the *fasciculi graciles*, or Goll's columns, must, to a great degree, be regarded as a secondary degeneration.

"It is certain that the disease does not begin in the posterior roots.

"It is, finally, probable that sclerosis of the posterior columns is not the exclusive and essential change in tabes, but that a simultaneous involvement of the posterior gray horns and of certain portions of the lateral columns is constant, and, perhaps, equally essential—a statement which, it is true, finds its support more in clinical observation than in the anatomical facts now before us."—H.]

In order to see how far the case of which the notes have just been given agrees or disagrees with other cases of the kind, I single out, as points to be noticed in turn, the following:—Difficulty in standing or in moving about from inco-ordination of movement in the lower extremities; no true paralysis in the lower extremities; neuralgic pains, in the feet and legs more especially; more or less numbness in all forms of sensibility except that by which difference of temperature is recognized; impaired sight and hearing; no strabismus or ptosis; some incontinence of urine, and some want of control over the lower bowel, without marked paralysis of the bladder or sphincter ani; no obvious impairment of sexual power; no tingling or kindred phenomenon; no marked tremulous, convulsive,

[1 Article, Tabes Dorsalis, in Ziemssen's Cyclopædia, vol. xiii.]

or spasmodic phenomena; no marked impairment of muscular nutrition and irritability; some impairment of mental and moral power; some injection of the conjunctivæ with contraction of the pupils; the sex and age; and, lastly, the frequent limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power, to the lower extremities).

Difficulty in standing or moving about from want of co-ordinating motor power to the lower extremities.—This difficulty is very evident, especially in the act of rising from a chair or in turning round suddenly when walking. If the patient cannot avail himself of some sufficient support at the time, the disorder in the movements of the leg produced by the act of rising from the sitting position, or of turning round suddenly when walking, is apt to throw him down. Walking is possible without a stick, but the gait is precipitate, staggering, the legs starting hither and thither vaguely, and the heels coming down at each step in a way which has gained for such patients at Gräfenberg the epithet of *stampers*. Moreover, it is less difficult to move on than to remain long in one position standing. In order either to stand or walk, however, the help of the sight is necessary. In less advanced forms of the disease, it may be difficult at first to detect inco-ordination in the movement of the legs, but this difficulty is not likely to last long. Often a first sign is reeling about upon getting out of bed in the dark. The patient may fall more than once under these circumstances, and think that he is only half awake or half sober. In that early stage of locomotor ataxy in which there is no evident inco-ordination of movement while the eyes are open, there is likely to be such disorder when the eyes are shut: and in an earlier stage still, even when it may be possible to stand steadily with the eyes shut, provided the patient be allowed to plant his feet where he pleases, it is more than likely that he will lose his balance if he be made to stand with the inner edges of the feet in close apposition. In more advanced stages of the disease, walking, or even standing, becomes altogether impossible, and it is curious to notice the extreme disorder in the movements of the legs when the patient is propped up under the arms, and made to try to walk or stand; for under these circumstances the legs are seen to go every way but the right way—backwards, forwards, sideways, unless it happens, as it often does, that they get foul of each other, and become interlocked. In all cases, indeed, the inco-ordination of movement in the lower extremities, by which standing and moving about are interfered with in a greater or less degree, is a constant symptom in locomotor ataxy; and in a case of average severity, like the one under consideration, the gait, arising from this want of co-ordination, is quite characteristic—namely, precipitate, staggering, the legs starting hither and thither, and the heels coming down with a stamp at each step.

No true paralysis in the lower extremities.—When the patient is sitting or lying, he can, provided he sees what he is doing, move either leg singly, into any position with tolerable precision, and keep it there steadily; and the muscular force at his command is such, that it is out of my power to straighten the limb if bent, or to bend it if straightened. There is plainly no paralysis. Nor is it otherwise in other cases of locomotor ataxia, not even in those extreme cases in which the inco-ordination of movement in the legs has proceeded to the extent of making standing an impossibility. And, certainly, it is no objection to the conclusion that my patient was speedily fatigued by the acts of standing or walking, for in reality this fatigue may easily be accounted for by referring it to the effort necessary to keep the ataxic movements of the leg in check.

Paroxysms of neuralgic pain, in the feet and legs principally.—My patient's chief complaint was of neuralgic pains—pains boring, stabbing, or shooting in their character, pains like those caused by a sharp electric shock—in various parts of the lower extremities, in the feet especially, and sometimes in the arms and abdomen, occurring in paroxysms varying in duration from a few minutes to many hours, flitting from one spot to another, but generally remaining at the same spot in the same paroxysm. And this was the chief complaint from the very beginning of the malady. Nor is this case at all exceptional in this respect: on the contrary, pain of the same character is met with in the great majority of cases of locomotor ataxy. Moreover, Dr. Trousseau speaks of this symptom as the most constant precursory phenomenon of the disease. In some cases, no doubt, pain is either absent altogether, or present only as an occasional symptom of very secondary importance. I have myself met with four cases of well-marked locomotor ataxy in which there was no pain, or none to speak of. The pain *may* begin in a way in which it may be mistaken for rheumatism, and be slow in acquiring its special character, but it has, as a rule, these special neuralgic characters from the first throughout.

Numbness in all the forms of sensibility excepting that by which differences of temperature are recognized.—In the case under consideration the sense of touch is almost annihilated in the soles of the feet and in the lower parts of the calves of both legs, and it is impaired greatly in the back of the thighs, in the nates to a less degree,

and in the palms of both hands. In the parts also which are thus benumbed tickling is felt very obscurely, or not at all, and very trifling pain or none at all is caused by pinching or pricking. In the legs also the "muscular sense," as well as the special sensibility of the joints and bones, are considerably impaired, as is evident in the fact that the patient does not know where his feet are unless he can see them. Indeed, the only form of sensibility which seems to be unimpaired, is that by which differences of temperature are recognized. In other cases of locomotor ataxy, also, a similar state of things as to sensation would seem to be the almost constant rule, the numbness beginning, first in tactility, then in the sensibility to pain and tickling, afterwards passing to the "muscular sense," and always, curiously, skipping over, or leaving off before reaching, the sense by which differences of temperature are perceived. In some instances the sensibility of the mucous membrane of the anus and urethra is greatly deadened. The numbness is most marked in the lower extremities, especially in the feet, and very often it is confined to these parts, but now and then it may extend further. I know of one case in which the tip of the nose and the middle of the upper and lower lip are thus affected.

It would seem to be the rule for numbness to make its appearance at the same time as inco-ordination of movement, and for the two symptoms to make progress *pari passu;* but there are cases of locomotor ataxy in which, to say the least, numbness in any form is a very inconspicuous phenomenon. Moreover, it is certain that cases of well-marked locomotor ataxy are met with in which the "muscular sense" is not affected. Out of nineteen cases, I have met with two such. I believe, also, that in cases of locomotor ataxy in which the "muscular sense" is affected considerably, it will be often found that this form of numbness makes its appearance *after* the inco-ordination of movement, and not before it. In a word, I believe that the history of locomotor ataxy furnishes little countenance to a theory which has been advanced—that the inco-ordination of movement in this disorder is nothing more than the consequence of loss of muscular sense.

In some exceptional cases of locomotor ataxy there may be numbness in some parts, and an opposite state of things in others. Thus, I have myself met with a case in which there is anæsthesia almost complete in the lower extremities generally, and the most distressing hyperæsthesia as to tickling in the thumb. But, as I have said, cases of this kind are quite exceptional.

Impairment of sight and hearing.—Impairment of sight appears to be a common symptom in locomotor ataxy; impairment of hearing an occasional symptom. In the former case, Dr. Hughlings Jackson has shown that in the cases where sight is impaired or lost there is a *gradual* whitening of the optic disk without any marked change in the size of the retinal arteries and veins—a chronic form of atrophy which is more common in men than women, and which is not at all peculiar to locomotor ataxy.

[T. Grainger Stewart reports the occurrence of *color-blindness*, in three out of twenty cases of the disease.[1]—H.]

Strabismus and ptosis.—Dr. Duchenne and Dr. Trousseau both speak of strabismus or ptosis as frequently met with in the early stage of locomotor ataxy, as frequently passing off after a time, and not unfrequently as returning, to remain permanently, at a later period. Dr. Duchenne has also twice met with paralysis of the fifth cranial nerve concurrently with paralysis of the third. Speaking of these symptoms, Dr. Trousseau says, "Some may be absent, but it rarely occurs that they are all absent in the same case. I have nearly always found them, and Dr. Duchenne is right in attaching great importance to them for diagnosing the disease at the onset. Remember, besides, that they may have been transitory, and been forgotten by the patient, so that the physician must needs make careful inquiries in order to discover their existence in the patient's previous history." Ptosis or strabismus was not present in the case which I have given, and never had been; and the same may be said of seven out of eighteen other cases of locomotor ataxy which have come under my notice. In the remaining eleven cases, strabismus or ptosis, one or both, were either present at the time of observation, or had been present for a time at an earlier period, generally at the onset of the disease. I find, also, as Dr. Trousseau did, that these paralytic affections of the muscles of the eye, or the impairment of sight or hearing, may be present at an early stage of the disease, may disappear for a while, and then reappear at a later stage.

No very obvious paralytic condition of the bladder or lower bowel.—Incontinence of urine at night, and now and then at other times, as after unusual fatigue, is a common and often a very early symptom in locomotor ataxy, and a less common, and usually a comparatively late symptom, is some trifling want of control over the lower bowel. Dr. Trousseau, speaking of the phenomena of the fully developed disease, says, "Just as in confirmed cases

[1 Brain, July, 1879, p. 189.]

of paraplegia, there is paresis of the bladder and rectum, or even paralysis of the sphincters." As it seems to me, however, there is a marked difference between cases of confirmed locomotor ataxy and common paraplegia in these respects, the difference being that in locomotor ataxy there is not that obvious state of paralysis of the bladder, or sphincter ani, which is so generally present in paraplegia. Indeed, I have never met with a case of locomotor ataxy in which the way in which the bladder could be emptied in a steady stream, did not prove that this viscus retained a fair amount of power; and in one or two cases of this disease, in which the feces have passed involuntarily at times, I have found a state of things which enabled me to account for this accident without assuming the existence of paralysis of the sphincter ani, namely, a want of sufficient sensitiveness about the anus. Moreover, I do not find in the cases which have come under my notice one in which the urine was retained, as it so often is in paraplegia, and where the consequences of such retention —cystitis, alkaline urine, and the rest— were present. Indeed, in all my cases the urine has been acid, and otherwise healthy — a state of things which is scarcely compatible with the presence of paralysis of the bladder.

No obvious impairment of sexual power. —From a sexual point of view, it is easy to see that, as a rule, there is a marked difference between locomotor ataxy and common paraplegia, the difference being that in the former disorder there is *not* that impairment of desire and power which is so constantly met with in the latter. Not unfrequently, indeed, it is plain that there is no impairment of sexual power in ataxy; and now and then there is a curious exaggeration of virility, evidenced, it may be, in the aptitude to repeated acts of connection within a short period. Thus, Dr. Trousseau instances two cases in which these acts could be repeated as often as eight, nine, or ten times in a single night, and I have met with one case which is a fit fellow to these. In all these cases spermatorrhœa was a symptom. I also know of two cases of advanced locomotor ataxy in which fertilization has been successfully effected, and other cases of the kind are on record.

No tingling or kindred phenomena.— Tingling, or sensations analogous to tingling, are not among the symptoms noted in the cases of locomotor ataxy which have come under my own notice, and, so far as I know, they have not occurred in other cases of the kind. At any rate, I think it cannot be doubted that such symptoms are infinitely more common in common chronic paraplegia than in locomotor ataxy.

No obvious tremulous, convulsive, or spasmodic phenomena.—Dr. Trousseau says: "At an advanced period of locomotor ataxy, spasmodic contractions are frequently observed, not only when the patient wills a regular movement, but even in the state of rest. In the latter case, they consist in very powerful jerks of the limbs, and are an important symptom of this singular neurosis." But my experience of the disease does not bear out this statement. Moreover, the cases given by Dr. Bazire, in the valuable appendix to his translation of Dr. Trousseau's lecture on locomotor ataxy, is not confirmatory of the passage in the lecture which I have just quoted. Indeed, if I except certain attacks of convulsive agitation, in which one or two patients have now and then awakened out of sleep, and the feeling of constriction in the abdomen and lower extremities, which is occasionally met with, and which may possibly have some remote connection with spasm, I know of nothing in the history of locomotor ataxy which requires a place in the category of tremulous, convulsive, or spasmodic phenomena.

No marked impairment of muscular nutrition and irritability.—This is another feature of locomotor ataxy, and, therefore, another point of difference between this affection and common paraplegia. The electro-sensibility is impaired in the muscles in which the "muscular sense" is impaired, not the electro-contractility.

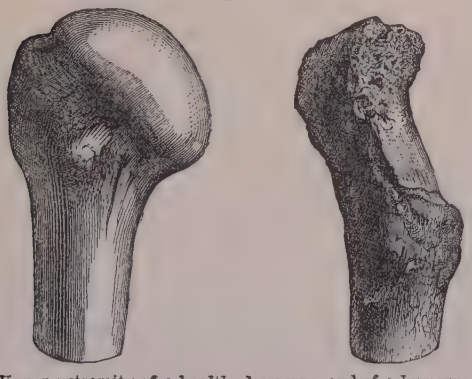
Some impairment of mental and moral power.—Bad memory, despondency, suicidal tendency, are mentioned among the symptoms in the case which serves as my text, but troubles of this kind do not figure in the history of other cases of locomotor ataxy. In fact, it would seem to be the almost constant rule for the mental faculties to be unscathed in this disease.

Some injection of conjunctivæ with contraction of pupils.—In the case under consideration, the pupils were contracted and comparatively disobedient to light, and the whites of the eyes were considerably bloodshot; and this appears to be a not unusual state of things in cases of the kind. Dr. Trousseau says that he has often noticed in ataxic patients, in the intervals between the paroxysms of pain, injection of the conjunctivæ, sometimes as marked as in the most violent conjunctivitis, sometimes amounting to a sort of chemosis, and, in association with this, a state of extreme contraction of the pupils; and he also tells us that he has seen this injection of the conjunctivæ and contraction of the pupils disappear during a paroxysm of pain. In J. C——, I failed to perceive this change during pain. Dr. Bazire also failed to perceive it in others who have come under his notice. I have observed it in two cases, of which that of my friend

M. Ernst, the prince of violinists, was one. In these two cases, what I noticed was this — that the eyes ceased to be bloodshot, and the pupils opened when the pain reached a certain degree of severity and continued for a certain time, and not otherwise. This I observed on several occasions in M. Ernst while he was staying with me on a visit; and I expect that the discrepancy which at present exists between the statements of Dr. Trousseau and his translator upon this point, will disappear as soon as the influence of the degree and duration of the pain is taken into account.

[*Occasional arthritic affections.*—Charcot has described what he designates as the *arthropathy of ataxic patients.*[1] Without known external cause, one of the limbs swells considerably, without pain or febrile reaction. In a few days the tumefaction subsides, except at a joint; commonly the knee, shoulder, or elbow, occasionally the hip-joint. Hydrarthrus exists, but within a week or two the fluid disappears, and crackling sounds on motion show the occurrence of change in the articular surfaces. The joint is at the same time very movable; luxations are not infrequent.

Fig. 60.



Upper extremity of a healthy humerus, and of a humerus affected by ataxic arthropathy. (Charcot.)

In a few months the ends of the bones at the joint may be almost entirely destroyed. Charcot has found these affections to coincide with the extension of sclerosis to the gray matter of the anterior cornua of the cord; a secondary trophic degeneration.

Cutaneous eruptions.—Papular and even pustular eruptions, as well as bed-sores, are, according to Charcot and others, not rarely associated with the lancinating or "fulgurant" pains of locomotor ataxy. These eruptions often follow, tolerably closely, the track of the nerves which are the subjective seats of those pains; and increase or disappear, from time to time, as the painful attacks experience exacerbation or remission.

Disappearance of tendon-reflex.—Within a few years Westphal,[1] Erb and others have called attention to the significance of the absence or modification of phenomena called those of tendon-reflex, in connection with spinal diseases. If a person in health sits with one leg crossed over the other, or upon a table with the legs dangling over its edge, and a smart blow is struck upon the tendon of the quadriceps femoris muscle, at its junction with the patella, the leg and foot will be jerked involuntarily forwards.

Experiments upon animals, by Schulze, Sürbringer, Tschirjew, Gowers and others, have shown the physiological relation of this movement to the nervous system. Section of the crural nerve will abolish it. Gowers found that the time between the tap upon the patella and the muscular movement was sufficient for a reflex process, through the spinal cord. Burkhardt's experiments led him to question the sufficiency of this interval. It is shown, however, that, as there may be loss of cutaneous sensibility with persistence of the movement described (*e. g.* under the influence of ether spray), and disappearance of it when the sensibility of the skin is exaggerated, the phenomenon must be concluded to be of a reflex nature. While some have thought it explicable by direct stimulation of the muscles, through their being suddenly made tense, the generally prevailing opinion is that the contraction is a true excito-motor nervous action, the centre concerned being low down in the spinal cord.

Similar to this is the *ankle-clonus* of recent authors. If the foot is firmly flexed by pressure on the sole, and then the tendo Achillis is tapped briskly, the foot at once undergoes flexion and extension, in rapid succession, for a considerable number of times. Gowers found the average number of contractions to be a little more than six in a second. Joffroy has regarded this as a cutaneous reflex. It has been shown, however, to be quite independent of the sensibility of the skin, or the liability of the muscles of the limb to reflex action from cutaneous excitation. By aid of the myograph, Gowers has ascertained that the commencement of the ankle-clonus occurs too soon after the blow upon the tendo Achillis for the impression to travel up to the spinal cord and back to the muscles of the leg. It is therefore concluded that, unlike the knee

[1 Lectures on Diseases of the Nervous System, Lecture III.]

[1 Archiv für Psychiatrie und Nervenkrankheiten, Bd. v. 1875, p. 819.]

phenomenon, the foot-clonus is produced by the direct stimulation of the muscles both on the front and back of the leg, through suddenly increased tension. The foot phenomenon is less nearly always capable of being brought about in healthy persons than that called the knee tendon-reflex.

Clinically, it has been established, that the tendon-reflex of the knee is quite constantly abolished, early, in cases of Locomotor Ataxy; while it is increased in cases of lateral sclerosis of the spinal cord (spasmodic spinal paralysis). Ankle-clonus has, as yet, been less fully studied in Locomotor Ataxy. It has been found to be produced with unusual facility and violence in lateral sclerosis of the cord. —H.]

The sex and age.—Locomotor Ataxy is, without doubt, more common in males than in females. As regards sex, indeed, it is with this as it is with other disorders of the spinal cord; for out of 177 cases of all forms of disease of the spinal cord tabulated by Dr. Brown-Séquard, as Dr. Bazire pointed out, 128 occurred in men, and only 49 in women. Locomotor ataxy is also a disorder of adult life. In the cases which have come under my own notice the age ranges from 23 to 60, and but few cases are on record in which the patient was under 20. Indeed, the only cases under 20 would seem to be three reported by Dr. Friedrich of Heidelberg; of which the ages are respectively 18, 16, and 15 years.

The probable limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power) to the lower extremities.—In many cases of locomotor ataxy the upper extremities are not affected at all; in others, their sensibility is blunted in one form or other, and their movements are wanting in precision, especially if the sight be defective, or the eyelids closed. In the cases in which the movements of the upper extremities are wanting in precision, there is always, so far as I know, more or less impairment of sensibility, of the "muscular sense" perhaps most frequently; and my belief is, that the want of precision in movement is rather to be ascribed to the want of the proper guidance of sensation than to the loss of any co-ordinating motor power. One ground for this belief is the fact that the disease of the posterior columns of the cord which is met with in locomotor ataxy, and upon which, there is every reason to believe, the want of proper co-ordination in movement is dependent, is confined to the lumbo-dorsal region of the cord in the great mass of cases. Moreover, it is to be remembered that the movements of the arms in a biped like man are not so interdependent as the movements of the legs, and that, on this account, movements of inco-ordination are less likely to occur in the arms than in the legs. It is also very possible that some of the cases in which the irregular movements of locomotor ataxy would seem to have extended from the legs to the arms may not have been true cases of locomotor ataxy. I remember one case of what at first seemed extreme locomotor ataxy, in which the arms were affected as much as the legs, but the patient in this case was totally blind and bedridden, and all but totally deprived of all kinds of sensibility in the arms, and of the "muscular sense" in the legs; and there was no difficulty in believing that the irregular movements of the arms (and possibly those of the legs also) were due, not to impairment in co-ordinating power, but simply to the muscular anæsthesia[1] and the blindness; and I do not remember any case in which the arms were affected in which the patient was not more or less in the same plight, as to muscular anæsthesia, if not as to blindness also.

Looking back, then, at the case which has been cited, and at the comments to which it has given rise, it is not difficult to see that locomotor ataxy is characterized by these symptoms:—

A peculiar gait arising from want of co-ordinating motor power in the lower extremities—a gait precipitate and staggering, the legs starting hither and thither in a very disorderly manner, and the heels coming down with a stamp at each step.

No true paralysis in the lower extremities or elsewhere.

Characteristic neuralgic pains, erratic, paroxysmal, in the feet and legs chiefly—pains of a boring, throbbing, shooting character, like those caused by a sharp electric shock.

More or less numbness, in the feet and legs chiefly, in all forms of sensibility, excepting that by which differences of temperature are recognized.

Frequent impairment of sight or hearing, one or both.

Frequent transitory or permanent strabismus or ptosis, one or both.

No very obvious paralysis of the bladder or lower bowel.

No necessary impairment of sexual power.

No tingling or kindred phenomenon.

No marked tremulous, convulsive, or spasmodic phenomena.

No marked impairment of muscular nutrition and irritability.

No impairment of the mental faculties.

Occasional injection of the conjunctivæ with contraction of the pupils.

[Occasional secondary inflammations of the joints.

[1] Vide article on Muscular Anæsthesia.

Absence or diminution of tendon-reflex movements.—H.]

The probable limitation of the distinctive phenomenon of locomotor ataxy (the want of co-ordinating motor power) to the lower extremities.

For the rest, I will only say that a chronic disease with these characteristics, and without fever or other signs of disordered health, may safely be pronounced to be locomotor ataxy.

Dr. Duchenne, whose description of the disease is the best as well as the first, marks out three stages in locomotor ataxy. In the first stage the patient suffers from paralysis, often only temporary, of one or other of the motor nerves of the eye, from some degree of amaurosis, usually accompanied with unequal pupils, and from the peculiar neuralgic pains. In the second stage the characteristic unsteadiness in standing and moving about begins to show itself together with anæsthesia, the interval between the first stage and the second varying from a few months to several years. In the third stage the malady becomes more profound and general, but the precise point at which the second stage ends and the third stage begins is not very clearly defined. Dr. Duchenne does not regard the affection of the bladder, the rectum, and the genital apparatus as essential symptoms of the disease in any of these three stages; he regards them as *épiphénomènes* only. Dr. Trousseau does not divide the disease into distinct stages, but he speaks of a *premonitory* stage in which paroxysms of pain, spermatorrhœa or impotence, paralysis of one or other of the motor nerves of the eye, and disorder of vision are the symptoms to be met with. As Dr. Bazire says, however, "it is hardly possible to regard these merely in the light of premonitory symptoms, because they form part and parcel of the fully developed disease:" and, in fact, the various symptoms are so mixed up together, and make their *début* at such varying periods, that it is not easy to separate symptoms and arrange them in this stage or that.

[For *Morbid Anatomy*, see *ante*, at close of account of the case whose symptoms are described.—H.]

2. Causes.—In some cases sexual excess would seem to figure as a cause, but not in others—not perhaps by any means in the majority. And this is one reason why it is not well to continue to use the name of *tabes dorsalis* as the equivalent of locomotor ataxy, for rightly or wrongly it has come to this, that the name *tabes dorsalis* is supposed to imply past abuse of the sexual organs. Nor is it possible to speak of syphilis, or rheumatism, or gout, or struma as a cause, for in a great many cases, to say the least, there is no evidence of one or other of these morbid conditions. In fact, it is not possible to refer locomotor ataxy to any special cause. What predisposes to other diseases of the nervous system predisposes to this, family predisposition especially, and this is all that can be said. With regard to family predisposition some curious instances might be given. I know of one case in which one brother is epileptic, another brother hypochondriac, and two sisters are suffering from different forms of paralysis; and Dr. Marius Carré instances a family in which eighteen members have become ataxic in turn—namely, the grandmother, the mother, eight relations of the latter, seven children, and one cousin.

3. Prognosis.—The prognosis of the disease is unhappily full of gloom. Usually, without doubt, the course is slowly but steadily in a downward direction—so slowly, often, that it is only after the lapse of many months, or even years, that the patient distinctly realizes the fact of having become decidedly worse; but on the other hand, several cases are on record in which the disease has advanced to the extent of destroying the power of standing and walking in four or five months. Long pauses in the progress of the disease are not uncommon; thus, for example, I know of one case in which the condition has remained stationary for fourteen years. Moreover, it is not impossible to find a few cases in which the symptoms have changed for the better considerably, and are still changing. Cases of this kind, it is true, are not very common, but they are to be met with. I myself can testify to the existence of several of them.

4. Diagnosis.—Locomotor ataxy, it is said, may be confounded with several diseases, especially with common chronic paraplegia, with simple loss of "muscular sense," with cerebellar disease, and with chorea, but this can scarcely be if only moderate care be taken.

In common chronic paraplegia there is unequivocal paralysis in the lower extremities, and the nutrition and irritability of the paralyzed muscles are, as a rule, unmistakably impaired. In these fundamental particulars, indeed, the difference between this affection and locomotor ataxy is as complete as it can be. In common chronic paraplegia the bladder and sphincter ani are implicated in the paralysis which affects the legs, and the sexual power is almost sure to be greatly weakened or entirely extinguished. In common chronic paraplegia the characteristic neuralgic pains of locomotor ataxy are wanting, and numbness is nothing like so prominent a symptom as

in the ataxic disorder. In common chronic paraplegia, where walking is possible, the gait, instead of exhibiting the want of co-ordination which is met with in locomotor ataxy, is hampered and slow, each leg being brought forward with evident difficulty even with the help of an upward hitch of the body on the same side, and the part of the foot first coming in contact with the ground being, as a rule, not the heel as in ataxy, but the toes. In common chronic paraplegia impairment of sight or hearing, strabismus or ptosis, injection of the conjunctivæ, or contraction of the pupils, frequent, if not constant, symptoms in ataxy, form no part of the history. In fact, in these respects, and in others of minor importance which might be mentioned, the histories of common chronic paraplegia and of locomotor ataxy are so different that it is not easy to see how, with only an ordinary amount of care, the two disorders can be confounded.

The ataxic movements which depend upon anæsthesia muscularis are only present when the patient does not see what he is doing: the ataxic movements which characterize *simple* locomotor ataxy continue whether the patient see what he is doing or not. Nor is this simple rule in diagnosis invalidated by the fact that in the majority of cases of locomotor ataxy the sight has a marked influence in keeping the unruly muscles in check, for the cases are almost exceptional in which loss of muscular sense does not form an important element in the disorder.

In some diseases of the cerebellum there appears to be, often at least, the same disorder of muscular movement which is met with in locomotor ataxy, but this resemblance is more apparent than real. In the next bed to that then occupied by the patient whose case has served as an instance of locomotor ataxy, was a boy, also under my care, whose cerebellum never seemed to have been properly developed, and whose gait was precisely that which I have seen in two cases of tumor of the cerebellum, and which seems to be associated with serious cerebellar disease in all cases. This boy reeled and rolled about in walking, but there was nothing peculiar in the way in which he moved his legs and planted his feet; on the contrary, these movements were those which would be instinctively made to prevent falling. He was not giddy, but merely unsteady, and the volitional and automatic movements of his legs were what they ought to be under the circumstances, no more. His mode of progression was widely different from that of the ataxic patient, as was at once apparent when the two were set to walk side by side; how different I need not again stay to say. In certain diseases of the cerebellum also, some symptoms are likely to be present which will assist in the formation of a correct diagnosis, especially violent pain, often augmented by movement, in one or other part of the head, and frequent and obstinate vomiting, and at the same time other symptoms are likely to be absent which are present in locomotor ataxy; namely, neuralgic pains and anæsthesia in the feet and legs, and elsewhere.

In chorea there is great want of co-ordinating motor power, but the history is quite different from that of locomotor ataxy. Chorea is an affection of childhood and early youth; locomotor ataxy of adult life. The choreic muscular disturbances affect especially the head and arms; the ataxic are chiefly confined to the legs. Moreover, there are not in locomotor ataxy those involuntary movements which in chorea keep the affected muscles in a state of almost perpetual unrest. And as to the other symptoms, it is, in fact, a question of differences, not of resemblances.

It must not be forgotten, however, that the different diseases of the nervous system, like all other diseases, are not fenced in by any boundaries except those which have been fixed almost arbitrarily for the convenience of description, and that cases of a mixed character are continually being met with, which in reality lie across these boundaries in every direction.

5. Treatment.—The treatment of locomotor ataxy is not a subject upon which much can be said at present. No specific treatment can be recommended on good grounds, not even that by nitrate of silver, about which so much has been said of late in Germany and France; and the only treatment which finds favor in my eyes is one of a general character in which figure some preparation of phosphorus with or without cod-liver oil, or arsenic, or bichloride of mercury. [Althaus[1] says that he has "completely cured" two cases with drachm doses of liquid extract of ergot, three times a day, continued for six or eight months.—H.] I should endeavor to act upon general principles, meeting as well as I could any special indication, as syphilis, or gout, or rheumatism, or struma. I should trust to a liberal allowance of stimulants rather than to sedatives for the relief of pain: and for the relief of pain also I should have much confidence in regular shampooing, in faradization, and the use of positive statical electricity. I am also disposed to think that good may be done by the use of irons and crutches, one or both. What is chiefly at fault is the

[1] Amer. Journal of the Med. Sciences, Oct. 1878, p. 348.]

motor power by which the two legs act in concert in standing and moving about; and what is wanted primarily is to do away, as far as possible, with the necessity for calling into exercise this power until it can have had time to recover by rest. This is an intelligible indication, and the use of irons or crutches is an intelligible means of carrying it out. Perhaps it is too much to expect that great good can be done in any way in advanced stages of the disease; but in early stages I cannot but think that the disease, to say the least, might be arrested if the patient would consent for a longer or shorter time to the use of these means. For surely it must go far to neutralize the good to be derived from treatment if the patient is continually trying to do, by walking about without help, or with only the imperfect support of a stick, what the diseased condition of his spinal cord incapacitates him from doing. Nor are these remarks alone applicable to the treatment of locomotor ataxy; on the contrary, they apply equally to the treatment of all forms of spinal disease in which the acts of standing and moving about are at all compromised.

VI. Spinal Irritation.

The first important work on the disorder now generally known as *spinal irritation* was published by Mr. Teale, of Leeds, more than forty years ago;[1] the next by the brothers Dr. and Mr. Griffin, of Limerick, about fifteen years later.[2] To Mr. Teale, indeed, belongs the credit of being the first to direct attention to this disorder, for, in reality, his claim either to priority or originality is scarcely, if at all, invalidated by the short communications which were made previously to medical periodical literature by Mr. Player, of Malmsbury,[3] by Dr. Brown, of Glasgow,[4] by Dr. Darwell, of Birmingham,[5] and still less so by anything written about the commencement of the century by Franks, Nicod, Ludwig, and others. It would also seem to be difficult to find any work of more recent date which deserves to be mentioned as at all equal in merit and importance to that of the brothers Griffin. The name "spinal irritation" was first proposed by Dr. Brown, of Glasgow.

1. Symptoms.—The symptoms of spinal irritation at first sight appear to be as vague and various as those of hysteria. They are in reality so far hysterical as to be not readily distinguishable. When further examined, however, one symptom stands out prominently, with which the others are obviously connected in a peculiar manner, namely, spinal tenderness; and the upshot of the whole matter appears to be that spinal irritation is a definite malady which must not be confounded with hysteria or with any other disorder. For example:—

Case.—In the early part of 1863, an unmarried lady, aged twenty-three, consulted me for pains in the head and face, loss of appetite, nausea, flatulence, palpitation, breathlessness, "sinking feelings," weakness, and low spirits. The pain, which was the chief suffering complained of, was sharp and neuralgic in its character, and varying in its seat, being sometimes in one part of the head or face, sometimes in another, and generally on the left side only. In the head it was often limited to a spot which might be covered with the tip of the finger, as in true clavus hystericus. Headache in one form or another was brought on or exaggerated by any effort, physical or mental: it was usually relieved by lying down and keeping perfectly still; it was scarcely ever absent except when face-ache had its turn; and sometimes it was so continuous and oppressive as to necessitate remaining in bed for days together. Nausea and sickness were its frequent accompaniment, and vomiting and great prostration were its common termination. In the cervical region of the spine there were considerable tenderness and a disagreeable feeling of weight, and pressure there brought on or increased the headache—the pain shooting from the occiput forwards—and caused a feeling of great nausea and oppression at the præcordia. The feet were always cold; "chills and flushes" were of frequent occurrence, and so were yawning, sighing, and stretching of the arms. Sleep was often made hideous by nightmare; fits of lowness of spirits and crying, attended by a sense of choking, as from a ball or knot in the throat, and followed by plentiful gushes of pale, limpid urine, were brought on by the most trival causes; and the manner and appearance were altogether those of an eminently nervous or hysterical person. Menstruation was regular, neither excessive nor deficient, and it could not

[1] A Treatise on Neuralgic Diseases dependent upon Irritation of the Spinal Marrow and Ganglia of the Sympathetic Nerve. By T. P. Teale, 8vo. London: Highley, 1829.

[2] Observations on the Functional Affections of the Spinal Cord and Ganglionic System of Nerves, in which their Identity with sympathetic, nervous, and irritative Diseases is illustrated. By William Griffin, M.D., and David Griffin. 8vo. London: Burgess and Hill. 1844.

[3] Quarterly Journal of Science, January, 1822.

[4] Glasgow Medical Journal, May, 1828.

[5] Midland Medical and Surgical Reporter, May, 1829.

be said that the sufferings were either more or less at this time. The bowels also acted properly, and (but for the disposition to pass large quantities of pale urine, which has been already mentioned) so did the kidneys and bladder.

These symptoms, it appears, had their starting-point about twelve years ago in the shock and grief caused by witnessing the death of a brother, her last remaining near relative, in an epileptic fit, and ever since this time they have continued very much as they now are, with but little intermission. Before this time the personal history of the patient was tolerably good, but not so her family history; for, in addition to the brother whose death in epilepsy has just been mentioned, it appears that her father died years before of phthisis, and that her mother is now in a lunatic asylum.

Under the use of a more liberal diet, with ammonia and calumba, and with occasional blisters to the nape of the neck, health was re-established in little more than a month, notwithstanding the fact that several days at the commencement were wasted in overcoming a dislike to take the wine and medicine necessary—in converting, in fact, the patient from a firm belief in teetotalism and homœopathy.

Towards the close of the same year, 1863, this young lady again returned to me, looking very worn and thin, with all her old symptoms in force, and with cough and difficulty of breathing in addition. The cough was very violent, barking, unattended with expectoration, and often carried on until it ended in retching and vomiting. The difficulty of breathing was chiefly at night: usually it did not amount to more than what might be met by a voluntary effort at inspiration; now and then it seemed to deserve the name of asthma; almost invariably it was accompanied, not by a feeling of a ball or knot in the throat, but by a sharp pain in the left hypochondrium, or else by severe aching in the left shoulder and down the left arm. Percussion and auscultation failed to bring to light any signs of disease in the heart or lungs, but pressure along the spine revealed tenderness in the cervical and upper dorsal regions, in the latter especially, and at the same time brought on cough, deep inspirations, pain and throbbing at the epigastrium, and a feeling of great faintness and breathlessness.

On this occasion a very fair state of health was soon re-established by the plan of treatment which proved successful in the first instance.

At the beginning of 1865, this lady again required my services. For the three weeks before my seeing her she had been in bed, with her thighs drawn up tightly against her abdomen, and with her heels buried in her nates. This contraction was unremitting during the waking state, and only partially remitting during sleep: it was unattended by pain; and it could be partially overcome, for a time, without causing much pain in the contracted muscles, by slow and steady extension. The headache and face-ache had gone months before, and so had the pain in the epigastrium and in the left shoulder and arm; the cough and difficulty of breathing and palpitation were of very unfrequent occurrence; the appetite and digestion and the action of the bowels were tolerably natural; and what was complained of now were colicky pains in the lower part of the abdomen, pains often very severe and sickening about the loins and hips, and in the region of the left ovary, with constant calls to pass water, and much pain in the urethra in attending to these calls. The spine was now tender, not as before in the cervical and dorsal region but low down in the lumbar region, and pressure on the tender part brought on colicky pains in the lower part of the abdomen, and a cutting pain in the urethra, with an almost irresistible impulse to pass water then and there. Pressure in the cervical and dorsal regions of the spine gave rise, not to the marked symptoms produced in this way in the two previous illnesses, but simply to a disagreeable thrill all over the body. There was no numbness or tingling in the legs or elsewhere, and no hyperæsthesia, except perhaps to a very trifling degree over the left ovary. Tickling the soles of the feet gave rise to painful spasmodic shocks in the legs, to a disagreeable thrill passing up the body as high as the throat, and to the involuntary escape of a small quantity of urine. The condition as to general health was tolerably good—much better than during the two previous illnesses: and, in fact, the only sign of disorder, in addition to those which have been indicated (and this can scarcely be reckoned as such), was the absence of menstruation since the birth of a child about three months ago.

Somewhat more than twelve months ago, after having been quite well for the year previously, this patient married and became pregnant. In the early months of pregnancy she had much headache, depression, weakness, and sickness; but after a while these symptoms passed off, and everything went on smoothly and satisfactorily until two months after confinement, when her baby died suddenly. And then began her present troubles. The fretting about her baby brought back the old headaches, the headaches produced great sleeplessness and irritability of the stomach, and then came on a state of uncontrollable fidgetiness, which kept her

incessantly moving about until her legs, one leg especially, failed altogether, and obliged her to take to her bed. The very next morning her legs had become contracted, and she herself is convinced that this change for the worse, as she regards it, was brought about by the pain and loss of blood produced by introducing a large speculum and by applying leeches to the os uteri on the previous evening.

The treatment on this occasion consisted chiefly in a liberal allowance of food and wine, in repeated blisterings to the lumbar region of the spine, and in the administration of bromide of potassium and ammonia; the result was the cessation of the contractions in about three weeks, and the complete re-establishment of health in about two months and a half.

In commenting upon this case with the view of separating the general phenomena of spinal irritation from the particular, I take the following as the points which most deserve to be attended to, namely, these: Spinal tenderness, neuralgia, spasmodic cough and difficulty of breathing, palpitation and vascular throbbings, nausea, vomiting and eructations, and irritability of the bladder, all in connection with spinal tenderness; the connection of particular symptoms or groups of symptoms with tenderness in particular parts of the spine; prolonged muscular contraction; no paralysis of the limbs; no paralysis of the bladder or rectum; no numbness; variability and inconstancy of the symptoms; a nervous constitution.

Spinal tenderness.—In the great majority of cases this symptom would seem to be present in spinal irritation and absent in spinal meningitis, myelitis, or spinal congestion, acute or chronic. It would seem, indeed, to deserve to be regarded as the pathognomonic symptom of spinal irritation; for in the few cases of spinal meningitis, myelitis, or spinal congestion in which it is met with, there is reason to believe that its presence may be accounted for by the association of the phenomena of irritation with those of inflammation or congestion. At any rate, it is certainly the rule that spinal irritation without spinal inflammation or congestion is accompanied by spinal tenderness, and that spinal inflammation and congestion without spinal irritation is *not* accompanied by spinal tenderness. Spinal tenderness, however, can scarcely be spoken of as a prominent symptom in spinal irritation. It is often not complained of until it is specially inquired after; and now and then its existence is not even suspected by the patient until he or she is made to wince under pressure applied to the spine. In a few cases which from their symptoms would seem to come under no other head than that of spinal irritation, there is no spinal tenderness—only five such cases are met with among the 148 cases brought together by the brothers Griffin, and these may without difficulty be in great measure explained away; but such cases are much too exceptional and doubtful to throw discredit on the rule in question, that spinal irritation and spinal tenderness go together. Spinal tenderness, however, does not appear to be equally marked in all forms of spinal irritation. It appears to be much less marked where the irritation shows itself in spasm and prolonged muscular contraction than in the cases where it shows itself in pain; and it is certainly absent in tetanus, which in one sense may be looked upon as the manifestation of spinal irritation in its most aggravated form.

Nervous pains, often in connection with tenderness in a particular part of the spine.—Nervous pains, neuralgias, in one place or another, often intermittent and more or less regularly periodical, and often shifting suddenly from one place to another, are a very common, perhaps the most common, symptom in spinal irritation. They are often brought on or exaggerated by lifting any weight, by twisting or straining the back in any way, or by any effort, mental or physical: and as often they are relieved, to some extent at least, by lying down. Very often, also, there is tenderness in the portion of the spine corresponding to the insertion of the affected nerves—in the upper cervical region, where the pains are in the scalp (clavus hystericus, megrim, and others), face, or neck; in the lower cervical region, where they are in the upper extremities, shoulders, and upper part of the thorax; in the dorsal region, where they are in the lower part of the thorax and upper part of the abdomen (pleurodynia, gastrodynia, infra-mammary stitch, and others); in the lumbar and cervical regions, where they are on the lower part of the abdomen, hips, loins, and lower extremities. In the majority of cases the pain would not seem to be in the part of the spine which is tender, or in any other part. In some cases there may be aching in some part of the spine, or else a sense of weight and heat; but I am very much inclined to believe that these last mentioned symptoms, and "back-ache" generally, have often to be referred to spinal congestion rather than to spinal irritation in its uncomplicated form. When the spinal tenderness is very great, slight pressure will often cause pain to strike from the tender spot of the spine to the distant seat of pain, or will bring about or exaggerate this pain. This fact is illustrated in the case I have given, and better still in some of the cases related by the Griffin brothers. In one of these cases, for example, where the whole spinal column was found to be

acutely tender, "pressure of the first or second vertebra occasioned pain, which shot forwards from the occiput to the brow; a little lower, pain was excited at the larynx; on pressing one of the lower cervical, it occurred at the point where it dips behind the sternum; on pressing the upper dorsal, at the middle of the sternum; from the third or fourth dorsal to the eighth or ninth, it was excited at the ensiform cartilage; yet lower, at the sides; and in the lumbar vertebræ, pain was excited in the iliac and pubic regions" (p. 19). And in another case, where there was some tenderness of the middle cervical vertebræ, and acute tenderness from the fourth dorsal to the eighth or ninth, "pressure on any of those last, especially the seventh or eighth, brought on violent pain, which darted forwards to the ensiform cartilage. When the last-mentioned vertebra was pressed upon, the patient said that she thought her 'heart would break'" (p. 119). The pain is often curiously localized: sometimes it gives the idea of a nail being driven into the part, as in clavus hystericus; sometimes the feeling produced by it is as if a walnut or other hard substance were pressed under a tight belt; sometimes it is very severe, and neuralgic in its character rather than rheumatic: and not unfrequently, when it has existed some time, the painful part becomes tender on pressure. Most generally this morbid sensation is in the form of pain, but now and then it may take that of cold, tingling, itching, or some other feeling which is disagreeable rather than painful. The amount of constitutional disturbance attending the pain varies very much, but it is usually comparatively trifling, and, as it would seem, quite out of proportion to the degree of suffering.

Nausea, retching, vomiting, eructation, &c. often in connection with tenderness in a particular part of the spine.—These are common symptoms in spinal irritation: next to pain, indeed, they are perhaps the most common. They are also intimately connected with certain forms of pain, especially cephalalgia and gastrodynia, sometimes preceding, sometimes accompanying, but more generally following, the pain. As regards the particular part of the spine which is likely to be tender when the stomach is the seat of irritation, the Griffin brothers say that "nausea and vomiting appear to bear more relation to tenderness of the cervical spine, pain of stomach to tenderness of the dorsal; but that where there was soreness of both, nausea and vomiting was still more frequent, and pain of stomach scarcely ever absent." The epigastric disorder in these cases is generally accompanied with tenderness on pressure, not merely in the spine but also in the epigastrium and in the left hypochondrium—with those three patches of tenderness which M. Briquet speaks of as the "trépied hystérique"—as the tripod upon which the diagnosis of hysteria rests.

Spasmodic cough, difficulty of breathing, &c., often in connection with tenderness in a particular part of the spine.—These again are symptoms which are common enough in spinal irritation, and mostly so, as it would seem, when the tenderness in the spine is in the cervical and upper dorsal region.

Palpitation, &c., often in connection with tenderness in a particular part of the spine.—Palpitation is another symptom of spinal irritation which seems to be oftenest met with when there is tenderness in the upper half of the spine. It seems to be not unfrequently associated with a feeling of epigastric pulsation, and with nausea, vomiting, and other signs of gastric disorder. Vascular throbbings in other places, as in the temples, and "chills and flushes," and a disposition to syncope, and other signs of disturbed balance in the circulation, may, and often do, go hand in hand with the palpitation, and seem to have to do with the same condition of the spine.

Irritability of the bladder, often in connection with tenderness in a particular part of the spine.—This was a marked symptom in the case which I have related when the seat of spinal tenderness shifted to the lumbar region, and it seems to be a very common, if not a constant symptom, in cases in which the tenderness is in this region.

The connection of particular symptoms or groups of symptoms with tenderness in particular regions of the spine.—The data best calculated to illustrate this connection are those supplied by the brothers Griffin. These consist of no less than 148 cases, of which 26 are in males, 49 in married women, and 73 in girls. In these 148 cases, the spinal tenderness was in the cervical region in 28, in the cervical and upper dorsal region in 46, in the dorsal region in 23, in the dorsal and lumbar region in 15, in the lumbar region in 13, and in the spine generally in 23. In the following table the prominent symptoms connected with each one of these forms of spinal tenderness are set forth in a way which requires no comment except this—that this grouping of symptoms with tenderness in particular parts of the spine must only be looked upon as approximating to the truth, and that now and then any symptom may appear out of the order in which it is set down.

Region of Spinal Tenderness.	*Prominent Symptoms.*
A. *Cervical region.* Cases 28 in number.	Headache, nausea, vomiting, face-ache, fits of insensibility, cough, pains in the upper extremities, &c. *** Nausea and vomiting in 5 cases, pains of stomach in 2 only.
B. *Cervical and Dorsal region.* Cases 46 in number.	In addition to the symptoms in group A, pain of stomach and sides, pyrosis, palpitation, oppression. *** Pain of stomach in 34 cases, nausea and vomiting in 10.
C. *Dorsal region.* Cases 23 in number.	Pain in the stomach and sides, cough, oppression, fits of syncope, hiccup, eructations. *** Pain in the stomach in almost all these cases, nausea or vomiting in only one.
D. *Dorsal and Lumbar region.* Cases 15 in number.	In addition to the symptoms in group C, pains in the abdomen, loins, hips, lower extremities, dysury, and ischury. *** Nausea in only one case.
E. *Lumbar region.* Cases 13 in number.	Pains in the lower part of the abdomen, testes, or lower extremities, dysury, ischury, disposition to paralysis in lower extremities. *** Retching and spasm of the stomach in one case only.
F. *All regions together.* Cases 23 in number.	A combination of the foregoing groups of symptoms, one group changing into another as the spinal tenderness becomes more marked in one region than in another.

Prolonged muscular contraction.—This is a very conspicuous symptom in the case which serves as my text, and it is no uncommon symptom in other cases of spinal irritation. The lower extremities appear to be the parts most commonly affected, one or both of them; but the upper extremities can claim no exception, nor even the muscles of the jaws and neck, trismus or torticollis being among the results in this latter case. "Occasionally," says Mr. Teale, "there is an inability to perform complete extension of the elbow, the arm appearing restrained by the tendon of the biceps, pain and tightness being produced in this part when extension is attempted beyond a certain point;" and to this fact I can testify. Moreover, I can testify as to the not unfrequent occurrence of long-continued closing of the fingers and thumb upon the palm. The rule appears to be, for the extremities to be affected before the trunk or head. This contraction, which is generally painless, may be prolonged for weeks or even months continuously, even during sleep, or with occasional intermissions of uncertain duration; and the attacks, primary or secondary, are usually found to begin and end suddenly and unexpectedly. The relations between this form of contraction and that which occurs in other cases, especially in tetanus and in that somewhat vague disorder to which Dr. Trousseau has given the name of tetany (tétanie), are not very easily determined. In tetanus, with very rare exceptions, the contraction is painful, especially in the paroxysmal bouts, and the order in which it attacks the body is different—first, the jaws; then the trunk; and the extremities only at a late period, if at all. In tetany, as in tetanus, the contraction is painful, but the order in which the body is attacked is different to that which is observed in tetanus, centripetal not centrifugal,—first the extremities, then the trunk or head; the contraction, in fact, being confined to the extremities, except in cases of unusual severity. In the way in which it affects the extremities first, and often exclusively, the contraction of tetany agrees with the contraction under consideration, but in other respects it differs. It differs, especially, in being ushered in and accompanied by symptoms which do not seem to form part and parcel of simple spinal irritation; namely, tingling and some degree of anæsthesia, and also (so it is said) in the form of the contracted hand being peculiar—like that which the hand of the accoucheur takes in order to be introduced into the vagina—and in the possibility of bringing on the contraction by firm pressure upon the principal nerves or arteries of the affected muscles. It may be questioned, however, whether there are absolutely fixed lines of

division between these different forms of prolonged contraction, and whether the difference which exists may not be accounted for as the result of different degrees of irritation, affecting, it may be, different parts of the spinal cord. It may be questioned, also, whether a sufficient case is made out for describing tetany as a definite disorder, and whether it is not rather a form of spinal irritation complicated with some graver spinal disease—spinal meningitis, myelitis, spinal congestion—in varying proportions. The association of tingling and numbness with the prolonged contraction is, as it seems to me, a reason for an affirmative conclusion. At any rate, prolonged muscular contraction, be its significancy in tetanus or tetany what it may, must be looked upon as a not unfrequent symptom in single spinal irritation—as a symptom, too, which is usually of no very grave import. Of this there need be no doubt.

No paralysis of the limbs.—In the case I have given in illustration there was great weakness of the legs, and one leg seemed to "drag" immediately before the contractions came on. There was a disposition to paralysis in the legs, but not more than this; nor do I find paralysis of the limbs among the symptoms of spinal irritation strictly so called. There is, no doubt, a connection between paralysis and spinal irritation which cannot be overlooked; and under that form of paralysis which is known as "hysterical paralysis," and about which more will have to be said in due time, and under spinal irritation, there is a common basis. As it seems to me, however, it is pathologically as well as physiologically incorrect to speak of hysterical paralysis as a symptom of spinal irritation. Also, it seems to me, the right place of this paralysis is after spinal irritation, not along with it, when the capability of morbid action which is implied in the term irritation is worn out; and so in the other exceptional cases in which paralysis is connected with spinal irritation, it will, I believe, be found on careful examination that the paralysis is not a symptom of actual spinal irritation, but of a state of vascular change into which this irritation may issue and has issued—spinal congestion, it may be, or even myelitis.

No paralysis of the bladder or bowel.—The remarks which have just been made apply equally to paralysis of the bladder or sphincter ani. Paralysis in either of these parts, or even a disposition to it, is rarely met with in any case which can be strictly brought under the head of spinal irritation; and in the few exceptional instances which do occur, it is plain enough, when the matter is fairly inquired into, that the boundary has been passed which separates the state of irritation from the state of exhaustion, and that, in fact, the case is no longer one of simple spinal irritation.

No numbness.—Numbness, again, is a symptom which is scarcely ever met with in cases to which the name of spinal irritation is strictly applicable, and, when it is met with, it is easily accounted for. In short, the relationship of numbness and paralysis to spinal irritation appears to be one and the same, the numbness and the paralysis being alike connected, not with the state of morbid action called irritation, but with the after-state of morbid inaction for which exhaustion seems to be one of the appropriate names.

Variability and inconstancy of symptoms.—One most characteristic feature of spinal irritation is the way in which one symptom or group of symptoms may change, and change suddenly, into another symptom or group of symptoms. It is now this disease which is simulated, now that, there being scarcely any disease which may not be copied: at one time the head is affected, at another the chest, at another the abdomen or the extremities: and the only thing constant among these ever-shifting phenomena appears to be this—that the spinal tenderness changes from one part to another in a manner which is intelligible enough when the connection of the spinal nerves with the affected part is taken into consideration.

A nervous constitution.—The subjects of spinal irritation, with few if any exceptions, may be spoken of as hysterical, hypochondriacal, or nervous. They have, in fact, that nervous constitution which Whytt, following in the steps of Sydenham, showed to be the common basis of hysteria and hypochondriasis. First in order among the signs of this constitution comes that sign which Sydenham regarded as pathognomonic of hysteria and hypochondriasis—namely, a proneness to pass, under or after strong emotion or excitement, large quantities of pale limpid urine. Then come other signs scarcely less characteristic: proneness to tenderness, not only in some part of the spinal column, but also in the epigastrium and left hypochondrium—*le trépied hystérique* of Dr. Briquet already referred to; proneness to sudden and distressing flatulent distension of the stomach and bowels, with loud rumbling and explosions, and with a feeling of a ball rolling about, first in the left flank, and then mounting, or tending to mount, into the throat, where it gives rise to a sense of choking and to repeated acts of swallowing; proneness to bursts of crying and sobbing or of laughing; proneness to sighing, yawning, and stretching the arms; and proneness to fits of convulsive agitation and struggling. Then comes a promiscuous series

of signs: proneness to erratic pains of a neuralgic character, breathlessness, nervous cough, palpitation, throbbing in the temples, epigastrium, and elsewhere; "flushes and chills," syncope, hiccup, nausea, vomiting, aversion to food or unnatural craving for it, heartburn, oppression at the præcordia, languor, debility, fidgetiness, tremulousness, vertigo (especially on rising hastily), ringing in the ears, fickleness, fancifulness and inability to discriminate between fact and fiction, undue lowness of spirits or the contrary, and other symptoms whose name is legion. Not only, indeed, is the name of these different symptoms legion, but there is ever going on a process of mutual metamorphosis in the symptoms themselves; and, in conclusion, it is this very hysterical or hypochondriacal variability and mutability of the symptoms which must be looked upon as the great characteristic of the nervous constitution.

2. Post-mortem Appearances.—The morbid structural changes strictly belonging to spinal irritation are *nil*. The disease is nervous or functional in its character, and on this account it leaves no obvious traces after death. Still, as Dr. Copland wisely says, "an affection which may with justice be viewed as functional to-day—as spinal irritation merely—may be inflammation on the morrow, and rapidly followed by the consequences of inflammation." Such a termination, however, is altogether exceptional; and when it does occur, the history during life will show very clearly that any traces of inflammation which are met with after death are to be ascribed, not to irritation, but to inflammation. How far irritation, which involves in its very essence, as I believe, capillary contraction and bloodlessness, not capillary paralysis and congestion, may involve changes which are opposed to inflammation—deficiency of blood and organic changes brought on by the part being starved for want of blood—remains to be seen. I take it that such changes would have been found if they had been looked for with the same amount of care which has been expended in the search for inflammatory changes; but the investigations have yet to be made which will verify or disprove this conjecture.

3. Causes. — Neglect of gymnastic training, insufficiency of wine or other alcoholic drinks, over-indulgence in sexual matters, onanism, would seem to deserve a conspicuous place among the causes of spinal irritation. It is idle, however, to weigh the importance of particular causes, or even to attempt to individualize them, and it is enough to be content with the broad fact that everything which tends to induce a nervous habit—that is, everything which exhausts vital power—must be reckoned as a cause. I believe that the starting-point of the disorder will very often be found in some strain or blow to the back, and I also believe that a congenital predisposition may also be detected in very many cases.

4. Diagnosis.—The fundamental question for consideration in this place is how to distinguish between functional and organic affections of the spinal cord, and this question fortunately is one which is less difficult to answer than it might seem to be at first sight. In fact, the characteristics of spinal irritation indicated by the Griffin brothers are sufficient of themselves to supply the answer to any one who has tolerably clear ideas respecting the principal diseases with which spinal irritation may be confounded. These characteristics are:—"1st. The pain or disorder of any particular organ being altogether out of proportion to the constitutional disturbance. 2d. The complaints, whatever they may be, being usually relieved by the recumbent posture, and always increased by lifting weights, bending, stooping, or twisting the spine. 3d. The existence of tenderness at that part of the spine which corresponds with the disordered organ, and the increase of pain in that organ by pressure on the corresponding region of the spine. 4th. The disposition to the sudden transference of the disordered action from one organ or part to another, or the occurrence of hysterical symptoms in affections apparently acute; and 5th. The occurrence of fits of yawning or sneezing, which, though not very common symptoms, yet, as rarely ever occurring in acute organic disease, may generally be considered as characteristics of nervous irritation."

In the diseases of the spinal cord which have already been under consideration—spinal meningitis, myelitis, spinal congestion, and tetanus—it has been seen that it is the rule for the spine *not* to be tender on pressure, and in spinal irritation it has been seen that such tenderness is so constant as to deserve being reckoned as the distinctive feature. Here, then, is a point of difference which will serve as a guide to a correct diagnosis in several important cases in which guidance is necessary—which will serve as a guide in almost all cases except in that with which spinal irritation is most readily confounded. This case, which is strumous disease of the vertebræ, is one in which spinal tenderness is also present, as well as many other symptoms of spinal irritation—pain in the side, stomach, or bowels, cough, oppression, tightness around the waist, and so on—and in which relief is obtained by reclining. Nay, there may even be in spinal irritation a yielding and projection

of the tender vertebræ, with some puffiness of the overlying skin, which simulates in no imperfect manner the earlier stage of angular curvature. There are many resemblances, in fact, but, as the brothers Griffin have pointed out, there are also certain differences which are so well marked as not to leave the diagnosis in doubt. Thus it is found:—"1st. That strumous disease of the vertebræ attacks the young, and most frequently those under the age of puberty, who are least of all liable to be affected by spinal irritation. 2d. That disease of the vertebræ, when attacking young girls, is seldom accompanied by symptoms of a purely hysterical character, while any serious irritation of the cord can scarcely exist without them. 3d. That an apparent prominence of the tender portion of the spine, which sometimes exists in cases of irritation, is never strictly angular; for, if four or five of the vertebræ seem to project, the prominence is nearly equal in all, whereas in caries of the bones it is greatest in the middle, the prominence depending, in fact, on a slight puffing of the ligaments or investments of the spine, and not on displacement or curvature. 4th. That absolute paralysis of the lower limbs is a rare consequence of irritation, and a frequent one of caries of the bones. 5th. That the general health suffers less in the former complaint, and it is not attended with the look of serious organic disease which is indicative of the latter. 6th. That the constitution of the patient may also prove useful as a guide, the disposition to spinal irritation, as well as to scrofula, being hereditary."

5. Prognosis.—However urgent the symptoms may be, the prognosis in spinal irritation is favorable rather than unfavorable. It must always be borne in mind, however, that spinal irritation is a state which may issue in inflammatory or other organic changes in the cord or in its membranes, and that a favorable prognosis must be qualified by this contingency, especially in those cases in which there is some obvious vice of the constitution—scrofulous, gouty, rheumatic, syphilitic, or other.

6. Treatment.—"Local depletion by leeches or cupping," says Mr. Teale, "and counter-irritation by blisters to the affected portion of the spine, are the principal remedies. A great number of cases will frequently yield to the single application of any of these means. Some cases, which have even existed for several months, I have seen perfectly relieved by the single application of a blister to the spine, although the local pains have been ineffectually treated by a variety of remedies for a great length of time." Of the efficacy of blisters in these cases I have had abundant proof. As to the good effects of local depletion I have had less experience, partly because I found that the blisters were sufficient of themselves, and partly because I believe that the state of irritation is associated with a state of capillary contraction and bloodlessness, and not with a state of capillary paralysis and congestion. Still, I can well believe that there are many mixed cases in which irritation has issued in some degree of capillary paralysis and congestion, especially in the skin at the seat of spinal tenderness, and in which this state will be greatly relieved by local depletion.

As regards medicine, I should certainly be disposed to trust most in common tonics—quinine, steel, or cod-liver oil; to the latter in conjunction with some preparation of phosphorus most of all, perhaps. And certainly I should be disposed to fight against pain and spasm, as I have sufficiently explained elsewhere, by remedies which rouse the circulation to greater activity, and not by those which have a contrary action. Nay, I should even have more confidence, as a local application for pain, in some application which would produce a hyperæmic condition of the skin, than in any one which had a deadening effect upon the sensitiveness of the part.

It is, no doubt, an indispensable part of the treatment to avoid standing or walking to the extent of producing fatigue, but there would seem to be no necessity, except as a very temporary measure, perhaps, to insist upon a recumbent position being retained for any length of time. Upon this point Mr. Teale says (and he says all that need be said), "When my attention was first directed to this subject, I considered recumbency a necessary part of the treatment; it is, for a moderate length of time, undoubtedly beneficial, and frequently very much accelerates recovery; but subsequent observation has convinced me that it is by no means essential. I have seen several instances of the most severe forms of these complaints, occurring in the poorer classes of society, where continued recumbency was impracticable, which have, nevertheless, yielded without difficulty to the other means of the treatment, whilst the individuals were pursuing their laborious avocations."

As regards diet I have only this to say—that I believe the great thing to be done is to supply wine or some other alcoholic drink as well as nutritious food in sufficient quantity. I believe that nutritious food in itself is not enough. In very many cases it is found that alcoholic drinks are either abstained from altogether or taken in very insignificant quantities from a fear that they will aggravate the pain or

spasm, or for some other reason: in very many cases it is found also that relief is obtained only when this practice is abandoned, and the diet made to include at least an average share of the drinks in question. Indeed, the result of my own experience is unequivocal in this respect —that the somewhat bold use of alcoholic drinks is a cardinal point in the treatment of spinal irritation, and this indication must be fully acted upon if this treatment is to lead to anything like satisfactory results. [Yet, for safety, this *boldness* must be regulated by the discretion of the physician, not trusted to the mere inclination of uninstructed patients; or else harm, instead of good, may be done by it.—H.]

Of the spinal maladies remaining to be noticed the principal are these:—General spinal paralysis, hysterical paraplegia, reflex paraplegia, infantile paralysis, hemorrhage, non-inflammatory softening, induration, atrophy, hypertrophy, tumor, concussion, compression, caries of the vertebral column, spina bifida, &c.

VII. General Spinal Paralysis.

There is a form of general paralysis to which Dr. Calmiel gave the name of general paralysis of the insane, and with which all who know anything of insanity are sufficiently familiar. It may coexist with any form of insanity, but it is most commonly associated with the monomania in which the patient believes himself to be possessed of unbounded opulence. The first signs are likely to be thickness of speech, quivering of the lips and tongue, fumbling and clumsy movements of the fingers, with an unsteady and sideling gait. Then the urine escapes now and then involuntarily, or even the feces. Once begun, the downward course of the malady is headlong, and in a few months, in a few weeks it may be, within two or three years at the most, the patient is in bed, altogether without the power of supporting himself on his feet, unable to use his hands so as to help himself in any way, incapable of sitting up or even of turning over in bed, requiring to be fed like a child, and, when fed, in no small danger of choking if left to masticate the morsels, with urine and feces escaping under him unheeded, and with every power of body and mind an utter wreck. With few exceptions the thickness of speech shows that the muscles of the tongue and lips are the first to fail, but in fact all parts of the muscular system show signs of weakness about the same time, and it is difficult to fix upon any one part and say that it is affected before the rest. Sometimes the paralyzed muscles become considerably atrophied, but the rule appears to be that such atrophy is less marked than in cases where the paralysis is the result of disease in the spinal cord: always, according to Dr. Duchenne, the paralyzed muscles, whether atrophied or not, retain their full share of electro-contractility. After death signs of disease are found in the brain, but not in the spinal cord; these signs being increased vascularity, with serous or sero-fibrinous infiltration in the pia mater, in the cortical substance, and in the brain structure generally.

General spinal paralysis is the name used by Dr. Duchenne to describe a form of paralysis which, until he pointed out the differences, was confounded with general paralysis of the insane. Looking hastily at the phenomena of paralysis when clearly developed, it is, indeed, not to be wondered at that these two disorders should have been confounded; but in reality general spinal paralysis, as defined by Dr. Duchenne, possesses peculiarities which are sufficiently characteristic. In general spinal paralysis the mental faculties are natural; in general paralysis of the insane they are fundamentally deranged. In general spinal paralysis the electro-contractility of the paralyzed muscles is abolished or greatly impaired; in general paralysis of the insane it is intact. In general spinal paralysis the paralysis usually begins in the legs and travels upwards, often remaining in the lower parts of the body a long time before attacking the tongue, face, and upper extremities; in general paralysis of the insane all parts of the muscular system would seem to be affected simultaneously, or, if there be any difference as to time, it is the tongue and upper parts of the body which are the first to suffer. In general spinal paralysis there is a marked disposition to atrophy in the paralyzed muscles and elsewhere, to bedsores, and to other signs of defective nutrition; in general paralysis of the insane these evidences of wasting are, to say the least, far less conspicuous. In general spinal paralysis the progress of the disease is slow, often extending over several years; in general paralysis of the insane the whole course of the disease is comprised within three or four years at most. In general spinal paralysis the post-mortem signs of disease are in the spinal cord and not in the brain; in general paralysis of the insane the reverse of this obtains, the cord being healthy and the brain the seat of the disease. Much, no doubt, remains to be done before it is possible to speak positively as to the character of the diseased changes in the cord which are met with in general spinal paralysis; and

at present it must suffice to say, that in one case related by Dr. Duchenne there was softening and injection of the anterior columns in the cervical region of the spinal cord, and that in one case which I had the opportunity of examining there was want of proper consistence, not exactly amounting to actual softening, and a perceptible degree of atrophy, in these columns, throughout the whole of their course from the middle of the neck downwards. Whether general spinal paralysis will prove to have that relation to disease of the anterior columns of the cord which locomotor ataxy has to disease of the posterior columns, remains to be seen.

General spinal paralysis blends, no doubt, with other spinal diseases, and its symptoms vary accordingly; but still it occurs with sufficient frequency in the form described by Dr. Duchenne to deserve the position which he assigns to it as an individual malady. There are also relations equally intimate between general spinal paralysis and cerebral maladies, and I am very much disposed to think that the cases in which the mental powers are obviously weakened will be found to be at least as numerous as those typical cases in which these faculties are natural. At the same time it must be borne in mind that in some cases of general spinal paralysis the mind may seem to be weakened, when in reality it is not so—that in some cases there may be an air of stupidity, or even fatuity, arising from the slow play of the features, the thickness of the speech, the fumbling of the fingers, and like symptoms, which air has its origin in the paralyzed state of the muscles and not in the enfeebled state of "the man behind the mask."

General spinal paralysis cannot be confounded with local Cruveilhier's atrophy or lead palsy, and it must not be confounded with the general forms of these maladies. In general Cruveilhier's atrophy, as well as in local, the atrophy of the muscles is partial, certain muscles being, as it were, dissected out, and others left untouched, capriciously; in general spinal paralysis the atrophy is *en masse*. In general Cruveilhier's atrophy, what remains of muscle obeys the will and reacts with electricity properly—there is no paralysis; in general spinal paralysis there is true paralysis, and the paralyzed muscles have lost much of their electro-contractility. In general lead palsy, also, the history will be sufficient to prevent any confusion as to diagnosis—the paralysis at first electing the extensor muscles of the forearm, the blue line upon the gums, the colic, the constipation, the possibility of lead contamination, and so on.

As regards treatment there is nothing to be said except that it must be conducted upon the same principles as those which apply in analogous cases.

VIII. Hysterical Paraplegia.[1]

Paralysis is certainly entitled to a place among the symptoms of hysteria. Dr. Briquet met with it in 113 out of 430 hysterical patients, its seat being in the four extremities and in the principal muscles of the trunk in 6, in the left arm and leg in 46, in the right arm and leg in 14, in both arms in 5, in the left arm only in 7, in the right arm only in 2, *in both lower limbs in* 18, in the left lower limb in 4, in the feet and hands in 2, in the face in 6, in the larynx in 3, in the diaphragm in 2; and my own experience is more in harmony with these statistics than with the statement of Todd, that the face and tongue escape in hysterical paralysis, that the hemiplegic form of paralysis is less common than the paraplegic, and that "hysterical aphonia" is the form which is most frequently met with.

Hysterical paralysis, so called, is generally met with in persons of a nervous habit of body, and in conjunction with symptoms of an unmistakably hysterical character. As a diagnostic feature, Todd laid stress on a peculiar expression of countenance, which he denominated *facies hysterica*—an expression characterized by a remarkable depth and prominent fulness, with more or less thickness of the upper lip, and by a peculiar drooping of the upper eyelids; and, as it would seem, with good reason. Often, moreover, there is a definite history of symptoms which clearly come within the category of hysterical phenomena—emotional excitability, globus, plentiful gushes of pale urine, and the rest. In diagnosing hysterical paralysis, however, it is not necessary to trust solely, or even chiefly, to evidence such as this, for the paralysis itself is found to have certain features which in themselves are sufficiently distinctive.

Hysterical paralysis is characterized by the paralysis being more or less incomplete, by a marked degree of numbness being associated with it, and chiefly (according to Dr. Duchenne) by the paralyzed muscles, which are not wasted, *having lost their electro-sensibility without losing their electro-contractility*—a loss which, by the way, does not support Sir Benjamin Brodie's opinion that it is the power to will contraction, and not the power of executing the orders of the will, which is at fault in this form of paralysis.

It would also seem to be a peculiarity of hysterical paralysis, as well as of hysterical hyperæsthesia, anæsthesia, and

[1] See also article on Hysteria, p. 630 *et seq.*

clonic convulsion, to affect the *left* side of the body rather than the right. Thus, M. Briquet found pleurodynia nineteen times, hyperæsthesia and anæsthesia five times, clonic convulsion twice, and paralysis thrice as frequent on the left side as on the right side. He found, indeed, a state of things which presents a contrast to what is met with in rheumatism, neuralgia, pleurisy, pneumonia, and other maladies, in all of which it is the right side of the body which is most prone to suffer.

Very frequently, I believe, hysterical paralysis is preceded by symptoms which come under the head of spinal irritation, and not unfrequently, especially when the upper part of the body is affected, it is ushered in by emotional and other symptoms which may at times deserve to be spoken of as an attack of hysteria.

Hysterical paraplegia agrees in its essential features with other forms of hysterical paralysis. The paralysis is usually incomplete. Numbness of the paralyzed parts is a conspicuous phenomenon; as conspicuous, it may be, as the paralysis. The paralyzed muscles have lost their electro-sensibility without losing their electro-contractility. The bladder and bowel (as much apparently for want of proper sensibility as from true paralysis) are little under control, if at all; less so, as a rule, than in common paraplegia. The paralysis is often preceded by symptoms of spinal irritation, in the lumbar region especially, — spinal tenderness, pains about the pelvis and in the legs, irritability of the bladder, and the rest; and now and then it is ushered in by some ordinary hysterical disturbance of one kind or other. And where one leg only is affected, there would seem to be, as Todd pointed out, a gait which is not less characteristic than that which is seen in common hemiplegia. In common hemiplegia the trunk in walking is first of all inclined to the sound side, and the whole weight of the body made to rest upon the sound leg, and then the paralyzed limb is raised from the ground and thrown forwards by swinging it outwardly; the whole series of movements being very like those which are necessary in walking with a wooden leg. In hysterical paralysis, where one leg only is affected, the paralyzed limb, instead of being raised from the ground, as in common hemiplegia, and thrown forward by an outward swing, is dragged directly forward, with the foot trailing on the ground.

The prognosis in hysterical paralysis would always seem to be favorable. Sooner or later, in one way or another, a cure is brought about—most tardily, perhaps, in the paraplegic form of the disorder.

As regards treatment, all that need be said is, that general rules must be followed out, and that, if anything special has to be done, most help will probably be derived from sharp faradization with electrodes which allow the currents to act on the sentient nerves rather than on the muscles—that is, with metal ends rather than with the moistened sponges commonly used. At any rate, sharp practice of this kind has often served to bring about results as sudden and satisfactory as those which have now and then followed the exercise of faith in the power of St. Médard and other kindred agencies.

IX. Reflex Paraplegia.

Paraplegia is one of the consequences of primary disease in the spinal cord: of this there can be no doubt. Paraplegia may also be the result of disorder or disease beginning at a distance and affecting the cord secondarily—beginning in the urinary and genital organs more especially: of this there can be but little doubt. In the former case the paraplegia is spoken of as centric; in the latter as eccentric or *reflex*.

The chief characteristics of that form of reflex paraplegia which is associated with disease of the urinary organs—*urinary paraplegia*, as it is often called—the commonest and most important of all the forms of reflex paraplegia, as it certainly is, are these; or at any rate these are those upon which Dr. Brown-Séquard, who has paid much attention to this subject, insists. Usually the paralysis is incomplete both as to degree and extent, some muscles being obviously more affected by it than others; usually the paralysis is *not* associated either with tingling or numbness, or anæsthesia; usually the bladder and rectum are only slightly implicated in the paralysis; usually there are changes for the better or worse in the degree of paralysis corresponding to changes for the better or worse in the disease of the urinary organs; usually there is no marked atrophy in the paralyzed muscles. Not unfrequently a cure or marked amelioration in the paralytic condition is brought about by the removal of the disease in the urinary organs. Dr. Brown-Séquard indicates these as the chief characteristics of reflex paraplegia connected with disease of the urinary organs, and of other forms of reflex paraplegia as well, the only difference in the description of these latter forms of disease being the substitution for the term "urinary" of the name which indicates the starting-point for the paralysis.

Thus defined, reflex paraplegia differs diametrically from the paraplegia produced by myelitis. In paraplegia from

myelitis the paralysis is usually complete, and all the muscles are affected equally; not so in reflex paraplegia. In paraplegia from myelitis the paralysis is associated with tingling, numbness, or anæsthesia: not so in reflex paraplegia. In paraplegia from myelitis paralysis of the bladder and lower bowel is a marked phenomenon: not so in reflex paraplegia. In paraplegia from myelitis the paralyzed muscles are usually atrophied and degenerated: not so in reflex paraplegia. In paraplegia from myelitis cure, or even improvement, is the exception: in reflex paraplegia it is the rule.

It is, indeed, easy enough to find marked differences between paraplegia from myelitis and reflex paraplegia; but the case is far otherwise when a comparison is instituted between hemiplegia from spinal congestion and reflex paraplegia. In reflex paraplegia the paralysis is not associated with tingling, numbness, or anæsthesia: in paraplegia from spinal congestion it is the same, with the single exception, that there may be at one time or other a trifling degree of tingling at the extreme tips of the fingers or toes. In reflex paraplegia there are fluctuations in the degree of the paralysis; so also in paraplegia from spinal congestion. In reflex paraplegia there is no marked change in the nutrition of the muscles: so also in paralysis from spinal congestion. And, lastly, in reflex paraplegia, as in paraplegia from spinal congestion, a cure is neither an impossible, nor even an improbable, event. As to essential characteristics, indeed, I can find marked differences when reflex paraplegia is compared with paraplegia from myelitis, but none when reflex paraplegia is put in comparison with hemiplegia from spinal congestion.

Nor is reflex paraplegia *always* to be distinguished by being obviously *preceded* by eccentric disorder in the urinary organs or elsewhere. It is, indeed, as Sir W. Gull has well pointed out, "not always easy to determine at this point whether symptoms have a central or a peripheral origin. . . . There is, perhaps, no fact to be more insisted upon than the normal dependence of the sympathetic upon the integrity of the spinal system. As a result of this dependence, we learn that dyspepsia, vomiting, constipation, colic, vesical catarrh, prostatic irritation, pains in the joints, and many other peripheral disturbances, may seem to precede the central malady, and to be the cause of it, when in truth they are its effects." And again: "It is no new fact in medicine, that cerebral exhaustion may impair the functions of the cord (especially of the lower segments), and give rise to precisely those symptoms which have been set down as pathognomonic of urinary paraplegia."

Dr. Brown-Séquard has taken a very different view of reflex paraplegia to that which is here taken. He regards this disorder as due, not to spinal congestion, but to a state of the circulation diametrically opposed to this. He believes that a state of irritation, commencing eccentrically, is propagated along the vaso-motor nerves, of which the result is, primarily, contraction of bloodvessels in, and, secondarily, exclusion of the due amount of blood from, one or more of the three parts following — the spinal cord, the nerves proceeding to or coming from the cord, the muscles. He believes that the proper activity of the nervous tissue or muscle is starved into paralysis for want of blood; and he founds this view on the fact that a state of irritation in the vaso-motor nerves may proceed from a distant point and produce contraction of the vessels, and upon the fact that traces of organic disease are wanting after death in many cases of reflex paraplegia. The argument, indeed, is all but as conclusive as it is masterly and original. The same evidence, however, admits of a very different construction, and that even without anything like special pleading. It is, no doubt, true enough that a state of irritation in vaso-motor nerves may lead to contraction in bloodvessels, and thereby exclude a due amount of blood from the part to which these vessels belong; but it is not less certain that the same state of irritation carried beyond a given degree, either in time or in intensity, may, by paralyzing the vaso-motor nerves, lead to relaxation of vessels, and, thereby, to the admission into them of an undue amount of blood.

Moreover, it may also be assumed, as a thing by no means improbable, that the contraction of the coats of the relaxed and paralyzed vessels in rigor mortis may prevent any marked traces of such vascular engorgement being met with after death; at any rate it is impossible to infer, from the absence of such traces of congestion after death, that there was no such congestion during life. In itself, indeed, the evidence adduced by Dr. Brown-Séquard in favor of his theory of reflex paraplegia is insufficient to decide whether his view or that which I venture to put in opposition to it is the correct one, for in reality it may be used equally in support of either view. And certainly it would seem to be a collateral objection to the view which connects reflex paraplegia with a state of capillary contraction and comparative bloodlessness brought about by irritation in vaso-motor nerves, in states where the whole nervous system is in a state of great irritation, as in tetanus, and in the state specifically designated spinal irritation, and where it may be assumed that the vaso-motor nerves

participate in this state of irritation, and produce vascular contraction and comparative bloodlessness in the spinal cord and elsewhere, that paraplegia or any form of paralysis is precisely the symptom which is not present. Moreover, Sir W. Gull makes some remarks on urinary paraplegia which have an important collateral bearing on the subject in hand, as tending in no ordinary degree to support the conclusion to which all the previous considerations tend: "If," he says, "we regard the nature of the urinary disease which most commonly leads to paraplegia, we shall find that it is an inflammation, either in the prostate, bladder, or kidneys; and we shall also find, that it is only after chronic inflammation has lasted a long time that the paraplegic weakness supervenes. It is in just those cases where there is most irritation, and but little inflammation, that paraplegia does not occur. Uric acid and oxalate of lime calculi may cause hæmaturia and any amount of irritation, but unless *suppurative* inflammation set in, paraplegia is not produced. A review of all the recorded cases of urinary paraplegia will show that it is the *inflammatory* condition of the urinary organs which leads to paralysis, and not one of irritation."

In speaking in this manner, however, I do not wish to confound reflex paraplegia with spinal congestion. On the contrary, the more I see of practice the more I am disposed to think that there is a reflex variety, not only in paraplegia from spinal congestion, but in every form of paraplegia; that, in fact, the causes at work in producing all spinal maladies are reflex in their character as well as centric,—reflex, it may be, rather than centric. [Dr. Brown-Séquard's theory upon this subject finds its only support in a supposition, in regard to vaso-motor excitation and its results, which is more purely hypothetical than almost anything else asserted in modern Physiology. For such cases of paraplegia as are, by exclusion, made out to be truly reflex in origin, the explanation proposed by Handfield Jones may be considered to be the best. This is, that a morbid impression conveyed from an organ (as the kidney) in a state of disorder, to the nerve-centres, may be so overwhelming as to be *inhibitory* of the normal action of the parts innervated by those centres. "Paralysis without apparent lesion," in this sense, has been known since the time of Whytt. Morgagni recorded the occurrence of amaurosis, immediately produced by a blow (from the spur of a cock) upon the eyebrow, wounding the supra-orbital nerve. Handfield Jones reports a case in which strabismus from paralysis of the *rectus oculi externus* disappeared after a piece of dead bone was removed from a whitlow on the thumb. Lawrence cured blindness (in one eye) in a case of thirteen months' standing, by the extraction of a carious tooth, into which a splinter of wood had been forced by an accident. Worms, other causes of intestinal disturbance, and uterine irritation, have also occasionally produced reflex paralysis (not nearly always *paraplegic*), by a similar morbid inhibitory action.—H.]

If the true view of reflex paraplegia be the one which is here taken, it follows that the treatment of that form of this disorder which is defined by Dr. Brown-Séquard will be substantially the same as the treatment of paraplegia from spinal congestion, and not that which has been recommended on the supposition that the spinal cord is starved for want of blood in consequence of its vessels being kept in a state of contraction by irritation of the vaso-motor nerves. Nay, even the necessity to treat eccentric disorder or disease in the urinary organs or elsewhere can scarcely be considered a peculiar feature in the treatment of reflex paraplegia; for, in fact, it is always an essential part of any sound plan of treatment in any disease of the spinal cord, whether originating in the cord or at a distance from the cord, to make a point of doing everything to remove or mitigate any eccentric malady. It is always necessary to do this, because an eccentric malady, whether primary or secondary to the spinal disorder, or whether having no other than a purely accidental relation to this disorder, invariably *reacts* prejudicially upon the cord. This eccentric malady must of course be dealt with on general principles, this thing or that being done according as irritation or inflammation may happen to be the predominating condition. In urinary paraplegia, for example, it is very possible that the local application of opium or belladonna to the urethra, as recommended by Dr. Brown-Séquard, may be of much use; this is very possible on any hypothesis: but with respect to the frequent introduction of catheters, with a view to relieve irritation, I think it is difficult to come to a different conclusion to that which Sir W. Gull has arrived at. "This course," says this able physician, "is not unattended with danger. There is no part of the treatment which calls for more discrimination. The diseased textures and veins about the neck of the bladder are so prone to suppuration, that the catheter is often a fatal weapon. The few scattered instances, such as that recorded by Dr. Graves, where immediate good effects have followed, have had undue influence towards promoting mechanical interference. Carefully considered, they do not warrant the inference drawn from them. If the urinary passages are so contracted

that the bladder cannot empty itself, the catheter is obviously required; but it must be simply prescribed on these grounds. The rule for its use is the same as in the treatment of the aural passages, when the middle ear is diseased. If there be a free exit for the excretions, the less mechanical interference the better. As meddlesome midwifery is bad, so is the meddlesome employment of the catheter in urinary paraplegia. Cases might be quoted where a fatal issue has been induced by the meddlesome interference with a diseased bladder, under the hope of removing some hypothetical cause of reflex irritation."

X. Infantile Paralysis.

This disorder, to which attention seems to have been directed first of all by Underwood, Marshall Hall, and Kennedy, is the *paralysie (dite essentielle) de l'enfance* of several French writers. [*Polio-myelitis Anterior Acuta*, Kussmaul, Erb.[1]—H.] It attacks children indiscriminately, without any regard to sex, between the age of six months and two years, at the time of the first dentition more especially: and it is the grand source of shrivelled, half-dead limbs, club-feet, and other sad deformities.

Mr. William Adams, who has had ample opportunities for becoming practically acquainted with the history of infantile paralysis, and whose account of this disorder is more to the point than any other with which I am acquainted, indicates these as the most trustworthy characteristics: 1. The paralysis is usually partial, single muscles or groups of muscles only being affected. 2. The sensation in the paralyzed parts is usually perfect, or all but perfect. 3. The bladder and lower bowel are usually not distinctly implicated in the paralysis. 4. The paralyzed muscles are at no time rigid. 5. Great improvement or complete recovery is the rule, and not the exception. 6. The paralysis is usually neither accompanied nor preceded by "head symptoms."

The onset of the disorder is generally sudden and unexpected. The child is put to bed well, and in the morning it is found to be paralyzed. Or the paralysis may be grafted upon some marked febrile disorder, as gastric or remittent fever, measles, or typhus; or upon some other malady, as hooping-cough or pneumonia. In some cases there may be transitory and trifling feverishness at first, but fever is certainly no essential accompaniment at any time. Now and then, but only in exceptional cases, the disorder may be ushered in by convulsions or drowsiness.

The paralysis has usually a wider range at first than that which it takes afterwards; in other words, the paralysis is more or less general at first, and more or less localized afterwards. Thus it is a common thing for all the limbs to be attacked and for only one leg to remain paralyzed, or, rather, to remain partially paralyzed, for there is a certain degree of recovery in certain muscles, even in the worst cases. It is the constant rule, indeed, for recovery to be slower in the legs than in the arms, and in certain muscles than in others. Usually the disease does not mount high enough to paralyze muscles whose nerves are given off above the true limits of the spinal cord. There is certainly no loss of sensation in infantile paralysis. On the contrary, as Dr. West remarks in his admirable treatise on the diseases of infancy and childhood, "sensation in the affected limb appears to be exalted when the paralysis is recent, the degree of hyperæsthesia in the early stage being in such cases proportionate to the loss of power which afterwards is apparent." Moreover Dr. West proceeds to say, "In some instances the exaggerated sensibility continues for several weeks, though this is unusual; and when this is the case, the leg being the seat of the affection, and the paralysis incomplete, the existence of hip-joint disease may very likely be suspected. In such a case the child bears all its weight on the healthy limb, turns the foot of the affected side inwards when walking, and stands with the toes of that foot resting on the dorsum of the foot of the healthy side. Still it will usually be found that the exaggerated sensibility of the paralyzed limb varies greatly at different times, while that extreme increase of suffering produced in cases of hip-joint disease on striking the head of the femur against the acetabulum by a blow upon the heel, and the fixed pain in the knee of the affected side, so characteristic of diseases of the hip-joint, are absent; and these points of difference will enable you to distinguish between the two affections. One other important means of diagnosis is furnished by the presence or absence of an increased temperature over the suspected joint, the value of which means in determining the presence or absence of inflammation about any particular spot is dwelt upon by Mr. Hilton in his lectures delivered recently at the College of Surgeons."

The peculiarities of infantile paralysis, so thinks Mr. Adams, point to a special pathology which has yet to be made out satisfactorily. As it seems to me, however, these peculiarities, instead of showing, as Mr. Adams believes, that infantile paralysis is unlike paralysis in adults, only show a close analogy to, if not an actual identity with, the paralysis which has

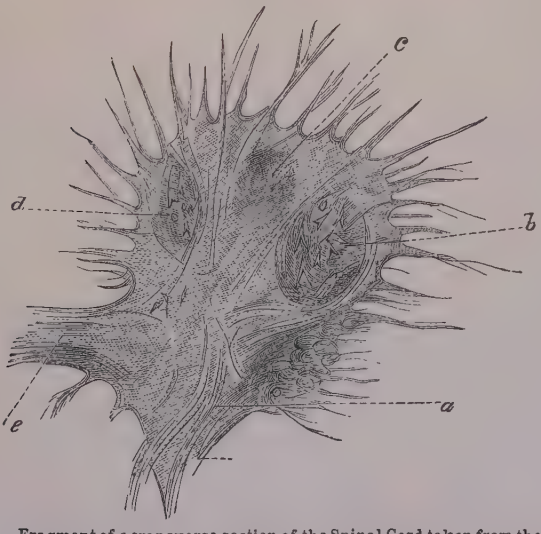
[1 Ziemssen's Cyclopædia of Practice of Medicine, vol. xiii.]

been seen to result from spinal congestion. In infantile paralysis the paralysis is partial: in paralysis from spinal congestion it is the same. In infantile paralysis sensation is exaggerated rather than dulled in paralyzed parts: in paralysis from spinal congestion it is the same. In infantile paralysis the bladder and lower bowel are obedient to the will: so also in paralysis from spinal congestion. In infantile paralysis the paralyzed muscles are limber, not rigid: so also in paralysis from spinal congestion. In infantile paralysis recovery more or less complete is the rule rather than the exception: so also, and very much in the same order, in paralysis from spinal congestion. In infantile paralysis "head symptoms" are exceptional phenomena at any time: so also in the paralysis from spinal congestion. Neither do I know of anything to invalidate the conclusion which those resemblances would seem almost to necessitate—that infantile paralysis, as defined by Mr. Adams, is nothing more than paralysis from spinal congestion.

Moreover, this conclusion is not discredited by the disclosures of morbid anatomy. There were no traces of organic disease either in the spinal cord or brain or nerves in the four cases of genuine infantile paralysis which were examined after death by MM. Barthez and Rilliet, Dr. Fliess, and Mr. Adams, all four most competent observers. The evidence supplied by these cases is indeed purely negative. Nor is evidence more positive to be found in the two cases examined after death by M. Laborde, the writer of a very able treatise on infantile paralysis recently published. In these two cases, without doubt, there were certain organic changes in the spinal cord and in some of its nerves, but these changes are plainly not essential to infantile paralysis as defined alike by M. Laborde and Mr. Adams; for the simple fact is, that the clinical history of these cases is not clearly that of infantile paralysis so defined. In a word, there is nothing in the scanty contributions of the dead-house to show that the very closest relations may not exist between the disorder under consideration and spinal congestion.

[Accumulated observations by Prévost, Vulpian, L. Clarke, Charcot, Joffroy and others, have led to the conclusion, that the most characteristic anatomical changes in this disorder affect the gray matter of the anterior cornua of the cord. While it is probable that congestion only has occurred in those cases whose duration is shortest, and whose progress is most favorable, Charcot and others regard the process as inflammatory in very many cases; commencing in the nerve-cells, and affecting afterwards the neuroglia; later, with atrophic alteration, the motor nerves and muscles. In the anterior cornua, degeneration soon follows inflammation. The large pyramidal cells undergo pigmental change, and shrink away, whole groups of them disappearing in time completely. This atrophy is not always symmetrical; it may be either uniform or in patches, for a considerable distance in the length of the cord.—H.]

[Fig. 61.



Fragment of a transverse section of the Spinal Cord taken from the lumbar region, in a case of Infantile Spinal Paralysis occupying the right inferior extremity. The right anterior cornu of gray matter is represented. The lesions affect exclusively the antero-external group of nerve-cells: *a*, *cervix cornu posterioris*; *b*, postero-external group of nerve cells; *c*, antero-external group. The cells of the latter group have completely disappeared, whereas those of groups *b* and *d* are perfectly distinct; *d*, internal group; *e*, the commissure. (Charcot.)]

The duration of infantile paralysis is very variable. It may pass off in a few days, or even a few hours: it is more likely to occupy several weeks or months in this process of improvement. Improvement, to a greater or less degree, is indeed the rule, and not the exception; and it may even be said that the cases which stop far short of recovery are by no means common. Mr. Adams says, "It is generally supposed that, unless recovery takes place within a few months, the paralysis is persistent through life; but I have seen many cases in which improvement has proceeded, to a very useful extent, several years after the seizure;" and to the truth of this remark my own experience bears ample testimony. Indeed, I should say from what I have seen, that if the paralyzed muscles retain their electro-contractility and electro-sensi-

bility, and so show that they have not passed into that state of fatty degeneration into which they always tend to pass eventually, there appears to be scarcely any limit to the time in which improvement, and even complete recovery, is possible.

The group of muscles most frequently affected in infantile paralysis, according to Mr. Adams, are—1. The muscles of the anterior part of the leg, forming the extensors of the toes and the flexors of the foot; 2. The extensors and supinators of the hand, these muscles being always affected together; and 3. The extensors of the leg, and with them generally the muscles of the foot, as in the first group. When single muscles are affected, the most likely to suffer are these: 1. The extensor longus digitorum of the toes; 2. The tibialis anticus; 3. The deltoid; and 4. The sterno-mastoid.

The deformities produced by infantile paralysis are most frequently met with in the feet and legs, because these are the parts most frequently affected; and the particular kind of deformity varies, of course, with the muscles involved in the paralysis.

"The most frequent kind," says Mr. Adams, "is that of (1) talipes equinus; and the other deformities occur in the following order—(2) equino-varus; (3) equino-valgus; (4) calcaneus, or calcaneo-valgus of one foot is generally found with equino-valgus of the other."

Mr. Adams is of opinion that the great cause of the deformities which are met with in infantile paralysis is the "adapted atrophy" of Sir James Paget, this change taking place chiefly in the opponents of the muscles which have suffered from paralysis. If, for example, the anterior muscles of the leg are paralyzed, the anterior portion of the foot drops, and the heel is raised, not by active contraction of the posterior muscles—for the division or paralysis of one set of muscles does not excite active contraction in the opponent muscles—but in consequence of the position assumed by the foot from its mechanical relations with the leg. Another cause of deformity is obviously atrophy and actual or comparative arrest of development in the paralyzed muscles; for, unless the paralysis soon passes off, it is plain that the muscles will not only waste, but be left behind in the rapid process of development which is everywhere at work in a young and growing child. Mr. Adams is also of opinion that the early and late rigidity of Todd and true spasm have very little to do in causing the deformities in question: and so it may be in the deformities connected with that form of paralysis to which he restricts the term infantile—that form which is undoubtedly the common variety of infantile paralysis, and which, as it would seem, is dependent on spinal congestion. It is very certain, however, that infants and children are liable to more than one form of paralysis, and that there are deformities associated with rigid as well as with flaccid muscles. It is very certain that this rigidity may be either "early or late," as distinguished by Todd, or even still more decidedly spasmodic than that form which is called "early rigidity." In a word, infantile paralysis is a designation as little to be defended as would be the term adult paralysis; for on inquiry it is found that in children, as in adults, there is more than one form of paralysis, and that all the forms which may happen in adults may be repeated in children. The form of paralysis which has been described as infantile is unquestionably the commonest, and the other forms are so uncommon as to be little more than exceptional; and this, in fact, is all that can be said to justify the notion that infantile paralysis is a definite disorder of the spinal cord peculiar to infants.

The treatment of the deformities, especially of club-foot, resulting from the so-called infantile paralysis, is a subject of much practical interest and difficulty. Mr. Adams says: "The probability of benefit in such cases by any surgical procedure seems scarcely ever to be entertained. The existence of paralysis is supposed to contra-indicate any surgical interference; but, from these apparently hopeless and essentially incurable cases some of the most striking and most valuable results of surgery are obtained by a combination of surgical and mechanical treatment. Mechanical aid, alone, is frequently sought from the instrument-maker, but his art is powerless when any considerable amount of deformity exists; and it is only by a scientific combination of surgical and mechanical skill that much good can be effected. In all these cases the treatment essentially consists in the removal of existing deformities by tenotomy and mechanical means, and a subsequent compensation for the existing paralysis by mechanical support, varying in different cases according to the extent of the paralysis." And no doubt very satisfactory results are obtained by those means. At the same time it is certain that in many cases very satisfactory results may be obtained without tenotomy, and without apparatus, by means used with the view of bringing back power into the paralyzed muscles—electricity,[1]

[1] There are certain forms of paralysis in which the paralyzed muscles do not react to the most powerful induced electric currents, but react energetically to a galvanic current of low tension, slowly interrupted (the *labile current* of Remak). The diagnostic and therapeutic bearings of this fact have yet to be

movements of various kinds, shampooings, and others; and my own experience has convinced me that this fact is not yet sufficiently recognized and acted upon in practice. That in many cases neither tenotomy nor apparatus can be dispensed with, I fully believe: that in all cases the electrical and gymnastical parts of the treatment are of primary rather than of merely secondary importance I am every day more and more convinced, because every day I meet with instances of muscles which I should once have looked upon as hopelessly paralyzed being resuscitated by those means. Indeed, I cannot but think that so long as institutions especially set apart for orthopædic purposes are wanting in properly furnished electrical rooms and gymnasiums, there must be in some essential points a necessity for a great reformation in orthopædic practice.

[*Acute Spinal Paralysis of Adults.*—Since 1865, it has been shown by Meyer, Duchenne, Frey, Seguin, Erb, and others, that an affection closely corresponding to Infantile Paralysis occurs sometimes later in life. Beginning rather abruptly, with febrile symptoms, violent headache, sometimes delirium, and vomiting, muscular paralysis soon follows; generally in the lower limbs. Sensation is not impaired; nor is control lost over urination and defecation. In some cases improvement begins in a few weeks, and goes on to complete recovery. Much more frequently, the muscles very slowly, and some of them to the last imperfectly, regain motility. Contraction of the muscles of the limbs may become permanent; although deformities never result, to anything like the extent observed in children. This form of attack does not appear to be dangerous to life; but its effects seldom entirely pass away. Cases which may be correctly called transitory or temporary are rare.[1]—H.]

worked out, but so far the therapeutic promise is good. The phenomenon in question has been already observed in several very different cases—in facial palsy (first noted by Baierlacher), in certain cases of infantile paralysis (discovered by J. Netten Radcliffe, of London, and Hammond, of New York, independently of each other), in certain cases of local palsy, *e. g.*, palsy of the extensors of the forearm and of other muscles, from lead-poisoning (Bruckner and J. N. Radcliffe), in paralysis of the deltoid, not from lead (J. N. Radcliffe), in certain cases of muscular atrophy (J. N. Radcliffe), and in paralysis from traumatic injury of a nerve (Bruckner).

[[1] Amongst the cases of this disease reported in America, may be mentioned those of Seguin, forty-five cases (Myelitis of the Anterior Horns, New York, 1877); two recorded by Althaus (Amer. Journal of Med. Sciences, April, 1878); and five by W. Sinkler (Am. Journal of Med. Sciences, October, 1878).]

XI. Spinal Hemorrhage.

Blood may be effused into the substance of the cord between the arachnoid and pia mater, into the sac of the arachnoid, between the dura mater and arachnoid, or between the dura mater and the osseous canal—anywhere in or about the spinal cord, in fact. Hemorrhage in the substance of the cord, the *hæmatomyélie* of Ollivier, may be a consequence of myelitis, the bloodvessels breaking up in the softening of the cord, and so allowing the blood to escape. It was so in the acute case which I took as my text when speaking of myelitis, for here the blood was collected at one point in the softened nerve matter to an extent which at first sight suggested the idea of hemorrhage into the cord rather than that of myelitis. Hemorrhage under or upon the spinal membranes, the *hæmatorachis* of Ollivier, may be a consequence of cerebral hemorrhage, the blood overflowing from the cranial into the spinal cavity, and perhaps mixing with the spinal fluid; or it may result from spinal congestion, spinal meningitis, myelitis, tetanus, hydrophobia, and certain other maladies. All these cases, however, are so uncommon as to be little more than exceptional. In fact, hemorrhage either into the substance of the cord, or under or above the spinal membranes—except as the result of some accidental injury to the spine, as in death by hanging, or in cases of still-birth where it has been necessary to employ much force to bring about the delivery—is, to say the least, a very uncommon affection.

The symptoms of spinal hemorrhage are by no means clearly marked. Sudden and acute pain in the spine at the seat of the effusion, and sudden paralysis and loss of sensation, more or less complete, in the parts below this point, appear to be the chief symptoms where extensive hemorrhage has taken place into the substance of the cord. Sudden and acute pain in the spine would also seem to be a prominent symptom in hemorrhage below or above the spinal membranes, but not sudden paralysis and anæsthesia. In this latter case, indeed, instead of paralysis there have been some convulsive or spasmodic symptoms, and instead of anæsthesia some hyperæsthesia. In some cases, as in one quoted by Dr. Copland, the pain may be not in the back, but at a distance from the back; and in other cases, and this not unfrequently, pain may be greatly masked by the shock of the accident which has caused the hemorrhage, or by the shock attendant upon the laceration of the spinal cord by the effused blood. When the hemorrhage is in the medulla oblongata, and high up in the cord, the symptoms may be rather like those of epilepsy than

anything else—loss of consciousness, convulsion more or less general, choking noises, and the rest—and this equally whether the blood is effused into the substance of the cord or around it: and this fact suggests the possibility, to say the least, that the convulsive or spasmodic symptoms, which have by some writers (on what to me seem to be insufficient grounds) been supposed to distinguish hemorrhage under or above the spinal membranes from hemorrhage into the substance of the cord, may in reality be due to irritation transmitted to the medulla oblongata and upper part of the cord, and not to irritation acting upon the membrane or membranes. Moreover, when the hemorrhage is high up in the cord, priapism and distress of breathing are found to figure conspicuously among the symptoms, as they do also in other cases where this part of the cord is damaged by disease or injury. In a few instances, the symptoms of spinal hemorrhage are preceded by symptoms indicative of spinal congestion, or inflammation, or irritation.

Remains of old apoplectic cysts, similar to those so often found in the brain, have been met with in the spinal cord, even in the medulla oblongata and upper part of the cervical region; but these signs of partial recovery are, to say the least, exceedingly exceptional. Indeed the mischief done by the hemorrhage is generally not only irreparable, but very speedily fatal, and that too in spite of everything that can be done to promote recovery.

XII. Non-inflammatory Softening.

Two well-marked varieties of softening of the spinal cord are detected by the naked eye—the red and the white. In both varieties the microscope brings to light broken down nerve-tissue mixed up with a number of bodies known as *granule masses*—large bodies, whose principal constituent is fat, black-looking, from not transmitting light, and somewhat like mulberries, from being built of a number of round bodies or granules. "It was once thought," said Dr. Wilks, "that these masses denoted inflammation. But you find them in any degenerating part, as a decaying strumous gland, or a cancerous tumor, or a phthisical lung: and the question of their formation in the brain or cord is not yet answered; whether they originate in inflammatory cells, or are the natural cells of the nerve-structure degenerated. In some you may still see a wall and a nucleus, which points to the former opinion as the more correct." The red variety of softening is often in parts yellow rather than red: the redness being due to increased vascularity or effused blood-corpuscles, one or both; the yellowness to the presence of fibrillated tissue, nucleated fibre, pus-corpuscles, or some other form of distinctly inflammatory product. In a word, there can be no doubt of the inflammatory origin of the red variety of softening. In the white variety of softening, on the other hand, there are generally an atheromatous state of the vessels and other signs of true degeneration, the vascularity is evidently diminished, and there is an absence of those distinctly inflammatory products which have just been enumerated. It would seem, indeed, that the white variety of softening differs essentially from the red, in that, instead of being the result of inflammation, it is brought about by the parts being starved and atrophied for want of blood. With respect to the reality of these differences between these two varieties of softening there appears to be little or no reason for doubt: at the same time it must not be forgotten that it is not always easy to draw the line between these two varieties, and that they both may exist together in the same cord.

The symptoms of non-inflammatory softening would seem to be identical with those of the more chronic forms of myelitis. The more tardy the development of these symptoms, and the older the patient in years or in constitution, the more likely is the case to be one of non-inflammatory softening: and this is all that can be said in the matter of diagnosis. Practically, however, this want of definiteness is of no moment; for in the chronic form of myelitis the degenerative process has more to do in bringing about the diseased changes in the cord than the inflammatory, and more to do also in supplying the indications for treatment. Nay, it may even be held that the same remark applies to some extent to the more acute forms of myelitis as well as to the more chronic, for it is with the ruin rapidly produced by the inflammation rather than with the inflammation itself that the practitioner in medicine has to cope almost, if not altogether, from the very onset of the disease.

XIII. Induration.

Like the opposite condition of softening, induration (sclerosis) of the spinal cord is one of the consequences of myelitis, chronic or acute, of the chronic form more especially. Induration of the cord is generally associated with atrophy—atrophy often more marked in the white matter than in the gray—and with a condition so curiously bloodless that a section is not unlike that of white of egg boiled hard. In its highest degree the cord may have a leather-like or fibro-cartilaginous hardness and consistency. Induration is a

much less common change than softening: it has no symptoms by which it can be distinguished from softening: and it is often met with when it was not expected, and under very different circumstances, as after acute myelitis on the one hand, or after long-standing epileptic disease on the other.

[In recent neuro-pathology, *sclerosis* has a much larger place than it had a few years ago. By the researches especially of Vulpian, Charcot, Ordenstein, Frerichs, Rindfleisch, Türck, Frommann, Westphal, Erb, Gull, Lockhart Clarke, Moxon, Seguin, and Hamilton, it has now been more exhaustively studied than almost any other morbid affection of the nervous apparatus.

From the nature of the diseases in connection with which it has been observed, whose progress is ordinarily slow and protracted, the actual primary character of the *process* of which it is the result is known, almost alone, by inference and analogy. While very many autopsies of sclerosis have been made at the end of maladies of several years' duration, it has been only cases in which life has been shortened by intercurrent attacks of other kinds that have given opportunities for examining the morbid changes going on in the same maladies in their earlier stages. It is, however, the common opinion of pathologists, that *inflammation* has generally to do with the beginnings of sclerosis of the brain or spinal marrow, very much as it has with the commencement of cirrhosis of the liver, lung, or other organs. Some writers make the term sclerosis synonymous with *chronic inflammation* (Bristowe).

At all events, the later and more important alterations are atrophic and degenerative. The nervous elements, cells, or fibres, according to the part involved, waste away, and in their place the connective tissue (neuroglia) in embryonic form undergoes increase; with fibroid, amyloid, and pigmentary degeneration, in various degrees, proportions, and extent.

In progressive muscular atrophy and general spinal paralysis, sclerosis has been found in the anterior horns of the gray matter of the cord. In unfavorable cases of infantile paralysis, and the similar spinal paralysis of adults, the same morbid change is believed, upon good evidence, to occur. Locomotor ataxy is now known to have for its characteristic lesion sclerosis of the posterior white columns of the cord. Lateral sclerosis of the cord gives rise to the symptoms of progressive spasmodic paralysis; a like affection of the motor nuclei in the medulla oblongata, is connected with glosso-labio-laryngeal paralysis; and multiple or disseminated sclerosis is the name now given, on an anatomical basis, to a disease whose clinical phenomena vary according to the seats of the disease in different parts of the brain, medulla oblongata, and spinal marrow. It seems appropriate, in this place, to give some attention to such of these disorders as are not otherwise treated of in this work, viz., lateral spinal sclerosis; sclerosis of the medulla oblongata; and multiple or disseminated sclerosis.

Lateral Spinal Sclerosis.—This has been found anatomically associated with at least two affections: one primary, in the cord, and the other secondary, following cerebral hemorrhage, or softening; or a similar lesion of the crura cerebri, medulla oblongata, pons, or of the spinal marrow itself.

The latter of these forms (secondary) is apt to be unilateral (like the lesion which it follows), as a descending sclerosis. It occurs on the side opposite to the primary lesion, and can be traced upwards through the decussation to its site. In its descent, the sclerosis becomes more and more limited, so as, in the lumbar part of the cord, to include often but a small portion of the lateral column. The gray sclerotic patches in this form are rounded when seen in transverse section, and do not reach the pia mater exteriorly. This is the case in "Türck's degeneration," following cerebral hemorrhage, and also, generally, in sclerosis descending from inflammation or hemorrhage of the cord. In the latter instance, particularly, the sclerotic change stops short of the posterior cornu, being separated therefrom by a thin layer of healthy white nervous tissue.

In idiopathic or primary lateral sclerosis, the characteristic lesion is symmetrical upon the two sides of the cord. It occurs at first and chiefly in the crossed pyramidal columns of Flechsig; posterior to the lateral columns proper, but anterior to the direct cerebellar columns. Extension may take place, however, horizontally, so as to include the whole of the posterior half of the lateral column, reaching to the posterior cornua. The form of the sclerotic patches is then wedge-like or triangular in transverse section. The appearance of these patches is much the same as that observed in disseminated or multiple sclerosis, of which a more minute account will be given presently.

Primary lateral sclerosis (O. Berger) corresponds most nearly with the *tabes dorsalis spasmodica* of Charcot, or *spastic spinal paralysis* of Erb; *tetanoid paraplegia* of Seguin. When the disease extends so far as to involve the anterior cornua, and is attended by wasting of some of the muscles, it meets the description of Charcot's "sclérose latérale amyotrophique." Both forms usually commence between the thirtieth and the fiftieth years of life.

Symptoms of the early stage of lateral sclerosis are mainly those of muscular weakness and *paresis;* that is, diminution of motor power short of actual paralysis. Gradually, however, a truly paralytic condition is developed. The limbs at first drag heavily; but before becoming paralyzed, signs of irritation of the motor centres occur, in spasmodic symptoms. These are, twitchings of the legs, when at rest, especially after exertion; trembling of the limbs when, in sitting, the toes are made to touch the floor; and an increasing tension of the muscles, producing a peculiar gait in walking. The legs are held close together, the knees bend forward, the patient rises at each step on his toes, with a sort of hopping motion, so that he is constantly in danger of falling forward.

Fig. 62.

A. Antero-lateral Sclerosis. B. Posterior Spinal Sclerosis. (After Charcot.)

At this stage, there is a marked increase in the tendon-reflex (see Locomotor Ataxy, in this volume) of the knee and other parts. This phenomenon can, in patients having lateral sclerosis, be readily shown with the triceps and biceps muscles of the arm, the biceps flexor femoris, and other muscles. The foot-clonus also, induced by tapping the tendo Achillis while the foot is held in a flexed position, is exaggerated in the same cases.

No disturbance of sensibility or intelligence exists, nor muscular atrophy; and no troubles affecting the bladder, rectum, or sexual organs. There is not, as in the subjects of locomotor ataxy, a disposition to throw out the limbs in walking, nor any inability to stand erect with the eyes shut or in the dark.

Slowly the disease advances, to complete paralysis; first of the lower and then of the upper limbs; with, also, rigid contractions of their muscles. At last the patient is unable to walk or stand; yet the vegetative functions of the body are commonly unimpaired. In such a condition he may continue to live for many years, death finally resulting from some other disease.

As already said, should the sclerotic change extend as far as the anterior cornua of the cord, muscular atrophy is added to the above symptoms; giving the features of the "sclérose latérale amyotrophique" of Charcot.

Recovery from spasmodic spinal paralysis is not to be expected. Charcot doubts its possibility in any case. Westphal has reported one instance of the disappearance of the disease, after all its symptoms had been progressively developed. Dr. R. von der Velden has recorded[1] the particulars of a case occurring in Kussmaul's Clinic at Strasburg, in which, after a rather acute onset, entire recovery finally resulted.

Sclerosis of the Medulla Oblongata is the anatomical designation of what was first described by Duchenne as muscular paralysis of the tongue, soft palate, and lips; the glosso-labio-laryngeal paralysis of Trousseau; the progressive bulbar paralysis of Nachsmuth; or, more descriptively yet, progressive bulbo-nuclear paralysis of Kussmaul.[2] It has been most generally referred to in medical works by the name proposed by Trousseau, upon clinical grounds; *glosso-labio-laryngeal paralysis.* If the anatomical basis of nomenclature continues to obtain the preference of pathologists for the group of diseases now under consideration, *bulbo-nuclear sclerosis* would seem to be most consistent with the other titles which have been named.

Amongst chronic diseases, there is none more terrible in its course than this. Beginning mostly after the age of forty, generally in males, its first symptoms are those of pain and oppression in the neck and back of the head, with dizziness and hesitation in speech. The tongue becomes more and more clumsy in its action, both in articulation and in mastication. Weakness of the lips soon increases the difficulty felt in speaking. The mouth falls, giving

[1 Berliner Klinische Wochenschrift. Sept. 23, 1878.]

[2 Erb, in Ziemssen's Cyclopædia, vol. xiii.]

a lugubrious expression to the face. Swallowing also is impeded, by enfeeblement of the muscles of the soft palate; and, later, those of the pharynx and epiglottis. In the act of deglutition, food, and especially liquids, at times may escape into the larynx, producing suffocation. The voice is altered, growing more and more indistinct, and attacks of dyspnœa are frequent. At the same time sensibility and intelligence are unaffected. Month after month, the disability of speech, mastication, swallowing, and respiration grows worse, until it becomes almost impossible for any nourishment or drink to be swallowed. At last, unless anticipated by some intercurrent attack, as, for example, of pneumonia, death will ensue from starvation.

Not unfrequently, coincident with the above symptoms, progressive atrophy may be observed in a number of the muscles; it is first, and especially, noticeable, in the smaller muscles of the hand.

After death, sometimes the unaided eye can perceive no morbid change in the medulla oblongata. In many cases, however, diminution in bulk and alteration of consistence, with gray discoloration in spots or patches, may be seen. Microscopic examination has shown the characteristic change to be a yellow pigmentary degeneration of the nuclei of the hypoglossal, spinal accessory, vagus, and facial nerves. The nerves and nerve-roots themselves partake of this atrophy and degeneration. Allied changes, especially fatty atrophy, are found to have taken place in the muscles supplied by these nerves; those especially of the tongue, lips, palate, and lower portion of the face. It appears to be at the origin of those filaments of the facial nerve which are distributed in its inferior branches only, that the lesion affecting that nerve occurs. Dribbling of the saliva is an almost constant symptom at a late stage of the disorder, from paralysis of the lips, tongue, palate, and pharynx; without any ascertained increase in the salivary secretion itself.

The marked atrophy of many muscles in this affection has led several pathologists (Kussmaul, Hallopeau, Charcot, Erb) to assert its essential identity with *progressive muscular atrophy*. Others (Duchenne, Friedreich, Hammond) oppose this view. At all events, while the anatomical appearances observed after death do not greatly differ from those of sclerosis of other parts of the cerebro-nervous axis, the evidence that inflammation has any important share in its pathology is very imperfect. Atrophic degeneration of the motor nuclei of the medulla is its characteristic lesion; as a similar change of other motor nuclei coincides with ordinary progressive muscular atrophy.

From one to five years is the common period of duration of this disease; from which, when clearly established, there appears to be no prospect of recovery.

Multiple Cerebro-spinal Sclerosis.—This is the "sclérose en plaques disséminées" of Charcot; the insular sclerosis of Moxon. Although disseminated sclerosis may occur in the brain alone, or in the spinal cord only, yet such a limitation is exceptional; and the typical history of the disease can be best studied by giving attention to cases in which both brain and spinal cord are affected.

Charcot (to whom is accorded the credit of having made the most valuable contributions to our knowledge of this affection) asserts that the first mention of it occurs in Cruveilhier's "Atlas d'Anatomie Pathologique," 1835–1842. Carswell, Türck, Rokitansky, Frerichs, and others subsequently added similar observations. But the systematic attachment of definite sclerotic lesions of the cerebro-spinal axis to the clinical symptoms now understood to belong to them, dates from the records and studies of Vulpian, Charcot, and Bouchard of cases at La Salpêtrière, from 1862.

Multiple sclerosis is most frequent in women, between the ages of twenty and thirty; it is not unknown, however, in quite young persons. Hereditary predisposition appears to be more marked in connection with it than with either of the other sclerotic affections of the nervous apparatus. Like them, it is obscure in its immediate causation. Exposure to cold and wet, and powerfully disturbing mental or moral influences, are believed to have the most to do with its production. Excessive exertion, of mind or body, injuries of the head, or shock from concussion of the whole body, pregnancy,

Fig. 63.

Lesions observed on a section taken from the uppermost portion of the lumbar region, in Disseminated Sclerosis. The posterior columns are invaded throughout their breadth, and the lesion predominates in their middle region. (Charcot.)

and acute diseases, as typhus, cholera, smallpox, have all been supposed to promote or determine its occurrence.

When the brain and spinal cord of a patient who has died from multiple sclerosis are examined with the naked eye, numerous grayish, or yellowish-gray, spots or patches are seen, irregularly distributed (plaques disséminées). They are almost translucent, somewhat of the appearance, in this respect, of cartilage; clearly defined from the surrounding healthy tissue. Sometimes they are slightly elevated above the surface; in other instances level with it, or sunken a little below it. They are roundish, but irregular and various in form and mode of dissemination; not symmetrical upon the two sides of the body. When exposed to the air, they become somewhat rose- or salmon-colored. In consistency, these *plaques* are firm enough to be felt distinctly by the finger. To the knife they present much more resistance than the normal nervous tissue of the brain and cord. On division, they show a clean smooth surface, giving out a small quantity of transparent liquid. In a few instances, in which death from other causes allowed examination at an early stage, they have been found (Zenker) soft, gelatinous, semi-fluid in consistence. In number they vary indefinitely in different cases. Their size also varies from microscopic minuteness up to the magnitude of a hazel-nut. In the cord they may extend longitudinally, in the same column, to a distance of from one to three or four inches.

The cortical part of the cerebrum seldom contains any sclerotic patches. Its white substance, the walls of the ventricles, the corpus callosum, pons, medulla oblongata, and cerebral peduncles, all may have numerous spots of sclerosis. In the cerebellum they are commonly fewer, and absent from its exterior surface.

In regard to the appearances observed after death from this affection, with the aid of the microscope, similar as they are to those of other forms or types of sclerosis (varying chiefly in location), our readers will profit most by the description given by Charcot.[1]

"Microscopical examination, even when a low power is used, enables us to state that the apparently healthy region bordering the sclerosed patch really presents, to a certain width, very plain traces of alteration. When you pass the apparent limit of the sound parts the lesions become more marked, and they augment gradually in intensity as you approach the centre of the patch, where they acquire their highest degree of development. Whilst proceeding thus, from the circumference to the centre, we are led to recognize the existence of several concentric zones, which answer to the principal phases of the alteration.[1]

Fig. 64.

Represents a fresh preparation, taken from the centre of a patch of Sclerosis, colored with carmine, and dilacerated. In the centre is seen a capillary vessel, supporting several nuclei. To the right and left of this are axis-cylinders, some voluminous, others of very small diameter, and all deprived of their medullary sheaths. The capillary vessel and the axis-cylinders were vividly colored by the carmine; the axis-cylinders present perfectly smooth borders, without ramification. Between them are seen slender fibrillæ of recent formation, which form on the left and in the centre a sort of network resulting from the entanglement or anastomosis of the fibrils. These are distinguished from the axis-cylinders, 1st, by their diameter, which is much smaller; 2d, by the ramifications which they present in their course; 3d, by taking no coloration from carmine. Nuclei are seen scattered about; some of them appear to be in connection with the connective fibrils; others have assumed an irregular form, owing to the action of the ammoniacal solution of carmine.

a. "In the *peripheral zone* the following appearances are observed: The trabeculæ of the reticulum are markedly thickened; sometimes they have acquired a diameter twice as great as that possessed in the normal state. At the same time, the nuclei which occupy the nodes of the reticulum have become more voluminous; they are occasionally found to have multiplied, and you may count two or three nuclei, rarely more, in each node;[2] the cellular form becomes more distinct, owing to the thickening of the trabeculæ; the nerve-tubes appear to be further apart each from each—in reality, they have chiefly diminished in volume, and this kind of atrophy goes on at the expense of

[1 Lectures on Diseases of the Nervous System, Lect. VI.]

[1 Charcot, "Société de Biologie," 1868.]

[2 Occasionally some of these nuclei present towards their middle region an indentation which seems to indicate the beginning of scission.]

the medullary sheath, for the axis-cylinder has preserved its normal diameter, or it may even be hypertrophied. The amorphous matter which surrounds the fibres of the reticulum, on all sides, appears to be more abundant than in the healthy state.[1]

Fig. 65.

Patch of sclerosis in the fresh state: *a*, lymphatic sheath of a vessel distended by voluminous fatty globules; *b*, a vessel divided transversely. The adventitious coat is separated from the lymphatic sheath by a free space, the fatty globules which distended the sheath having disappeared; *cc*, fatty globules, gathered into small groups, dispersed here and there over the preparation.

b. "The nerve-tubes in the *second zone*, which may also be called the *transition zone*, have become still more slender. Many of them seem to have disappeared; in reality, they have been merely deprived of their medullary sheaths, and are now only represented by their axis cylinders, which, indeed, sometimes acquire comparatively colossal dimensions.[2] As to the trabeculæ of the reticulum, these offer not less remarkable alterations. They have become more transparent, their outlines are less distinct; finally, in certain parts, and this is a really fundamental fact, they are replaced by bundles of long and slender *fibrils*, closely analogous to those which characterize common connective tissue (laminous tissue). These fibrils are disposed in a direction parallel to the greater axis of the nerve-tubes; hence but little of them is seen in transverse sections, except their extremities, which present the appearance of a multitude of very fine dots. They tend, we have said, to usurp the place of the fibres or trabeculæ of the reticulum; but they, also, invade the meshes which contain the nerve-tubes, according as these diminish in size by loss of medullary matter, so that the reticulated or alveolar appearance which the connective gangue or matrix shows so distinctly in the healthy state, tends to become more and more effaced.[1]

c. "The *central region* of the sclerosed patch, you are aware, is that in which the most marked alterations are observed. Here all vestige of fibroid reticulum has disappeared; we no longer meet with distinct trabeculæ or cell-forms; the nuclei are less numerous and less voluminous than in the external zones; they are shrunken in every direction, appear shrivelled, and do not take so deep a tint as usual under the action of carmine.[2] They may be observed forming little groups here and there in the interspaces between the bundles of fibrillæ. The latter, however, have invaded every part. They now fill up the alveolar spaces, from which the medullary matter has completely disappeared. Nevertheless, a certain number of axis-cylinders, those last vestiges of the nerve-tubes, still persist in the midst of the fibrils; but they, in general, no longer retain that comparatively large volume they accasionally possessed in the early phases of the alteration; most of them, indeed, have even diminished to such a degree that they might be mistaken, so close is the resemblance, for the fibril filaments of new formation, from which, however, we shall soon learn how to discriminate them."

"I cannot pass over in silence the different alterations which those bloodvessels undergo that traverse the nodules of sclerosis. These changes may be well studied in the longitudinal sections of the cord, hardened by chromic acid. At the commencement, that is to say, in the peripheral zone, the parietes of these vessels, even of the finest capillaries, appear much thickened, and contain a larger number of nuclei than in the normal state. Nearer the centre of the nodule the nuclei are still more abundant, and, besides, the adventitious coat is replaced by several layers of fibrils quite similar to those which are simultaneously developed in the substance of the reticulum.[3] Lastly, at the final term of alteration, the walls of the vessels have become so thickened that their calibre suffers a notable diminution.[4]

"I should notice, in passing, the habit-

[1 Frommann, 2 Theil, Pl. ii. figs. 1, and *passim.*]

[2 Frommann, Charcot.]

[1 Frommann, 2 Theil, loc. cit., P. iv. figs. 1, 2, and 3.]

[2 Frommann, Charcot.]

[3 Vulpian, "Cours de la Faculté."]

[4 Frommann, loc. cit.]

ual presence of a certain number of amyloid corpuscles in the midst of the fibrillary tissue. But I should at the same time mention the singular fact that these bodies are always less abundant in disseminated sclerosis than in the other varieties of gray induration."

The *symptomatology* of multiple sclerosis is a variable composite, so to speak, of that belonging to sclerosis of the different portions of the cerebro-nervous apparatus. Generally, the first signs of the disorder are obscure. Weakness and dragging of the lower limbs, difficulty in using the hands, in writing or otherwise, pains in various parts of the body, muscular rigidity, and sometimes giddiness and headache, are among the earlier indications of cerebro-nervous disorder. There are cases in which, later, genuine *ataxia* is present, with all the characters of locomotor ataxy. In such instances, the posterior white columns of the cord have been invaded. Other examples exist in which the features of spasmodic spinal paralysis are equally well marked; with a corresponding localization of the lesion; and the same is true of bulbo-nuclear paralysis, as it has been above described.

These associations of symptoms with special localities of morbid change having been anticipated in cases of multiple cerebro-spinal sclerosis on the ground of clinical indications, have been repeatedly confirmed by post-mortem examination. The disease is truly polymorphous. Yet, since in most cases the dissemination of the morbid process involves several centres of innervation, a combination of resulting symptoms is observed in typical cases. Of such, the following features are most prominent:—

Tremor accompanying all voluntary movements; impairment of speech, with monotony of voice and a "scanning" articulation; nystagmus (movement of the eyeballs from side to side); amblyopia (feebleness or indistinctness, without total loss, of vision); diplopia (double vision); vertigo; and apoplectiform attacks. The last named, however (attacks resembling apoplexy), according to Charcot, are met with only in about one-fifth of the cases observed.

Of the volitional tremor, it is to be noticed that it differs in a marked manner from that of paralysis agitans. In the latter, trembling exists while the patient is at rest, even when lying upon his back; while in multiple sclerosis, it is absent except when an effort is made with some of the muscles; it then at once begins. The head is usually involved in the shaking of multiple sclerosis; almost never in that of shaking palsy. The tremor is smaller, finer, in its vibrations in the latter than in the former complaint. In chorea, again, the erratic movements are more irregular and excursive, having less relation to the intended direction of movement, than in multiple sclerosis.

The explanation of the tremor in this disease has been considerably debated. Charcot refers it to the persistence of many of the axis-cylinders of the spinal nerve-fibres, giving rise to an imperfect and jerking conduction of the impulses of the will to the muscles. Erb and Ordenstein object to this view; and urge the greater probability of the tremor being due to the localization of the sclerotic lesion in some part of the brain. Ordenstein asserts the pons, and the portion of the brain anterior to it, to be the seat of this lesion. Hammond reports that tremor is absent in cases of spinal, without cerebral, disseminated sclerosis. Ebstein, Vulpian, and Engesse have observed spinal cases without trembling; and Kelp, one case entirely cerebral, in which tremor was present. Erb[1] examined twenty-two cases after death with a view to determining this question. In all of them which had presented tremor during life, the pons, medulla oblongata, and pedunculi, besides other portions of the brain, were particularly involved; while in the few cases which had been without tremor, although there were nodules elsewhere in the brain, there were none, or those of small size only, in the pons, medulla oblongata, cerebellum, &c.

Apoplectiform attacks, occurring in cases of multiple sclerosis, may be diagnosticated from true apoplexy, by the high temperature (104°–105⅘° F.) they present; by the gradual approach and deepening of the coma; and by the transitory nature of the hemiplegia which follows them. The patient may remain unconscious for a day or two; after the return of consciousness he falls asleep, and awakens in his usual condition of health, except the persistence of hemiplegia for a few days longer. Such attacks may be repeated at variable intervals, usually of a few or several months. Not unfrequently, however, the patient dies in the comatose state.

Ordinarily, the duration of cases of multiple sclerosis is from five to ten years. Remissions of many of the symptoms are not uncommon; but they seldom last long. Vulpian has given an account of one case, in which an attack of varioloid was followed by the disappearance of all the symptoms; but they returned after an interval of three years.

Upon the *treatment* of this and all other forms of sclerosis of the brain and spinal marrow, nothing satisfactory can as yet be said. Many remedies have been exten-

[1] Ziemssen's Cyclopædia, vol. xiii.]

sively tried, with only occasional, doubtful or transitory benefit.

Formerly, *counter-irritation* was much relied on; by blisters, issues, setons, and moxas applied to the back. Under the reaction caused by their frequent failure to produce cures, such measures are now too much neglected. If it be true that the pathological state preceding or inaugurating sclerosis is one of *inflammation*, powerful derivants applied to the spine ought to be serviceable at that period. For milder effect, nothing is more convenient than croton oil, rubbed (a few drops) over a limited surface, to produce an artificial eruption. Painting with pure tincture of iodine will also produce moderate irritation. For the most vigorous action of this kind, the moxa may be employed.

Among the medicines used in treatment of sclerosis, have been nitrate of silver, arsenic, chloride of gold, ergot, phosphorus (or phosphide of zinc), strychnia, belladonna, iodide and bromide of potassium, and chloride of barium. Charcot, Erb, and Schüle have reported temporary improvement after the use of cold water (hydropathic) treatment. Nitrate of silver, and hypodermic arsenical injections, have each had like transient influence only. Dr. Da Costa recommends, and in early stages of the disease has seen marked benefit from, the use of corrosive sublimate; given internally, for months together.[1]

Electricity continues to be the last resort of therapeutics in such cases. The continuous galvanic current affords, in them, the greatest hope of possible advantage.

Massage, or systematic kneading of the muscles of the trunk and limbs, may be useful, especially if combined with inunction of olive or cocoa oil.[2]

Palliative remedial treatment, according to the symptoms, may often be of much importance to the patient. In lateral spinal sclerosis (spastic spinal paralysis), for example, Dr. S. Weir Mitchell[3] recommends, for the relief of suffering, hypodermic injections of morphia, alone or with atropia. The same or similar anodyne treatment is also often appropriate for the mitigation of the distress of those affected with the severe pains of locomotor ataxy; or, most of all, the horrors of glosso-labio-laryngeal paralysis (bulbo-nuclear sclerosis). Erb, in regard to the late stages of this last affection, remarks that "to close the tragic course, the physician might well think of euthanasia."[1]

Hygienic measures will, in all such cases, afford means of valuable aid on the part of the medical adviser, especially in the earlier and middle stages. Proper regulation of the diet; sufficient warmth of clothing, and protection otherwise from exposure; choice of climate for residence; avoidance of, or extreme moderation in, sexual indulgence; occupation, rest, mental and moral surroundings and influences: all these may not only make a difference of months or years in the duration of prolonged attacks, but may lessen very greatly the sufferings of those to whom recovery is made impossible by the nature of their disease.—H.]

[1 N. Y. Medical Record, April 5, 1879, p. 314.]

[2 Unfortunately, professional *rubbers* are apt to lack discretion in their performance, and to weary their patients by too violent or long-continued manipulation. Such excess should be prevented, if possible, by the medical attendant.—H.]

[3 N. Y. Med. Record, June 28, 1879, p. 605.]

XIV. Atrophy and Hypertrophy.

Atrophy of the spinal cord, like atrophy of the brain, is one of the changes which must be looked upon as natural to old age. In elderly persons, indeed, the cord becomes shorter and narrower and firmer, the spinal fluid increases in quantity, so as to fill the space left vacant by the shrunken cord, and the spinal nerves are sensibly wasted at both their roots. All this has been abundantly proved by Chaussard, Ollivier, and others. Atrophy, more or less general, is also associated with many forms of paralysis in which the cord has been long left in a state of comparative functional inactivity; and local atrophy is one of the consequences of tumor, displaced vertebræ, or anything which exercises pressure upon the cord. Of partial forms of atrophy resulting from disease, the only one about which there is any certain knowledge is that which is associated with the disease called locomotor ataxy—namely, atrophy of the posterior columns; and about this form enough has already been said in a separate article.

In a few instances the spinal cord has been found to be so much enlarged, apparently by a true hypertrophy of its natural tissues, as to occupy the whole space of the vertebral canal; but most generally what seems to be hypertrophy at first sight is due, chiefly at least, to congestive swelling and œdema. True hypertrophy has been met with in the fœtus: it occurs mostly in children: and it presents, so far as is known, no symptoms by which it can be recognized. Hypertrophy of the brain is a very uncommon affection, but it is common as compared with hypertrophy of the spinal cord.

[1 Ziemssen, loc. citat.]

XV. Tumor, etc.

"Tubercle and cancer," says Rokitansky, "are frequent in the brain, unfrequent in the spinal cord. Tubercle I have observed only in combination with other advanced tuberculoses. Its principal seat is the cervical or lumbar portion of the cord, where it sometimes occupies the white fibres, sometimes the gray substance. As in the brain, it leads to inflammation (red softening) and to yellow softening. I have never seen a tuberculous cavity in the cord. Sometimes several tubercles are grouped together, none exceeding the size of millet or hempseed; at other times only one exists, which is of large dimensions, equalling a pea or a bean. Exclusively of several cases of circumscribed callous induration of the white columns, as to the cancerous nature of which I am still in doubt, I have met with but one case of cancer of the cord. It was a solitary nodule of medullary cancer. Ollivier mentions several examples of diffused carcinomatous growths, as well as of the so-called colloid cancer. Among the entozoa I have repeatedly seen the cysticercus in the cervical portion of the spinal marrow. The acephalocyst sacs, as far as has been observed, have no connection with the cord; their nidus is even outside the dura mater. In one case the cyst forced its way into the cavity of the arachnoid."

Nor are exostoses, cartilaginous growths, or aneurisms frequently met with in positions which can exercise pressure upon the spinal cord. Cartilaginous growths, or rather bony plates, it is true, are not unfrequently met with in the visceral arachnoid of the cord—a condition which appears to be rarely met with in the brain; but these growths or plates can scarcely be brought under the head of tumors. Except, perhaps, in connection with scrofulous disease of the vertebræ, the pia mater of the cord is not the seat of tuberculous deposits; and here again is another point of difference between the pathological history of the spinal cord and the brain, for it is a well-known fact that the pia mater of the brain is a favorite seat of these deposits.

The symptoms produced by tumor vary greatly. Neuralgic pain in the back, over the seat of the tumor, appears to be an almost constant symptom. "Pain," says Dr. Reynolds, "is more marked in cases of carcinoma than of tubercle." If a particular nerve be irritated by the tumor, there may be pain, tingling, or some other anomalous sensation in the part or parts supplied by its sentient fibres, or some morbid form of contraction in the muscles supplied by its motor fibres. If a particular nerve be pressed upon more decidedly by the tumor, there may be local anæsthesia, or paralysis instead of morbid sensations or muscular contractions. It is but seldom, however, that these symptoms of irritation or pressure are so strictly localized; and, in fact, the presence of the tumor is made known usually only by more general symptoms of irritation, or compression, or inflammation, which, instead of being in any way pathognomonic of tumor, may arise from many other causes. "There is, indeed," as Sir W. Gull says, "no symptom, or single group of symptoms, which, taken alone, can serve as a secure basis for diagnosis." Tuberculous or carcinomatous deposits elsewhere, with signs of the peculiar dyscrasia of tubercle or cancer, aneurism elsewhere, nodes elsewhere, may help to a diagnosis by showing that symptoms which appear to point to a tumor may have such a cause, and at the same time may supply some information as to the special character of the tumor; but this possibility of help in diagnosis is too remote to be of much practical value, if any. It may be supposed that any scrofulous deposit in the cord is more likely to occur in children, and any cancerous growth in older persons; but even this rule has too many exceptions to make it of much use.

XVI. Concussion.

Concussion of the spinal cord, like concussion of the brain, is the result of a fall from a height, a blow on the back, or some other accident, and its symptoms vary with the intensity of the shock. Sudden paralysis and loss of sensation, more or less complete, with some inability to pass water or to prevent the escape of flatus or feces, are the more special symptoms. Sudden and marked failure in the circulation and respiration, as shown by pallor, feebleness of the pulse, diminished temperature, slow and shallow breathing, and other signs of common shock, are also associated with the more special symptoms. Great pain along the spine or in some part of the spine has been considered as one of the symptoms of spinal concussion; but neither pain nor spasm is met with in the cases which I have examined; and Dr. Reynolds comes to the same conclusion, for speaking of these cases, he says, "There is in them neither marked pain nor spasm." Indeed, in the majority of cases the patient is obviously rendered incapable of experiencing pain by the fact of being *stunned*.

The symptoms of spinal concussion not unfrequently issue in those of spinal congestion, or myelitis, or spinal meningitis, or else death without any signs of reaction may be the result. Often, without passing into any definite disease, the cord, even after what might at first seem to be

only a slight degree of concussion, may not recover its former power perfectly, the patient ever afterwards being weak in many respects, especially in his legs and bladder. Indeed, concussion of the spine sufficiently severe to produce at the time any marked degree of paralysis in the limbs and bladder and lower bowel, with loss of sensation, is certainly a very grave matter, and it may be questioned whether in such a case recovery is ever more than partial.

The appearances after death may present nothing unnatural, or they may be those of hemorrhage more or less extensive. It is very possible that the cases in which severe pain in the back was a symptom would prove, if *all* the *facts* were fully known, to be cases in which the symptoms of concussion were mixed up with those of hemorrhage: at any rate, there was hemorrhage in one case of spinal concussion in which pain in the spine was a conspicuous symptom, which case came under my own notice not long ago. In fatal cases, in which the reaction after the concussion has issued in inflammatory and other changes in the cord, these changes will be met with after death; and if fracture or dislocation of the vertebræ was produced at the time of the concussion, the evidence of such injury will of course not be wanting.

XVII. Compression.

When the spinal cord is compressed by a dislocated or fractured vertebra, by a tumor, by a bullet, or in any other way, the symptoms will of course vary with the seat and degree of compression. The symptoms will, in fact, be as variable—for they will be the same—as those which are produced by experimental division of the parts compressed, and about which more than is convenient had to be said in the preliminary remarks. All, therefore, that is necessary here is to refer to those preliminary remarks for the information which may help to make the symptoms of compression intelligible, and, in passing, to express a hope that trephining or other operative procedures which have been recommended and practised in certain cases of spinal compression may not be altogether unjustifiable.

XVIII. Caries of the Vertebral Column.

This disease is usually limited to the bodies of the vertebræ and to the intervertebral substances, but sometimes it extends backwards to the arches and processes of the vertebræ as well. It commences, very generally, in the middle dorsal region, and, as generally, it does not extend beyond this region; but there is no part of the spinal column in which it may not begin, or to which it may not extend: it invariably, when sufficiently advanced, gives rise to "angular curvature," or projection directly backwards of the diseased part of the spine, this deformity being due to the way in which the thinned and diseased bodies of the vertebræ become crushed in under the weight of the upper part of the body. In the great majority of cases caries of the vertebræ is an unmistakably strumous affection, being neither more nor less than tuberculous infiltration of the bodies of the vertebræ; and the changes in the bone are due to the melting down of this deposit rather than to any strictly inflammatory process.

The earlier symptoms of caries of the vertebræ are not at all well marked. Of these the most conspicuous are—weakness in the back, generally in the dorsal region, with aching or pain, more or less severe, in the weak part, causing a disposition to lean forward and to use the arms as props; some prominence of the spinous processes of the weak and painful part of the spine, with some puffiness of the overlying skin; a feeling of undue heat, or even burning, in the weak and painful and prominent part, which is not felt in other parts of the spine, when a sponge soaked in moderately warm water is passed down the spine; and a state of tenderness on pressure or percussion, which is equally restricted to the same weak and painful and prominent part. Afterwards, when the disease is more advanced, there are more marked symptoms, namely these:—unmistakable "angular curvature," the formation of abscess, slight hectic in the evening, a feeling of constriction around the waist, it may be, and still later, more or less paralysis of the legs, more or less loss of control over the bladder and bowel, and other symptoms indicative of secondary myelitis or spinal meningitis. Abscess may be one of the earlier symptoms preceding any obvious deformity, or it may not occur at all. In fact, abscess appears to be a symptom of strumous disease of the vertebræ exclusively, and not of the non-strumous variety of caries. When it does occur, which is certainly in the great majority of cases, there is usually some diminution of pain and other evidences of irritation. When it does occur, as is well known, it generally makes its appearance at a distance from the diseased vertebræ, most commonly as "psoas abscess" in the groin, but by no means exclusively in this form and locality. It is seldom that the spinal cord becomes *compressed* by the giving way of the bodies of the vertebræ in the progress of the disease: but sooner

or later it almost constantly happens that the cord or its membranes opposite the diseased vertebræ become the seat of inflammatory changes, which changes, rather than the drain from an abscess, are indeed the reason why, in so many cases, sooner or later, caries of the vertebræ proves to be destructive to life.

The diagnosis between "angular curvature" from caries of the spine, and the curvatures forward, backward, and sideways, without other structural changes in the vertebral column than those of simple adaptation to the altered position, is not very difficult. These latter curvatures, in fact, want all the special and grave features which have been indicated as characterizing the former. Nor yet is the diagnosis difficult between "angular curvature" in its earliest stage and spinal irritation, with which it is sure to be associated, and with which there is certainly no small danger of its being confounded. This topic has been already touched upon when speaking of spinal irritation, and here it is enough to say that the occurrence of the symptoms which are present in the beginning of caries of the vertebræ (which are no other than those which may belong to simple spinal irritation), in children or youths of a manifestly scrofulous habit—at an age, that is to say, and in a habit, in which symptoms of simple spinal irritation are not likely to be met with—are sufficient to do more than create a bald suspicion of the existence of disease of the vertebral column.

The prognosis of caries of vertebræ is always bad enough. A hump-back is the best result to be hoped for. The end to be aimed at in treatment is, of course, to promote anchylosis of the diseased bones of the vertebræ by allowing them to fall together—by favoring, that is to say, the deformity which is inevitable by letting the back bend and not by trying to prevent it by keeping the back straight,—and to keep up the strength in every way. But these are matters which I cannot touch upon without trespassing upon the domains of surgery, and I therefore leave them to those who are better able, and whose right it is, to deal with them.

XIX. Spina Bifida, &c.

The commonest congenital affection to which the spinal cord is liable is dropsy, or hydrorachis, and of this dropsy *spina bifida* is the variety most frequently met with, and of most practical interest. The spine is bifid in this disorder from the non-development or separation of the spinal processes and laminæ, and the consequence of this malformation is that an opening is left through which, very often, the dropsical fluid presses outwards, and distends in so doing the integuments and subjacent tissues into an hernial tumor. Very generally congenital hydrocephalus is associated with congenital hydrorachis. The fluid in hydrorachis is precisely of the same constitution and character as that which is met with in hydrocephalus: it varies in quantity from a few ounces to several pints: it accumulates between the arachnoid and pia mater, in the arachnoid sac, in the central canal of the cord, and even outside the dura mater, sometimes in one place, sometimes in another, sometimes in more places than one. The hernial tumor into which this dropsical fluid bulges outwardly varies greatly both in position and size, and in the condition of its coverings: it is almost invariably met with in the lumbar region, but it may be in any region: it is usually of the size of a walnut or orange, but it may be as large as a child's head, or even larger: it may be single or multiple: its bulk may vary considerably under different circumstances, or not at all, becoming, if it vary, fuller and more tense if the position of the child be made such as to cause the fluid to flow into it, emptier and flaccid if this position be altered so that this fluid may run out of it, or if pressure be made upon it so as to bring about the same result: it may swell during expiration and fall during inspiration: it may present distinct fluctuation or none at all; and the skin over it may be sound, thickened, inflamed, ulcerated, gangrenous, covered with tufts of hair, and so on. The dura mater and its lining of arachnoid membrane always enter into the composition of the coverings of the tumor, and these are the only constant elements in these coverings. In the lumbar region, the cord and its nerves, which are generally rudimentary, are out of the tumor altogether: in the cervical and upper dorsal region, on the contrary, it is no uncommon thing for the cord and its nerves to be adherent to the walls of the tumor.

In spina bifida the lower limbs are generally paralyzed as well as the bladder and lower bowel, and not unfrequently there is, in addition to the spinal deformity, deficiency of the abdominal walls, hernia of the bladder, imperforate anus, &c. But few cases recover, or even improve, death happening generally at an early period either in convulsions or from spinal inflammation, the immediate cause often being the bursting of the tumor: still there are cases on record in which life has been prolonged—and this too with tumors of no small size—not only for a few months, but for 17, 18, 19, 21, and even 50 years.

There is little to be done for the relief of spina bifida. Pressure on the tumor by means of an air-pad and suitable ban-

dages can do no harm; and occasional punctures with a grooved needle, as recommended by Sir Astley Cooper, may be a justifiable measure. Even cures have resulted from a combination of these punctures with pressure. "All the plans of treatment," says Mr. Erichsen, "by which the tumor is opened and air allowed to enter it, are fraught with danger, and will, I believe, inevitably be followed by the death of the child from inflammation of the meninges of the cord and convulsions."

There are several other congenital affections of the cord, of which the best account is still to be found in the classical pages of Ollivier. The cord may be entirely absent (*amyélie*); or it may be imperfect (*atelomyelie*). Of the imperfect forms of cord there are several varieties. The upper part may be wanting, as in anencephalous and acephalous monsters. The cord may be bifurcated at one extremity or the other, at the upper extremity in monsters with two heads and one body, at the lower extremity in monsters with one head and two bodies. It may be double. It may vary greatly in dimensions, being larger or smaller, longer or shorter than natural —longer, for example, in monsters with tails, shorter in monsters of a contrary sort. It may, as in one form of hydrorachis, be little more than a long bag in consequence of the distension of the central canal of the cord with the dropsical fluid. Or it may be discolored, as it is in the state which Ollivier designates *kirronese* or *coloration ictérique*. These malformations or morbid conditions, however, are of theoretical rather than of practical interest: and therefore they do not form fit subjects for further notice in an article like the present, which has solely a practical end in view.

B.—PARTIAL DISEASES OF THE NERVOUS SYSTEM.—
Continued.

3. Diseases of the Nerves.

Neuritis and Neuroma.	Local Paralysis.	Torticollis.
Neuralgia.	Local Spasms.	Local Anæsthesia.

NEURITIS AND NEUROMA.

By J. Warburton Begbie, M.D., F.R.C.P.E.

Morbid appearances, the results of inflammatory action, are occasionally met with in nerves. Such are the consequences usually of injury; the nerves have been divided by a sharp instrument; or if independent of wounds, they are in all probability connected with rheumatism or gout. There seems no reason to doubt that inflammatory action may likewise extend to nerves from the contiguous tissues.

In its general characters Neuritis resembles the inflammation of fibrous tissue. The fibrous investing sheath of nerves, or neurilemma, is indeed its usual seat; the appearance of inflammatory action being for the most part limited to it, and only seen in the form of red softening of the nervous tissue itself when the inflammation has been of an intense description.

A doubt as to the spontaneous occurrence of Neuritis has been entertained and expressed by several authorities. Boerhaave, for example, writes: "Nemo forte unquam vidit inflammationem in nervo; hæc vero si contingat, in sola *tunica vaginali* hæret."[1] Others, again, with even greater inaccuracy, have maintained the frequent existence of Neuritis.[2] Pathologically the inflammation of nerves may be acute or chronic; and these two conditions are described by Rokitansky as follows: The marks of the former (acute) are—(*a*) Injection and redness. The injection presents a linear arrangement, and the redness is partly caused by injection, and partly by small extravasations. (*b*) Looseness, succulence, and swelling of the nervous cord, due to infiltration of serum into the tissue of the neurilemma, and into the sheaths between the primitive nervous filaments. The nerve has lost its smooth, white, glistening appearance; its neurilemma is opaque, and has a rough and wrinkled look. (*c*) Exudation. This is generally a grayish or yellowish-red gelatinous product, which sooner or later becomes firm. It occupies the sheath and tissue of the neurilemma, and is likewise effused between the primitive filaments themselves. (*d*) The cellular tissue around the nervous cord always participates in these changes; it becomes injected, reddened, and infiltrated with a sero-fibrinous fluid. Not only the neighboring cellular tissue, but the sheaths of the muscles, the fascia, the subcutaneous cellular tissue, and the general integuments, become involved.

Such a degree of inflammation as that now described may terminate in *resolution*, occurring quickly or slowly in different cases, or in *induration* of the nerve, and a permanent loss of its function in whole or in part. If the latter be the result, the nerve continues thickened, and more or less misshapen, forming a grayish cord, which is sometimes marked with black pigment and crossed by varicose vessels. The nerve filaments diminish in size and finally disappear, this result being in part due to the pressure to which they are ex-

[1] De Morbis Nervorum.

[2] See on this point Animadversiones de Neuritide: Praxeos Medicæ Universæ Præcepta, auctore Josepho Frank; Partis secundæ, volumen primum, Sectio secunda, p. 131. Also Elements of General and Pathological Anatomy, by David Craigie, M.D., p. 379.

posed by the inflammatory product, and in part to the interrupted nutrition, for the vessels are obliterated by the inflammatory process. (*e*) In a more intense inflammation the primitive nervous filaments are destroyed. They are found in a state of red or grayish or yellowish-red softening, while the neurilemma is easily torn. (*f*) The fluid product of the inflammation may be purulent; and if so, the nerve appears highly discolored, and infiltrated with purulent fluid tinged with blood. The neurilemma is then much altered, and readily gives way, while the nerve is converted into a yellowish-red, brownish-red, or chocolate-colored pulp. The cellular tissue surrounding the nerves becomes infiltrated with yellow fibrinous exudation, and abscesses are formed in its course. (*g*) Ulcerative destruction of the nerve is the next step. But if the progress of inflammation be stayed before that point is reached, granulations appear, which become progressively changed into cicatricial tissue, as is observed in the stump of a nerve after amputation. Nerves, however, resist for a lengthened period the suppurative and sanious destruction which may be going on around them.

Chronic Inflammation is characterized by the varicose state of the vessels of the affected nerve, by products which become indurated, and gradually increase in quantity, and by a change of the nerve to a slate or lead-gray color. Sometimes the products are not deposited uniformly throughout the nerve, and then nodular swellings are formed on it.[1] Romberg, when directing attention to the anatomical knowledge we possess of sciatica, speaks of Neuritis being found, but of its rare occurrence.[2] The same writer, however, refers to the possible production of Neuritis, by the sciatic plexus being dragged and irritated by the head of the child in a difficult labor. Valliex and Beau have described inflammation of nerves more systematically than other authors. The latter has at considerable length, in his interesting memoir on the subject, directed attention to "Intercostal Neuritis."[3] Reference has been made to the occurrence of a rheumatic or gouty Neuritis. Dr. G. B. Wood considers it to be highly probable that in a large proportion of cases rheumatism lies at the foundation of the disease.[1] And Dr. Garrod, while admitting, according to the usually received notion, that the nervous affections occurring in connection with gout are generally functional, believes them sometimes to be dependent on inflammatory action, which, he adds, appears, so far as can be ascertained, to have the character of true gouty inflammation.[2]

The most characteristic symptom of Neuritis is pain, not limited to the precise seat of the inflammation, but felt in the course of the nerve, and sometimes to its minutest branches. Besides its severity, the pain in Neuritis possesses other distinctive features: it is darting, and tingling, and there often accompanies it a feeling of numbness. The pain has been further described as intermittent, but is more probably remittent, being, as long as the disease continues, never entirely absent. Tenderness over the affected nerve invariably exists. It is possible that in some forms of local palsies (*see* Local Paralysis from Nerve Disease) the loss of power, partial or complete, as well as the existence of various morbid sensations, of which formication is one, and perhaps the most common, is due to disorganization or other permanent change in the trunk of a nerve, resulting from inflammatory action.

It seems to be generally admitted, that the nerve most liable to such change is the sciatic; but the various branches of the brachial plexus, and especially the ulnar nerves, likewise suffer; and so in all probability do at times the other nerves in both lower extremities and trunk.

That inflammation may also attack the nerves of special sense, as Dr. Wood has conjectured, seems not improbable, particularly the nerves of hearing and of sight. Most assuredly a true gouty inflammation, apparently commencing, in some cases, in the nerves themselves, not unfrequently either damages or entirely destroys one or other of the delicate organs connected with these most important functions.

In the treatment of Neuritis the probable alliance of the affection with some peculiar diathetic condition, the gouty or rheumatic, or possibly with the syphilitic cachexia, must not be lost sight of.

Local abstraction of blood, and the application of emollient and anodyne poultices, rest, low diet, and the use of laxatives, are the chief remedies in cases of the acute Neuritis. When the disease is chronic, the use of blisters, issues, and even the cautery, has been recommended.

---

[1] A Manual of Pathological Anatomy, by Carl Rokitansky. Sydenham Society's Translation, vol. iii. p. 462.

[2] Lehrbuch der Nervenkrankheiten des Menschen: Neuralgie des Huftnerven.

[3] Valleix, Guide du Médecin Praticien, t. iv. p. 299; also Traité des Névralgies. Beau, Archives Générales de Médecine, 4e série, t. xiii. 1847.

[1] A Treatise on the Practice of Medicine, vol. ii. p. 843.

[2] The Nature and Treatment of Gout and Rheumatic Gout, p. 517.

Internally, besides opium or other narcotic for the relief of pain, it will be prudent to give a fair trial in both the acute and chronic Neuritis to quinine, and colchicum, the iodide and the bromide of potassium.

NEUROMA (Tumor of Nerve).—Growths of various sizes and natures occurring in the course of nerves had been described before the term Neuroma came to be applied to such. Dr. Robert Smith, in his valuable and elaborate memoir, makes a brief reference to the early history of the subject;[1] and so likewise does Mr. William Wood, in his important papers entitled, "Observations on Painful Subcutaneous Tubercle," and "On Neuroma."[2] The famous English surgeon, William Cheselden, is specially mentioned, as having given the first accurate account of the nervous tubercle, which has become familiar chiefly through the writings of Mr. Wood. "Immediately under the skin, upon the shin bone, I have twice seen little tumors, less than a pea, round and exceeding hard, and so painful that both cases were judged to be cancerous: they were cured by extirpating the tumor. But what was more extraordinary was a tumor of this kind, under the skin of the buttock, small as a pin's head, yet so painful that the least touch was insupportable, and the skin for half an inch round was emaciated; this, too, I extirpated, with so much of the skin as was emaciated, and some fat. The patient, who before the operation could not endure to set his leg on the ground, nor turn in his bed without exquisite pain, grew immediately easy, walked to his bed without any complaint, and was soon cured." The same writer describes and figures the cystic neuroma. "A tumor formed in the centre of the cubital (ulnar) nerve, a little above the bend of the arm; it was of the cystic kind, but contained a transparent jelly; the filaments of the nerve were divided and ran over its surface. This tumor occasioned a great numbness in all the parts that nerve leads to, and excessive pain upon the least touch or motion. This operation (for the removal of the tumor) was done but a few weeks since; the pain is entirely ceased, the numbness a little increased, and the limbs as yet not wasted."[3]

The term Neuroma, or rather *Neuromes*, was first employed by M. Odier of Geneva. "Enfin," writes Odier, "on peut donner le nom de Neuromes à ces tumeurs mobiles, circonscrites et profondes, qui sont produites par le gonflement accidentel d'un nerf, à l'extrémité duquel la compression de la tumeur fait éprouver des crampes très-pénibles."[1]

There have been various classifications of neuromatous tumors attempted by pathologists, such as local and general—that is, as affecting one nerve, or several nerves; and, again, those which are the direct consequence of a morbid process, and those resulting from an original vice of conformation. Dr. Smith, rejecting these divisions, has suggested, as sufficient for practical purposes, that Neuromata should be considered as of two kinds: 1st, of spontaneous origin, or *Idiopathic;* 2d, as the result of wounds or other injuries of the nerves, and therefore *Traumatic.*

Before offering a brief description of these varieties, it may be well to direct attention a little more fully to the *painful subcutaneous tubercle*, which we have the authority of Dr. Hughes Bennett and other pathologists for stating "must be referred to this class of tumors,"[2] that is, neuromatous fibrous tumors.

"Although," remarks Dr. Smith, "pathologists have hitherto failed to discover anything like nervous structure in these tumors, I still incline to the opinion that they are connected with the minute filaments and ultimate ramifications of the nerves. Upon any other supposition it is, I conceive, impossible to offer a rational explanation to account for the dreadful severity of the suffering which they induce." Sir J. Paget, who has carefully examined the "painful subcutaneous tumors" describes them as being formed of "either fibro-cellular or fibrous tissue, in either a rudimental or a perfect state." Alluding to a case described by the late Professor Miller, in his "Principles of Surgery," and by Professor Bennett, the same pathologist admits that their structure may sometimes be fibro-cartilaginous.[3]

Of this affection the first detailed account was given by the late Mr. William Wood of Edinburgh. After the publication of Mr. Wood's earlier papers,[4] cases were recorded by different observers, and in 1829 an instructive *résumé* of the whole subject was laid by him before the Medico-

[1] A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma. Dublin, 1849.

[2] Transactions of the Medico-Chirurgical Society of Edinburgh, vol. iii. pp. 317 and 367.

[3] The Anatomy of the Human Body, 12th edit., London, 1784, pp. 136 and 256.

[1] Manuel de Médecine pratique, ou Sommaire d'un Cours gratuit, donné en 1800, 1801, et 1804, aux Officiers de Santé du département du Léman, par Louis Odier. Paris, 1811, p. 362.

[2] Clinical Lectures on the Principles and Practice of Medicine. 3d edit. p. 171.

[3] Lectures on Surgical Pathology, vol. ii. p. 123.

[4] The Edinburgh Medical and Surgical Journal, 1812. Two articles, pp. 285 and 429.

Chirurgical Society of Edinburgh, and appeared, as already mentioned, in its "Transactions."

This disease consists in the formation of a small lump or tubercle seated in the subcutaneous cellular tissue, immediately under the integuments, which retain their natural appearance. The tubercle is met with in different parts of the body, but most frequently in the extremities. It is extremely small, pisiform in shape, of firm consistence, and apparently quite circumscribed.

The characteristic feature of the disease is the occurrence of violent pain coming on paroxysmally. The paroxysms vary in duration from ten minutes to upwards of two hours, their frequency as well as intensity appearing to increase in precise relation to the length of time the disease has existed. Some patients enjoy intervals of relief from pain for days or even weeks, while others have repeated attacks in the course of a single day. The paroxysms of pain frequently occur when the patient has fallen asleep. They are also apt to be excited by various external causes, such as pressure and blows; while in rarer instances mental disquietude and atmospheric changes have been their only apparent occasion.

Females are more frequently the subjects of this disease than males. Wood, referring to thirty-five cases collected by him, mentions that twenty-eight were females, five males; and in the account of two the sex was not stated. Of thirteen cases quoted by Descot, ten occurred in females, and three in males. Romberg has met with three instances, all in females.

The situation of the tubercle in the thirty-five cases referred to by Wood was as follows: in the lower extremities in twenty-two, in the upper extremities in eleven, in the chest in one, and in one in the scrotum. In only two of these cases was there more than one tubercle present.

This disease does not seem to be intimately connected with any particular period of life, as it has been noticed at all ages from thirteen to above seventy.

"It is a happy circumstance that this very painful affection is capable of being remedied by a very simple operation. The tubercle is easily removed by a single incision, and it is unnecessary to take away any portion of the integuments, or of the surrounding cellular tissue. No bad effect can follow the removal of the little body." (Wood.)

Although this subcutaneous tubercle has been considered as a variety of Neuroma, it must be held in remembrance that, its distinct connection with branches of nervous trunks never having been determined, this is more a matter of inference than of demonstration. Ollivier and Rayer together carefully dissected the tumor in a case to which reference is made in his latest paper by Wood, and the result is thus expressed: "Extérieurement il était enveloppé de tissu cellulaire, dans lequel nous ne pûmes distinguer aucun filet nerveux, même à l'aide d'une forte loupe."[1] Paget remarks that the general opinion is against the supposition of the intimate connection of these painful tumors with nerves. "Dupuytren," he writes, "says that he dissected several of these tumors with minute care, and never saw the slightest nervous filament adhering to their surface. I have sought them with as little success with the microscope. Of course I may have overlooked nerve-fibres that really existed. It is very hard to prove a negative in such cases; and cases of genuine Neuroma, *i. e.* of a fibrous tumor within the sheath of a nerve, do sometimes occur, which exactly imitate the cases of painful subcutaneous tumor."

We have now to consider the first of the two forms of Neuroma, as distinguished by Smith, and now generally recognized —namely, the *Idiopathic Neuroma*. Tumors of this nature are of an oval or oblong form, their long axis corresponding with the direction of the nerve to which they are attached. They vary considerably in size. One figured in his work by Smith is as small as a grain of wheat, while another is as large as a good-sized melon. Between these two extremes every variety of size occurs. There may be only one, or several may be found on the same nerve; occasionally they are found existing simultaneously upon all the spinal nerves. "In number," says Rokitansky, "they vary from one until they are almost countless." A remarkable general disease is thus constituted, of which three cases have been observed in the Vienna Hospital. Neuromata are comparatively rare in the ganglionic system. But although occurring most frequently on the spinal nerves, Neuroma is not limited to them; the cerebral nerves, motor as well as sensory, particularly those most closely resembling the nerves of the cord, present at times the same tumors.

In general, Neuromata are solid throughout their entire structure, but in some instances are of cystic formation, as in the case recorded by Cheselden, and already referred to. These tumors are of slow growth, but continue to undergo a steady increase in size, although many years may elapse before they attain such dimensions as to prove a source of serious inconvenience. They are movable in the transverse direction, but not in the

[1] Traité théorétique et pratique des Maladies de la Peau, seconde édit., t. ii. p. 290. Paris, 1835.

course of the nerve upon which they are seated. There may be difficulty in distinguishing tumors which are merely contiguous to nerves from the true Neuroma, having its origin within the neurilemma. Wood has specially alluded to this difficulty in diagnosis, and Smith has pointed out that the non-nervous tumors, unlike Neuromata, are generally movable in all directions, and, when drawn away from the nerve, cease to be painful on pressure.

Nerve tumors are described by Rokitansky as lying between the fasciculi of the nerves, and interwoven with their neurilemmatous sheath. Neuroma, the same pathologist observes, is never deposited in the centre of a nerve, but at its side, so that only a small part of its fasciculi is displaced; the displaced fasciculi are spread abroad and stretched over the tumor, while the greater mass of the nerve remains on the other side uninjured, and with its fibres in connection with one another.

The solid neuromatous swellings are of a tough elastic consistence, of grayish or pale yellowish-red color, and are invested with a distinct fibrous sheath. Dr. Hughes Bennett thus describes them: "On being minutely examined, they are found to consist of fibrous texture more or less dense, the filaments often arranged in wavy bundles running parallel to each other, but occasionally assuming a looped form, or intercrossing with each other. I have also found them to contain groups of cells. Not unfrequently they are fibro-cartilaginous; sometimes with the cells closely aggregated together, at others widely scattered. In some of the neuromatous swellings described by Dr. Smith I found the fibrous tissue to present wavy bundles, among which a few granule and cartilage cells were scattered and shrivelled, apparently from the action of spirit."[1]

Neuromata seldom contract adhesion to the investing integuments, unless they have been subjected to continued pressure. Smith has never known them to suppurate, or to be removed by absorption. Pain has been generally considered to be a characteristic feature of neuromatous swellings. In this respect, however, there is infinite variety. When a single Neuroma exists, there is almost invariably much suffering. The pain, moreover, occurs suddenly and paroxysmally, darting along the nerve with the violence and instantaneousness of an electric shock. On the other hand, in those examples of Neuroma which are distinguished by the number of the tumors, it is not uncommon to find these occasioning little or no inconvenience to the patient.

[1] Loc. cit. p. 171.

It is exceedingly difficult to determine with anything like exactness the real cause of the paroxysmal attacks and sudden aggravation of severe pain which occur in this as well as in many other forms of disease of the nervous system. Mental emotions and the ordinary atmospherical vicissitudes have been generally assigned as the occasion of these occurrences in Neuroma.

Paget has some very interesting observations on the cause of pain in Neuroma, as well as on the nearly entire absence of all suffering which has been noticed in some cases; and founding on the observations of Smith and others, including himself, this excellent writer is no doubt correct when he states "that we cannot assign the pain in these cases entirely to an altered mechanical condition of nerve-fibres in or near the tumor. We must admit, though it be a vague expression, that the pain is of the nature of that morbid state of nerve force which we call *neuralgic*. Of the exact nature of this neuralgic state indeed, we know nothing; but of its existence as a morbid state of nerve-force, or nervous action, we are aware in many cases in which we can as yet trace no organic change, and in many more in which the sensible organic change of the nerves is inadequate to the explanation of the pain felt through them." In short, Paget argues for the pain being functional, and not necessarily dependent at least on an organic disorder. If such a pain is found to be influenced by the remedies chiefly available for the relief of ordinary neuralgia—quinine, iron, arsenic, belladonna, stramonium, the bromide of potassium—this suggestion will receive corroboration.

We know that such Neuromata as are the seat of severe pain and of continual irritation may give rise to attacks of the so-called sympathetic epilepsy. Instances of this nature are to be found in the writings of several authors, and it is sufficient here to refer to the well-known views of Brown-Séquard respecting the exciting causes of the epileptic convulsion, and of many other nervous affections.[1]

In the idiopathic form of Neuroma the pain is generally limited to the parts below the tumor; and the sign of the true Neuroma, signalized by Aransoohn, has been accepted by others—namely, that when the trunk of the nerve is compressed above the tumor the pain ceases, and then the Neuroma previously acutely sensitive can be touched without any uneasiness being caused. The remark already

[1] Researches on Epilepsy, p. 35; also Course of Lectures on the Physiology and Pathology of the Central Nervous System, p. 181. Article Neuroma, by same Author, in Holmes's System of Surgery, vol. iii. p. 896.

made as to the solid variety of Neuroma not being necessarily painful applies likewise to the fluid or cystic tumor.

Our knowledge of the determining causes of Neuroma cannot be said to have advanced since the period when the important treatise of Dr. Smith first appeared, and we are still compelled to adopt his expression, "I feel it must be confessed that we know nothing with certainty regarding the causes of Neuroma."[1]

Neuromatous tumors have been frequently removed along with the corresponding portion of the nerve on which they were situated; and such operations, while entirely relieving the patients from suffering, have not been succeeded by any considerable loss of sensibility, or of the power of voluntary movement, in the parts supplied by even large nerves.

The sciatic nerve may be divided, as in a case of severe neuralgia of that nerve, by M. Malagodi, and a portion of it excised, without permanently destroying the functions of the limb.

The magnitude of the nervous trunk, which is the seat of the disease, will of course largely determine the period at which complete or partial restoration of the function in the limb is established. In some cases a few months, in others a year and upwards, have elapsed; but sooner or later, in all recorded instances, the banished sensibility and motor power have been regained.

The interference with the calorific function of the nervous system is strikingly exhibited in cases of operation for Neuroma. Mr. Adams and Dr. Smith have drawn attention to the diminution of temperature in the limb after the removal of the tumor, and with it a portion of nerve—a diminution readily noticed both by patients and operator, and which has lasted for a lengthened period, even after the restoration of the other functions.

It may then be concluded that when idiopathic Neuroma is seated in the hand, forearm, or upper arm (the positions in which it has most commonly been found), the operation of removal may be safely practised. It is possible that a similar plan might be adopted in the case of Neuroma in the lower extremity; but it is on record that amputation of the limb has been had recourse to by Chelius, in a case of nervous tumor occupying the popliteal space and stretching to nearly the centre of the back of the thigh. This was an illustration, and there are others which teach a similar lesson, of the disease having been permitted to attain a very large size—so large as to prevent any attempt being made for its simple removal.

[1] Loc. cit. p. 5.

TRAUMATIC NEUROMA.—Under this division are to be included tumors of nerves resulting from any form of mechanical injury, such as wounds, blows, pressure, or following amputation.

Traumatic Neuroma is almost invariably single. The tumor is the seat of intense pain, which, unlike the suffering in the idiopathic form of the disease, is not confined to the growth itself, or felt merely in the parts below it, but is frequently found extending along the nerve towards its origin. When Neuroma occurs as a consequence of a wound of nerve, it usually consists of a solid tumor, not invested by neurilemma, and destitute of any distinct capsule.[1] It is most likely to form, when the nervous cord has been cut, but not entirely divided; and cases of this nature are even more than ordinarily painful.

The following case is published by Mr. Wood in his "Memoir on Neuroma;" it occurred in the practice of Mr. Syme:—

"James Muir, aged 43. *30th June*, 1828.—On the inner side of the left knee, about a hand-breadth above the joint, there is a narrow depressed cicatrix, two inches long. Between this cicatrix and the sartorius there is a small tumor, about the size of an almond, and of very firm consistence. When the limb is extended, this tumor can hardly be perceived, being then overlapped by the sartorius; but when the knee is bent, it can be felt very distinctly. It is most movable in a lateral direction, but seems pretty firmly connected to the subjacent parts by condensed cellular substance.

"The patient states that the tumor is always painful when pressed, but is more so at one time than another. The pain is not confined to the part, but shoots all over the knee, and sometimes extends from the groin to the toes. He observes that the pain is more severe during cold and damp weather. It frequently, for days together, prevents him from walking, or even resting on the limb. His story is, that when a boy, about eleven years old, he strained his knee by jumping into a saw-pit, which led to the formation of a large abscess that opened on both sides of the knee, namely, at the part where the cicatrix above mentioned still remains, and exactly opposite, where also there is a similar cicatrix. Several small bits of bone were discharged, and at the end of two years he got quite well. For the following twenty-seven years he led an active life; ten of them were spent in a militia regiment. About eight years ago he strained his knee while walking in his garden, and thereafter became subject to flying pains about the joints. These

[1] Smith, loc. cit. p. 20.

pains induced him to rub the knee frequently; and in doing so, about two years ago, he noticed the tumor. It was then the size of a pea, and has gradually enlarged. The disagreeable symptoms also have become greatly aggravated; and, as he refers them all to the tumor, he is desirous of having it removed.

"12*th July*.—Mr. Wood (continues Mr. Syme), who was kind enough to examine the patient, having agreed with me that the tumor was seated on or in the nervus saphenus, and that it ought to be removed, I performed the operation, with his assistance, on the 1st of July.

"The tumor being divided showed a firm fibrous capsule, containing a soft, brownish-white pulpy matter. The nerve was traced into the tumor, but not through it. The patient made a good recovery, and remains free from his complaint."[1]

The foregoing case illustrates the proper treatment of Traumatic Neuroma, which is to excise the tumor with the corresponding portion of nerve, in every case when its situation will permit of this being done.[2]

The last form of Neuroma which requires any separate consideration is that succeeding to amputations. Smith remarks in regard to such, that "their existence is so constant that we may, perhaps, consider them as representing the normal condition of the ends of the nerves in stumps." Generally they cause no uneasiness whatever: but, on the other hand, they have occasionally been the source of severe neuralgia, occurring in paroxysms of great length.

The Neuroma of stumps varies in size, being in some instances not larger than a garden-pea, in others as large as a grape, or even plum. Such Neuromata are generally of an oval or oblong form, of grayish-white color, and of a firm dense texture.

The situation of the Neuroma in the stump is not always the same; it may be several inches above the surface of the latter, and be connected with the cicatrix by means only of a fibrous cord, itself destitute of any nervous structure.

It is the opinion of some pathologists, that the Neuromata succeeding amputation are produced by the pressure which is exerted upon the surface of the stump. An objection fatal to this explanation, however, has been advanced—namely, that in many stumps which have never been subjected to pressure these little tumors are found.

Dr. Smith believes their formation to be for the protection of the extremity of the nerve.[1]

NEURALGIA.

By Francis Edmund Anstie, M.D., F.R.C.P.

Definition.—A disease of the nervous system manifesting itself by pains, nearly always unilateral, which appear to follow the course of particular sensory nerves. The pains are usually sudden in their commencement, and of a darting, stabbing, boring, or burning character; they are at first unattended with any local change which can be recognized, or by any constitutional pyrexia. They are always markedly intermittent; sometimes regularly and sometimes irregularly so. The periods of intermission are distinguished by complete freedom from acute suffering, and in recent cases the patient appears quite well at these times. In old standing cases, however, persistent tenderness and other signs of local mischief are apt to be developed in the tissues which surround the distribution of the nerves which are the seat of acute pains. Severe attacks of Neuralgia are usually complicated with secondary affections of other nerves which are intimately connected with that which is the original seat of pain; and in this way congestion of bloodvessels, hypersecretion, or arrested secretion from glands, inflammation and ulceration of tissues, &c., are sometimes brought about.

Synonyms.—The word Neuralgia has a generally recognized force, and there is no equivalent to it (except foreign variations in mere terminology) which represents the whole group of disorders to which it applies, though there are nu-

[1] Loc. cit. p. 426.
[2] Smith, p. 22.

[1] See Wounds and Injuries of Nerves, by Drs. Mitchell, Morehouse, and Keen. Phila. 1864.—H.]

merous phrases for particular forms of the disorder.

Clinical History and Symptoms.—These vary so greatly in different cases of Neuralgia that it will be necessary to discuss the greater part of this subject under the headings of the special varieties of the disease. There are certain features, however, which are observed in all true Neuralgias.

In the first place, it is universally the case that the existing condition of the patient at the time of the first onset of the disease is one of debility, either general or special. I make this statement with great confidence, notwithstanding the contrary assertion advanced by so high an authority as Valleix, whose able[1] treatise really laid the foundation for all our accurate knowledge of the Neuralgias. In the first place, it is certainly the case that the larger half of the total number of patients coming under my care with various forms of Neuralgia are either decidedly anæmic or have recently undergone some exhausting illness or fatigue: and the reason why Valleix did not find so many cases of this type among his neuralgic patients appears certainly to be, that he limited the neuralgic class of diseases by an artificial definition, which we shall have to reject as untenable. On the other hand, although a considerable number of neuralgic patients are so far healthy in appearance, that they have a fairly ruddy complexion and a good amount of muscular strength, it is impossible to admit that these facts disprove the existence of debility, either structural or functional, *in the nervous system*, for the commonest experience teaches that such debility does frequently coexist with great robustness and development of the apparatus of vegetation and the lowest forms of animal function. And it will invariably be found, on carefully examining these apparently robust neuralgic patients, that the nervous system has given warnings of its weakness: thus, the patient who, after an exhausting confinement, attended with great loss of blood, is attacked with obstinate *clavus hystericus*, will inform us that whenever in earlier life she had suffered from headache, the pain was always chiefly, if not altogether, confined to the nerves which are now the seat of decided Neuralgia. In a large number of cases I have also found that the attack of acute pain was immediately ushered in by a remarkably *anæsthetic* condition of the parts about to become painful; and a slighter degree of blunted sensation may often be observed in the intervals between the earlier attacks in cases of Neuralgia. In short, I have never seen a case of neuralgic pain in which there were not marked evidences of nervous debility, either local or general.

Another circumstance is common to all Neuralgias of superficial nerves; and as a large majority of neuralgic affections are superficial in situation, this is, for practical purposes, a general characteristic of the disease. I refer to the formation of *tender spots* at various points where the affected nerves pass from a deeper to a more superficial level, and particularly where they emerge from bony canals, or pierce fibrous fasciæ. So general is this characteristic of inveterate cases, that Valleix founded his diagnosis of the genuine Neuralgias on the presence of these painful points, in which assumption I think there can be little doubt, that he committed an error.[1]

The third general characteristic of neuralgic affections is, that the pain is intermittent, or at the least remittent, in every stage of the disease.

The fourth general characteristic is, that fatigue and every other temporary depressing influence directly predisposes to an attack of acute pain, and aggravates it when already existent.

Varieties.—It is possible to classify the Neuralgias upon either of two systems: first (A), according to the constitutional condition of the patient; and, secondly (B), according to the situation of the affected nerves. It will be necessary to follow both these lines of classification, avoiding repetition as much as possible.

(A) In considering the influence of constitutional states upon the typical development of Neuralgia, it will be convenient to commence with (I.) the group of cases in which the general state of the organism exerts the least amount of effect. This is the case where the pain is the result of direct injury to a nerve-trunk, whether by external violence, by the mechanical pressure of a tumor, or by the involvement of a nerve in inflammatory or ulcerative processes, spreading to it from neighboring tissues. As regards the development of symptoms, the important matters are, that the pain in these cases commences comparatively gradually, that the intermissions are usually much less complete, and that the pain is far less amenable to relief from remedies than in

[1] Traité des Névralgies. Paris, 1841.

[1] Trousseau insists with much energy that a still more important "point douloureux" is constantly present in Neuralgia, viz., over the spinous processes of one or more vertebræ, corresponding to the origin of the painful nerve. It is true (as the Brothers Griffin had long before pointed out) that there is *tenderness* in this situation. But this "point apophysaire" is not always, nor frequently, the seat of *spontaneous pain*.

other varieties of Neuralgia. The little that can be said about the form which is dependent upon progressively increasing pressure, or involvement of a nerve in malignant ulcerations, caries of bones, or teeth, &c., falls under the heads of Diagnosis or Treatment, and need not detain us here. The clinical history of Neuralgia from external violence, however, requires separate discussion.

1. Neuralgia from external violence may be produced by a shock (as of a fall, a railway collision, &c.), which gives a jar to the central nervous system, or by severe mental emotion, operating upon the same part of the organism. Upon either of these circumstances the development of the affection seldom occurs at once, but ensues after a variable interval, during which the patient exhibit symptoms of a general depression, with loss of appetite and strength. Sometimes vomiting, and even, in other instances, actual paralysis of a partial and temporary kind occur. When once developed, the neuralgic attacks are undistinguishable from those which occur from causes internal to the organism. The affection is usually very obstinate. In a large number of cases the nerve or nerves affected have previously shown signs of weakness, by a tendency to painful affection in depressed states of the organism. In the greater number of instances, as far as my experience goes, it is the fifth cranial nerve which becomes neuralgic from the effects of central shock. Illustrative cases will be given in the sections on local classification.

2. Neuralgia from direct violence to superficial nerves is produced either by cutting, or, more rarely, by bruising wounds.

Cutting wounds may divide a nerve-trunk, (α) partially, or (β) completely.

(α) When a nerve-trunk is partially cut through, neuralgic pain commonly occurs, if at all, immediately on the receipt of the injury. One such example only has come under my own care, but many others are recorded.[1] In this case the ulnar nerve was partly cut through with a tolerably sharp bread-knife, at a point not far above the wrist; partial anæsthesia of the little and ring fingers was induced, but at the same time violent neuralgic pains in the little finger came on, in fits recurring several times daily, and lasting for about half a minute. Treatment was of little apparent effect in promoting cure, though opiates gave temporary relief, as did the local use of chloroform. The attacks recurred for more than a month, long after the original wound had healed soundly; and for a long time after this pressure on the cicatrix would reproduce the attacks. A slight amount of anæsthesia still remained when I last saw the patient, more than a year after the injury.

(β) Complete severance of a nerve-trunk is a sufficiently common accident, far more common than is the production of Neuralgia from such a cause; indeed so marked is this disproportion between the injury and the special result, that I have been led to the conclusion that a necessary factor in the chain of morbid events must be the existence of some antecedent peculiarity of organization in the central origin of the injured nerve. This opinion is rendered more probable by the fact that the consecutive Neuralgia is not unfrequently situated not in the injured nerve itself, but in some other nerve with which it has intimate central connections. Two such examples are recorded in my Lettsomian Lectures,[1] in which the ulnar nerve, and one in which the cervico-occipital, respectively, were completely divided: in all three instances the Neuralgia was developed in the branches of the trigeminus. In all the cases which have come under my notice the Neuralgia, whether direct or reflex, set in at a particular period, viz., after complete cicatrization of the wound, and while the functions of the branches on the peripheral side were partly, but not completely, restored. The same obstinacy and rebelliousness to treatment was noticed as in other instances of Neuralgia from injury.

A few words must be given, before quitting the subject of Neuralgia from wounds of nerves, to the cases in which a foreign body lodges, with more or less laceration, in the substance of a nerve trunk. I have never seen such a case; but many instances are recorded in which most violent and painful Neuralgia has been set up in this way. Not unfrequently the irritation produces no noticeable effect on the nerve actually pressed upon, but sets up Neuralgia in a nerve so distant that no connection is suspected between the neuralgic pain and the original accident. The removal of a small piece of glass, or such other irritating body from the cicatrix of an old wound, has in several recorded instances put an end to neuralgic pains in quite another situation, for which all manner of remedies had long been tried. Sometimes the neuralgic pain has been accompanied by tissue degeneration of an alarming character, and these have likewise ceased at once upon the removal of the peccant body which had been the unsuspected source of the evil.

Neuralgias which result from some local injuries of so peculiar a character as *gunshot wounds* scarcely fall properly within the province of this article. The

[1] *Vide* Lancet, 1866.

[1] *Vide* Lancet, 1866.

reader who desires to know all that can be said with regard to this particular class of affections is recommended to study the able and carefully compiled "Report" of Messrs. Mitchell, Morehouse, and Keen.[1]

The case of Neuralgia from injury, pressure, and local disease of nerves has been mentioned first, because this form of the disease is less influenced than others by general constitutional states. But it is an erroneous opinion, however common, that the general condition of the body is here without any influence on the development of the nerve-pain. It has been forcibly urged, by Dr. Brinton and Dr. Handfield Jones more especially, that a condition of general bodily vigor mitigates, and that constitutional debility decidedly aggravates, these forms of Neuralgia; and my own experience gives most practical proof of the justice of this argument.

(II.) *Neuralgias of intra-nervous origin.*—As regards the constitutional conditions with which the several varieties of Neuralgia that arise independently of external violence, or disease of extra-nervous tissues, are respectively allied, the following preliminary subdivision may be made:—

1. Neuralgias of malarious origin.
2. Neuralgias of the period of bodily development.
3. Neuralgias of the middle period of life.
4. Neuralgias of the period of bodily decay.
5. Neuralgias associated with anæmia and mal-nutrition.

1. *Neuralgias of malarious origin* were formerly far more prevalent than they are at present, within the sphere of the English practitioner of medicine; with the general decline of malarial fevers, consequent on improved drainage and cultivation of lands, they have become constantly more scarce. In former times, on the contrary, they were so common, that they forced themselves on the notice of every physician. The term "brow-ague," to this day applied by many medical men to every variety of supra-orbital Neuralgia, is a relic of the older experience on this point; as is also the very common mistake of expecting all neuralgic affections to present a distinctly rhythmic recurrence of symptoms.

My own experience of malarial Neuralgia has been very limited, and I may as well say all that I know of its symptoms at once. In fact, though the out-patient practice of the Chelsea Dispensary and Westminster Hospital has afforded me a considerable number of examples of ague in past years, I have only seen two undoubted and one doubtful case of malarial Neuralgia, in all of which the fifth nerve was affected. The periodicity in one of the genuine cases was regular tertian; in the other regular quotidian. An algide condition always ushered in the attacks; but this was gradually exchanged, as the pain continued, for a condition in which the pulse was rapid, soft, and bounding, and the strength was further depressed. In both of these cases there were unilateral flushing of the face, and congestion of the conjunctiva, to a slight degree, during the attack of pain. The pain became duller and more diffused contemporaneously with the lowering of arterial pressure (as estimated by Marey's Sphygmograph); and after the disappearance of active pain, moderate tenderness over a considerable tract around the course of painful nerves remained for some time. But there was no distinct development of the *painful points* of Valleix (to be hereafter described), a circumstance which I attribute to the rapid cure of the complaint, in each instance, by quinine.

2. *Neuralgias of the period of bodily development.*—By the "period of bodily development" is here understood the whole time from birth up to the twenty-fifth year, or thereabouts. This is the period during which the organs of vegetative and of the lower animal life are consolidating. The central nervous system is more slow in reaching its fullest development, and the brain more especially is many years later in acquiring its maximum of organic consistency and functional power.

That portion of the period of bodily development which is antecedent to puberty is but little obnoxious to neuralgic affections. From the moment when puberty arrives, however, all is changed. In the stir and tumult which pervade the organism, and especially in the enormous diversion of its nutritive and formative nisus to the evolution of the generative organs and the correlative sexual instincts, the delicate apparatus of the nervous system is apt to be overwhelmed, as well as left behind, in the race of development. Under these circumstances the tendency to neuralgic affections rapidly increases. It will, however, be seen later that there is a great preponderance of particular varieties of the disease among the cases occurring during this period.

3. *Neuralgias of the middle period of life.*—By this period is meant the time included between the twenty-fifth and about the fortieth or the forty-fifth year. It is the time of life during which the individual is subjected to the most serious pressure from external influences. The

[1] Report on Gunshot Injuries to Nerves, observed in the late American War. Philadelphia, 1864.

men, if poor, are engaged in the absorbing struggle for existence and for the maintenance of their families; or, if rich and idle, are immersed in dissipation, or haunted by the mental disgust which is generated by *ennui*. The women are going through the exhausting process of child-bearing, and supporting the numerous cares of a poor household in some cases, or are devoured with anxiety for a certain position in fashionable society for themselves and their children, or again they are idle and heart-weary, or condemned to an unnatural celibacy. Very often they are both idle and anxious.

It must not be supposed that there is a sharp line of demarcation between this period and the last: nevertheless it will be seen, when we come to discuss the local varieties of Neuralgia, that there are certain broad differences in the general tendencies of the two epochs. It must be noted that particular Neuralgias, which are first manifested in the development period, frequently recur, under special provocation, in the period of middle life.

4. *Neuralgias of declining bodily vigor.*—The period here referred to is that which commences with the first indications of distinct physical decay, of which the earliest that we can recognize (in persons who are not cut off by special diseases) is perhaps the tendency to atheromatous change in the arteries. The earliest development of this symptom varies very considerably in date; but whenever it occurs it is a plain warning that a new set of vital conditions has arisen; and especially notable is its connection with the characters of the neuralgic affections which take their rise after its commencement. The period of declining life is pre-eminently the time for *severe and intractable* Neuralgias. Very few patients indeed are ever permanently cured, who are first attacked with Neuralgia after they have entered upon what may be called the "degenerative" period of existence.

Perhaps a separate heading should be reserved for those Neuralgias which are the heralds of locomotor ataxy. But they seem naturally to fall under the present class, although the nervous degeneration which produces them is chiefly in the direction of sclerosis. The character of these pains is fully described in the article on Locomotor Ataxy.

5. *Neuralgias which are immediately excited by anæmia or mal-nutrition.*—Of the neuralgic affections which can be ranked within this group, the sole characteristic worthy of note here is the circumstances in which they arise. It would seem that conditions of anæmia and mal-nutrition simply aggravate the tendencies of existing weak portions of the nervous system to be affected with pain; just as they notoriously do aggravate lurking tendencies to convulsion and spasms.

(B) We come now to the consideration of local varieties of Neuralgia. The primary subdivision of these may be made as follows:—

(I.) Superficial Neuralgias. (II.) Visceral Neuralgias. The superficial Neuralgias may be subdivided thus:—

(*a*) Neuralgia of the fifth (trifacial or trigeminal) nerve.
(*b*) Cervico-occipital Neuralgia.
(*c*) Cervico-brachial Neuralgia.
(*d*) Intercostal Neuralgia.
(*e*) Lumbo-abdominal Neuralgia.
(*f*) Crural Neuralgia.
(*g*) Sciatic Neuralgia.

This classification is taken from Valleix, and appears to me substantially correct.

(*a*) The most important group of Neuralgias are those of the fifth cranial nerve.

Neuralgia of the fifth nerve always exhibits itself with especial violence in certain foci, which Valleix was the first to define with accuracy. These foci are always in points where the nerve becomes more superficial, either in turning out of a bony canal, or in penetrating fasciæ. In the ophthalmic division of the nerve the following possible foci are noticeable: (1) the *supra-orbital*, at the notch of that name, or a little higher in the course of the frontal nerve; (2) the *palpebral*, in the upper eyelid; (3) the *nasal*, at the point of emergence of the long nasal branch, at the junction of the nasal bone with the cartilage; (4) the *ocular*, a somewhat indefinite focus within the *globe* of the eye; (5) the *trochlear*, at the inner angle of the orbit.

In the superior maxillary division the following foci may be found: (1) the *infra-orbital*, corresponding to the emergence of the nerve of that name from its bony canal; (2) the *malar*, on the most prominent portion of the malar bone; (3) a vague and indeterminate focus, somewhere on the line of the gums of the upper jaw; (4) the superior *labial* point, a vague and not often an important focus; (5) the *palatine* point, rarely observed, but in some recorded cases the seat of intolerable pain.

In the inferior maxillary division the foci are:—(1) the *temporal*, a point on the auriculo-temporal branch, a little in front of the ear; (2) the *inferior dental* point, opposite the emergence of the nerve of that name; (3) the *lingual* point (not a common one) on the side of the tongue; (4) an *inferior labial* point, one rarely met with.

Besides these foci in relation with distinct branches of the trigeminus, there is

one of especial frequency, which corresponds to the *inosculation of various branches.* This is the *parietal* point, situated a little above the parietal eminence. It is small in size; the point of the little finger would cover it. It is the commonest focus of all.

Neuralgia of the fifth nerve may attack any one, or all three of the divisions; the latter event is comparatively rare.[1] The most common is the case of its limitation to the ophthalmic division, and incomparably the most frequent foci of the pain are the *supra-orbital* and *parietal* points.

The most common of all the varieties of trigeminal Neuralgia is Migraine, or sick-headache. This is an affection which is entirely independent of digestive disturbances, in its primary origin, though it may be aggravated by their occurrence. It almost always first attacks individuals at some time during the period of bodily development. Under the influences proper to this vital epoch, and often of a further debility induced by precocious straining of the mental powers, the patient begins to suffer headache after any unusual fatigue or excitement, sometimes without any distinct cause of this kind. The unilateral character of this pain is not always detected at first; but as the attacks increase in frequency and severity, it becomes obvious that the pain is limited to the supra-orbital, and sometimes to the ocular branches of the ophthalmic division of the fifth nerve of one side. In very rare cases, however, as in all forms of Neuralgia, the nerves of both sides may be affected. If the pain lasts for any considerable time, nausea, and at length vomiting, are induced. This is followed at the moment by the increase of the severity of the pain; but from this point the violence of the affection begins to subside, and the patient usually falls asleep. The history of the attacks negatives the idea that the vomiting is ordinarily remedial. This symptom merely indicates the lowest point of nervous depression; but it may happen that a quantity of food which has been incautiously taken, lying, as it does, undigested in the stomach, may of itself greatly aggravate the Neuralgia, by irritation transmitted to the medulla oblongata. In such a case vomiting may directly relieve the nerve-pain. When the patient awakes from sleep, the active pain is gone. But it is a common occurrence, indeed it always happens when the Neuralgia has lasted a certain length of time, that a *tender* condition of the superficial parts remains for some hours, perhaps for a day or two. This tenderness is usually somewhat diffused, and not limited with accuracy to the foci of greatest pains during the attacks.

Sick-headache is not uncommonly ushered in by sighing, yawning, and *shuddering*—symptoms which remind us of the prodromata of some graver neuroses, to which it is probably related by hereditary descent.

Another variety of trigeminal Neuralgia which infests the period of bodily development is that known as *clavus hystericus; clavus* from the fact that the pain is at once severe, and limited to one or two small definite points, as if a nail or nails had been driven into the skull. These points correspond either to the *supra-orbital* or the *parietal;* sometimes both these are the seat of the pain. But for the greater limitation of the painful area in clavus, that affection would scarcely differ from migraine, for the former is also accompanied, when the pain continues long enough, with nausea and vomiting. The adjective *hystericus* is an improper and inadequate definition of the circumstances under which clavus arises. The truth is that the subjects of it are usually females who are passing through the trying period of bodily development; but there is no evidence to show that uterine disorders give any special bias towards this complaint. Both migraine and clavus are often met with in persons who have long passed the period of bodily development. But their first attacks have nearly always occurred during that period of life.

The adult or middle period of life is not, according to my experience, fruitful in *first attacks* of trigeminal Neuralgia. But when the neuralgic tendency has once been set up, there are many circumstances of middle-adult life which tend to recall it. Over-exertion of the mind is one of the most frequent; more especially when this is accompanied by anxiety and worry; indeed the latter is a more powerful cause than the former. In women, the exhaustion of hemorrhage at parturition, or of menorrhagia, and also the depression produced by over-lactation, are frequent causes of the recurrence of a migraine or a clavus to which they had been subject when young. The middle period of life is also most obnoxious, on the whole, to severe mental shocks, and also to severe bodily accident, of a kind to produce damage to the central nervous system. Special mention ought to be made in the case of women, of the disturbing influences of the great series of changes which close the middle period of their life—viz. the involution of the sexual organs. This is doubtless a very frequent cause of the resuscitation of a tendency to facial Neu-

[1] It is with much diffidence that I make this statement, as it is opposed to the opinion of Valleix. But my own experience is very positive on the matter; and, besides, it appears to me that Valleix's definition of Neuralgia, which I cannot accept as sufficiently expansive, accounts for his views.

ralgia which had lain dormant, perhaps, for many years.

It is, however, the final or degenerative period of life which produces the most formidable varieties of facial Neuralgia. Neuralgias of the fifth which have previously attacked an individual, may recur at this time of life without any special character except a certain increase of severity and obstinacy. But trigeminal Neuralgias which now occur for the first time are usually intensely severe and utterly incurable. These cases correspond with the affection named by Trousseau "tic épileptiforme," and it is of them, doubtless, that Romberg is speaking, when he says that the true Neuralgias of the fifth rarely occur before the fortieth year of life. These affections are distinguished by the intense severity of the pain, the lightning-like suddenness of its onset, and the almost total impossibility of effecting more than the most temporary improvement in the symptoms. But they are also distinguished by another circumstance which too often escapes attention; namely, they are almost invariably connected with a family taint of insanity, and very often with strong melancholy and suicidal tendencies in the patient himself, which do not depend on, nor are commensurate in their development with, the intensity of the pain which he suffers. They are further remarkable for the frequency with which they are attended with two special complications—viz. muscular spasms, and the formation of exquisitely tender points, the least pressure on which is enough to cause the most violent agony. Often, a mere breath of wind impinging on them will produce a like effect. The history of these cases is most wretched; the unfortunate patient may survive for years before he completely succumbs to exhaustion; yet every hour of his life is a misery. The act of masticating usually causes intolerable darts of agony, and nutrition is often obliged to be kept up by liquids. If mere broth and slop diet be adhered to, there is probably under-nutrition, which aggravates the Neuralgia. And if, as often happens, the patient flies to drink as a relief, that again hastens the degeneration of the nervous centres, and renders the case more hopeless of cure than ever.

(*b*) *Cervico-occipital Neuralgia.*—As Valleix has remarked, there are several nerves (in fact the posterior branches of all the first four spinal pairs) which are more or less capable of being the seat of this affection. But amongst them all there is none comparable to the great occipital, which arises from the second spinal pair, for the frequency and importance of its neuralgic affections. This nerve sends branches to the whole occipital and the posterior parietal region. On the other hand, the second and third spinal nerves help to make up the superficial cervical branch of the cervical plexus, which is distributed to the triangle between the jaw, the median lines of the neck, and the edge of the sterno-mastoid, and those to the lower part of the cheek. Then there is the auricular branch, which starts from the same two pairs, and supplies the face, the parotid region, and the back of the external ear. Then, the small occipital, distributed to the ear and to the occiput. And finally there are the superficial descending branches of the plexus. These, altogether, are the nerves which, at various points, where they become more superficial, form the foci of cervico-occipital Neuralgia.

The most typical example of this form of Neuralgia which has fallen under my own notice, occurred (after exposure to cold wind) in a lady about sixty years of age, who had all her life been subject to neuralgic headache, approaching the type of migraine, and who came of a family in which insanity, apoplexy, and other grave neuroses had been frequent. The pain centred very decidedly in a focus corresponding to the occipital triangle of the neck. It occurred at irregular intervals, and in very severe paroxysms, and was entirely unaffected by any remedies, till *blistering* was tried, when it yielded at once. About twelve months later this patient suffered a severe hemiplegic attack of paralysis.

The tendency, however, of cervico-occipital Neuralgias, is certainly to spread towards the lower portions of the face, as observed by Valleix; in this case they become, sometimes, undistinguishable from Neuralgias of the third branch of the fifth. In the early stages of the disease, if the physician had been lucky enough to witness them, the true place of origin of the malady would have been easily discernible: at a later date it requires great care, and a very strict interrogation of the patient, to discover the true history of the disease.

Experience is too limited, if I am to judge by my own and that of the standard authors, to allow us to say anything of the conditions, as to age and general nutrition of the organism, which specially favor cervico-occipital Neuralgia. Apparently, however, there is good reason for thinking that the immediately exciting cause of it is most frequently *external cold.* But I am inclined to think also that it is seldom a primary Neuralgia, but occurs usually in subjects who have already experienced other forms.

(*c*) *Cervico-brachial Neuralgia.*—This class includes all the Neuralgias which occur in nerves originating from the brachial plexus, as from the posterior branches of the four lower cervical nerves. The

most important characteristic of the Neuralgias of the upper extremity is the frequency, indeed almost constancy, with which they invade simultaneously or successively several of the nerves which are derived from the lower cervical pairs. The neuralgic affections of the small posterior branches (distributed to the skin of the lower and back part of the neck) are comparatively of slight importance. But the "solidarité," which Valleix so well remarked, between the various branches of the brachial plexus, causes the Neuralgias of the shoulder, the arm, forearm, and hand to be extremely troublesome and severe, owing to the numerous foci of pain which usually exist. Perhaps Valleix's description of these foci is somewhat farciful and over-minute; but the following among those which he mentions I have repeatedly identified: (1) an *axillary* point, corresponding to the brachial plexus itself; (2) a *scapular* point, corresponding to the inferior angle of the scapula—(it is difficult to identify the peccant nerve here: the one to which it apparently corresponds, and to which Valleix refers it, is the sub-scapular; but we are accustomed to think of this as a motor nerve. Still it is certain that pressure on a painful point existing here will often cause acute pain in the nerves of the arm and forearm);—(3) a *shoulder* point, which corresponds to the emergence, through the deltoid muscle, of the superficial fillets of the circumflex; (4) a *median-cephalic* point, at the bend of the elbow, where a branch of the musculo-cutaneous nerve lies immediately behind the median cephalic vein; (5) an *external humeral* point, about three inches above the elbow, on the outer side, corresponding to the emergence of the cutaneous branches which the musculo-spiral gives off as it leaves the groove in the humerus; (6) a *superior ulnar* point, corresponding to the course of the ulnar nerve, between the olecranon and the epi-trochlea; (7) an *inferior ulnar* point, where the nerve passes in front of the annular ligament of the wrist; (8) a *radial* point, making the place where the radial nerve becomes superficial at the lower and external aspect of the forearm. Besides these foci, there are sometimes, but more rarely, painful points developed by the side of the lower cervical vertebræ, corresponding to the posterior branches of the lower cervical pairs.

The most common seat of brachial Neuralgia in my experience has been the ulnar nerve; the superior and inferior points above mentioned being the foci of great intensity; an axillary point has also been developed in one or two instances which I have seen. Rarely, however, does the Neuralgia remain limited to the ulnar nerve; in the majority of cases it soon spreads to other nerves which emanate from the plexus. A very common seat of Neuralgia is also the shoulder, the affected nerves being the cutaneous branches of the circumflex. I am inclined to think, also, that affections of the musculo-spiral and of the radial near the wrist are rather common, and have found them extremely obstinate and difficult to deal with. One case has recently been under my care in which the foci of greatest intensity of pain were an external humeral, and a radial point; but beside this there was an exquisitely painful scapular point. In another instance, the pain commenced in an external humeral and a radial focus; but subsequently the shoulder branches of the circumflex became involved. A most plentiful crop of herpes was an intercurrent phenomenon in this case.

Median cephalic Neuralgia is an affection which used to be comparatively common in the days when phlebotomy was in fashion, the nerve being occasionally wounded in the operation. I have only seen it in connection with this cause; that is to say, as a well-marked affection. One such instance has been under my care. But a slight degree of it is not uncommon, as a secondary symptom in Neuralgia affecting other nerves. The traumatic form is excessively obstinate.

In the Neuralgias of the arm we begin to recognize the etiological characteristic which distinguishes most of the neuralgic affections of the limbs; namely, the frequency with which they are aggravated, and especially with which they are kept up and revived, when apparently dying out, by *muscular movements*. In the case above referred to, of Neuralgia of the sub-scapular, musculo-spiral (cutaneous branches), and radial, the act of playing on the piano for half an hour immediately revived the pains in fullest force, when convalescence had apparently been almost established.

The liability of particular nerves in the upper extremity to Neuralgia, from *external injuries*, requires a few words. The nerve which is probably most exposed to this is the ulnar. Blows on what is vulgarly called the funny bone are not uncommon exciting causes of the affection in predisposed persons: and cutting wounds of the ulnar a little above the wrist are rather frequent causes. The deltoid branches of the circumflex, and the humeral cutaneous branches of the musculo-spiral, are much exposed to injury. The radial nerve near the wrist is very much exposed both to bruises and to cutting wounds. So far as I know it is only when a nerve-trunk of some size is injured that Neuralgia is a probable result. Wounds of the small nervous branches in the fingers, for instance, are very seldom followed by Neuralgia. I

have no statistics to guide me as to the effect of long-continued *irritation* applied to one of those small peripheral branches; but it is probable that that might be more capable of inducing Neuralgia. As far as my own experience goes, however, it would appear that a more common result is *convulsion* of some kind, from reflex irritation of the cord.

(*d*) *Dorso-intercostal Neuralgia.* — This form of Neuralgia has of late years assumed a position of much interest, in consequence chiefly of its rather frequent association with unilateral herpes, a circumstance which has considerably helped to elucidate the pathology of the latter disease.

This disease is surrounded with considerable diagnostic difficulties. Some of these will be discussed under the head of Diagnosis in part; but a few words must be given to them here. The disorder with which it is especially liable to be confounded is that for which Dr. Inman invented the term Myalgia, and which is represented in different localities by the affections called in old-fashioned phrase pleurodynia, lumbago, and (more generally) by the very inaccurate term muscular rheumatism (there being no reliable evidence whatever to connect it specially with the rheumatic diathesis). The principal feature by which dorso-intercostal Neuralgia can be separated from myalgia is its history; viz. its non-dependence, or much less dependence, on *excessive or long-continued local muscular action* than the latter complaint exhibits. There is also a more marked *intermittence* in the neuralgic affections. Finally, though this only applies to a limited number of cases, the intercurrence of *herpes* is a decided diagnostic of the neuralgic character of the disease.

Dorso-intercostal Neuralgia is an affection of certain of the dorsal nerves. These nerves divide immediately after their emergence from the intervertebral foramina into a posterior and an anterior branch. The former sends filaments which pierce the muscles, to be distributed to the skin of the back; the latter, forming the intercostal nerve, follows the intercostal space. Immediately after their commencement the intercostal nerves communicate with the corresponding ganglia of the sympathetic. Proceeding outwards, they at first lie between the pleura and intercostal muscles; towards the angles of the ribs they pass between the two layers of intercostal muscles, and, after giving branches to the latter, give off their large superficial branches. In the case of the seventh, eighth, and ninth intercostal nerves, which are those chiefly liable to Neuralgia, the superficial branch is given off about midway between the spine and the sternum. The final point of division, at which superficial filets come off, in all the eight lower intercostal nerves, is nearer to the sternum, and is progressively nearer to the latter in each progressive space downwards. There are thus, as Valleix observes, three points of division: 1, at the inter-vertebral foramen; 2, midway in the intercostal space; 3, near to the sternum. And there are three sets of superficial branches (reckoning the posterior primary division) which make their way towards the surface near these points.

In one of its forms, intercostal Neuralgia is one of the commonest of all neuralgic affections. I refer to the pain beneath the left mamma, which women with neuralgic tendencies so often experience, chiefly in consequence of over-lactation, but also from exhaustion caused by menorrhagia, and especially from the concurrence of this cause with the preceding one. Some care must be taken to distinguish this from the mere myalgic pain, which is produced by over-working the pectoral muscles in proportion to the existing state of their nutrition, and also by the vague conditions grouped under the name "Hysteria." The latter sort of pain is more diffuse in extent, and less markedly intermittent, than Neuralgia, and its history is different: and the effect of *rest* is far more marked in the former than in the latter.

It is only of recent years that the Neuralgia which had often been observed to attend herpes zoster has been even thought of as essentially connected with the latter disease. It is to M. Notta that some of the earliest observations leading to the latter view are to be attributed. But the matter was much more fully discussed by M. Barensprung, in a paper published in 1861.[1] This author showed the absolute universality with which unilateral herpes, wherever developed, closely followed the distribution of some superficial sensory nerve, and gave reasons, which will be discussed hereafter, for supposing that the disease originates in the ganglia of the posterior roots, and that the irritation spread thence to the posterior roots in the cord, causing reflex Neuralgia. This theory will be discussed further. Meanwhile, it seems to be established, by multiplied researches, that though unilateral herpes may, and often does, occur without Neuralgia, and Neuralgia without herpes, the concurrence of the two is due to a mere extension of the original disease, which is a nervous one.

In young persons zoster is not often attended with severe *Neuralgia*, but a curious half-paretic state of the skin, in which

[1] Annalen der Charité Krankenhauses zür Berlin, ix. 2, p. 40: Brit. and For. Med. Rev., January, 1862.

numbness is mixed with formication, or with a sensation as of boiling water under the skin, precedes the outbreak of the eruption by some hours, or even a day or two. Painless herpes is commonest in youth. From the age of puberty to the end of life the tendency of herpes to be complicated with Neuralgia becomes progressively stronger. The course of events is different in different cases, however. Usually, in adult and later life the symptoms *commence* with a more or less violent attack of neuralgic pain, which is succeeded, and for the time usually (though not always) displaced, by the herpetic eruption. This latter runs its course, and after its disappearance the Neuralgia very commonly returns again. In old people the *after*-Neuralgia is often distressingly severe, and most rebellious to treatment. Six weeks or two months is quite a common period for it to last, and in some aged persons it has been known to fix itself permanently, and cease only with life. In elderly subjects a further complication sometimes occurs. The herpetic vesicles leave obstinate and most painful ulcers behind them, which refuse to heal, and worry the patient frightfully, the merest breath of air upon them sufficing to cause agonizing darts of neuralgic pain. I have known one patient distinctly killed by the exhausting agony thus caused.

The foci of pain in intercostal Neuralgia are always found in one or more of the points, already mentioned, at which sensory twigs become superficial. In long-standing cases acutely tender spots are developed; not unfrequently the most decided of these are where they are too seldom sought for, namely, opposite the emergence from the inter-vertebral foramina.

(*e*) *Dorso-lumbar Neuralgia.*—The records of this affection are as yet in a state of considerable confusion. What has been done with any precision towards clearing up the history of the disease, related chiefly to the neuralgic affections of the pelvic organs in women; and to the Neuralgia of the testes in men, which will be treated of in a different place.

The principal foci of dorso-lumbar Neuralgia, when this affects external parts, are the following: (1) the *vertebral* points, corresponding to the posterior branches of the respective nerves; (2) an iliac point, about the middle of the crista ilii; (3) an *abdominal* point, in the hypogastric region; (4) an inguinal point, in the groin near the issue of the spermatic cord, from whence the pain radiates along the latter; (5) a *scrotal* or *labial* point, situated in the scrotum, or in the labium majus.

Such is the description given by Valleix; and as I have seen but few examples of the external forms of dorso-lumbar Neuralgia, I can only rely upon his observation. The few severe cases of this kind of Neuralgia, which I have observed, have been distinguished by foci in the vertebral region, and over the crista ilii; in two of these there were also distinct foci in the spermatic cord and testicle. In one patient there was an apparent focus of pain higher up in the groin also; but this man is a confirmed hypochondriac, and his morbid sensations are so shifting as to be very unreliable in their indications.

(*f*) The next group of Neuralgias which must be described is the *crural*. This, after all, includes very few independent cases. There are very few primary Neuralgias of the crural nerve; Valliex had only seen two in his very large experience, and I cannot say that I have seen any. Neuralgia of the crural nerve is almost always a secondary affection, arising in the course of Neuralgia, which primarily showed itself in the external pudic branch from the plexus.

(*g*) The last and one of the most important and numerous groups of external Neuralgias are the *femoro-popliteal, or Sciatic.*

Sciatica is a disease from which youth is comparatively exempt. Valliex had collected 124 cases; and in not one was the patient below the age of seventeen; only 4 were below twenty. In the next decade there were 22; in the next 30; and the largest number of cases, 35, were between the ages of forty and fifty. This completely tallies with my own experience; and seems to favor the suspicion which I have formed, that the pressure exerted on the nerve in locomotion and in sitting is one principal cause of the great liability to Neuralgia which distinguishes the sciatic nerve; and this idea seems to be favored by the further fact elicited by Valliex, that from thirty years onward the number of male is greatly higher than that of female sciatic patients.

There are three very distinct varieties of the disease, however, according to my experience. The first variety is obscure in its origin, but may be said, in general terms, to be connected with a strongly marked nervous temperament, which is indicated in the female by a tendency to hysteria, and in the male by an abnormal sensibility to nervous impressions. The subjects of this variety of sciatica are mostly below the age of forty, and have generally been liable to other forms of Neuralgia; the actual attack of sciatica is excited by some bodily fatigue or mental distress which, on other occasions, has produced sick-headache, or intercostal Neuralgia, &c. Very many of these patients are anæmic. The greater number of them are females, and in many (whether as cause or effect) there is decided amenorrhœa, and sometimes chlorosis. In this variety the pain, though chiefly affecting the sciatic nerve and its branches, is apt

secondarily to invade some of the nerves which issue from the lumbar plexus. I cannot avoid the suspicion, though the proof is most difficult, that the affection not unfrequently depends on, or is much aggravated by, an excited condition of the sexual apparatus; certainly, I have observed it with marked frequency in women who remain single long after the marriageable age, and in the case of several male patients there has been either the certainty or a strong suspicion of venereal excess. The actual outbreak of pain is generally sudden, but in many instances there has been a tendency to numbness, or abnormal sensations, in the skin of the back part of the thigh, or in some part in the course of the branches of the nerve for some time previously. Like all forms of sciatica, this affection is usually obstinate, and requires assiduous and sometimes prolonged treatment for its removal; but it is incomparably more manageable than other varieties.

The second variety of sciatica occurs for the most part in middle-aged or old persons who have long been subject to excessive muscular exertion, or have been much exposed to cold, and especially *damp* cold, or who have been subjected to both of these kinds of evil influences. One must include also, I think, in this group, a certain number of patients whose age need not be so advanced, but who have been liable, along with depressing influences of a constitutional kind, to prolonged pressure on the nerve from the habitual maintenance of the sitting posture, in their business, for many hours together.

The patients who suffer from this second variety of sciatica are mostly, as already said, of middle age or more; but this statement must be understood to be made in the comparative sense which refers rather to the vital condition of the individual than to the mere lapse of years. Many of them have hair which is prematurely gray; and in some the existence of rigid arteries, together with *arcus senilis*, completes the picture of organic degeneration. In particular cases where depressing influences have been at work for a long time, or unusually active, their appearances rectify the impression we should otherwise receive from learning the nominal age of an individual; this is especially the case with persons who have for a long time drunk to excess. I am at a loss to know how Valleix and many others can have overlooked the frequent occurrence of this type of constitution among the most numerous group of sciatic patients—those between thirty and fifty years of age; unless, indeed, we suppose that many of their "robust" patients were so fresh in color and possessed such good muscular strength as to lead the physician to ignore the far more significant vital indications which are given by the above-mentioned appearances.

A prominent feature in this variety of sciatica is its great obstinacy and intractability. Another equally marked is the development, around one or more foci of severest pain, of spots which are permanently and intensely tender, and the slightest pressure on which is sufficient to renew the agony of acute pain: this development of tender points is far less marked in the preceding form of the disease. The places which are specially apt to present this phenomenon are as follows:—(1) A series or line of points, representing the cutaneous emergence of the posterior branches, which reaches from the lower end of the sacrum up to the crista ilii. (2) A point opposite the emergence of the great and small sciatic nerves from the pelvis. (3) A point opposite the cutaneous emergence of the ascending branches from the small sciatic which runs up towards the crista ilii. (4) Several points at the posterior aspect of the thigh, corresponding with the cutaneous emergence of the filets of the crural branch. (5) A *fibular* point, at the head of the fibula, corresponding to the division of the external popliteal. (6) An *external malleolar*, behind the outer ankle. (7) An *internal malleolar*.

Another circumstance which distinguishes the form of sciatica which we are considering, is the degree in which (above all other forms of Neuralgia) it involves paralysis. By far the largest part of the whole *motor*-nervous supply for the limbs passes through the trunk of the great sciatic; it might therefore be naturally expected that a strong affection of the sensory portion of the nerve would in a reflex manner, produce some powerful effect on the motor element. This effect is the most frequently in the direction of paralysis. Complete palsy is rare, but in a large number of cases which have lasted some time there can be no doubt that there is a positive and very considerable loss of motor power, independently of any effect which may be produced by wasting of muscles. It is of course necessary to avoid the fallacy which might be produced by neglecting to observe whether movement was merely restricted in consequence of its *painfulness*.

Anæsthesia is also a common complication of sciatica, far commoner, as I venture to think, than it has been represented either by Valleix or Notta. It is necessary, however, to be explicit on this point. In the early stages both of this form of sciatica and of the milder varieties previously described, there is almost always partial numbness of the skin previous to the first outbreak of neuralgic pain, and during the intervals between the attacks.

By degrees this is exchanged, in the milder form, for a generally diffused hyperæsthesia around the foci of neuralgic pain, while other portions of the limb may still remain anæsthetic. In the severer forms it sometimes happens that, besides an intense hyperæsthesia of the skin over the painful foci, there is diffused hyperæsthesia over a greater part or the whole of the surface of the limb. But it is important to remark that both in the anæsthetic and the hyperæsthetic conditions (so-called), the *tactile sensibility is very much diminished.* I have made a great many examinations of painful limbs in sciatica, and have never failed to find (with the compass points) that the power of distinctive perception was very decidedly lowered.

Convulsive movements of muscles are met with in a moderate proportion of the cases of severe sciatica of middle and advanced life, in which affection they are entirely involuntary. They differ from certain spasmodic movements not unfrequently observed in the milder form (and especially in hysteric women), for these are more connected with defective volition, and are, in truth, not perfectly involuntary. In several cases of inveterate sciatica I have seen violent spasmodic flexures of the leg upon the thigh. Cramps of particular muscles are occasionally met with. I have seen the flexors of all the toes of the affected limb violently cramped; and in one case the patient was troubled with severe cramps of the gastrocnemius. It is chiefly at night, and especially when the patient is just falling asleep, that this kind of affection is apt to occur.

A third variety of sciatica is the rather uncommon one (so far as my experience goes) in which inflammation of the tissues around the nerve is the primary affection, and the Neuralgia is a mere secondary effect, from mechanical pressure on the nerve, which however is, apparently, not itself inflamed. I believe that these cases are sometimes caused by syphilis, and sometimes by rheumatism. It need hardly be said that this affection is essentially different, and requires a different treatment from Neuralgias in which the disturbances originate in the nervous system.

(II.) *Visceral Neuralgias.* — This most important class of diseases still remains very much unknown; but it is constantly assuming a greater consequence. The Neuralgias of viscera, of which anything can with confidence be said, are the following:—(1) Cardiac, (2) Hepatic, (3) Gastric, (4) Peri-uterine (including ovarian), (5) Testicular, (6) Renal.

It is, however, unnecessary to describe the clinical history of these disorders here, since they will be treated of under the headings of the morbid affections of the particular organs which they infest.

COMPLICATIONS.—This part of our subject is of the greatest interest, and the facts regarding it are, to a considerable extent, of recent discovery. If we turn to the excellent treatises of Valleix and Romberg, which appeared about a quarter of a century ago, we find a very inadequate importance assigned to the secondary affections which occur in Neuralgia. The convulsive movements of the facial muscles which occur in the severer forms of *tic douloureux* could not fail, of course, to attract attention even from the earlier times. Of the functions of special sense Valleix only mentioned *hearing* as liable to be affected. Injection of the conjunctiva he spoke of as if it were a rare phenomenon in trigeminal Neuralgia. He did not mention modifications of nutrition at all, except those of the hair; and of modifications of secretion he only enumerated lachrymation, mucous flux from the nostril, and salivation as occasional phenomena. Of disturbances of the stomach he took a more appreciative view; and he mentioned, as a remarkable fact, that he never knew facial Neuralgia caused by gastric disturbance, but had frequently observed the latter affection to occur in the course of a neuralgic attack, and apparently as the consequence of it. He gives no pathological explanation of the connection between them.

It is to M. Notta[1] that we owe the first scientific treatment of this subject of the complications of Neuralgia. The importance of these secondary affections is particularly brought out by this author in his remarks on trigeminal Neuralgia, of which he analyzes 128 cases. As regards special senses, he states that the retina was completely, or almost completely, paralyzed in ten cases, and in nine others vision was interfered with; partly, probably, from impaired function of the retina, but partly, also, from dilatation of the pupil, or other functional derangement independent of the optic nerve. The sense of hearing was impaired in four cases. The sense of taste was perverted in one case, and abolished in another. As regards secretion: Lachrymation was observed in sixty-one cases, or nearly half the total number. Nasal secretion was repressed in one case; in ten others it was increased on the affected side. Unilateral sweating is spoken of more doubtfully, but is said to be probably present in a considerable number of cases. In eight instances there was decided unilateral *redness* of the face, and five times this was attended with noticeable *tumefaction.* In one case the unilateral redness and tumefaction persisted, and were, in fact, accompanied by a general hypertrophy of the tissues. *Dila-*

[1] Archives Générales de Médecine, 1854.

tation of the conjunctival vessels was observed in thirty-four cases. Nutrition was affected as follows: In four cases there was unilateral hypertrophy of the tissues; in two, the hair was hypertrophied at the ends, and in several other cases it was observed to fall off or to turn gray. The tongue was greatly tumefied in one case. Muscular contractions, on the affected side, were noted in fifty-two cases: of these, in thirteen, the contractions were in the muscles of the lip and nostril; in ten, there was tremor of the eyelid; in a great number many muscles were simultaneously affected. *Permanent tonic spasm* (not due to photophobia) was observed in the eyelid in four cases; in the muscles of mastication, four times; in the muscles of the external ear, once. *Paralysis* affected the motor oculi, causing prolapse of the upper eyelid, in six cases; in half of these, there was also outward squint. In two instances, the facial muscles were paralyzed in a purely reflex manner. The pupil was dilated in three cases, and contracted in two others, without any impairment of sight; in three others it was dilated, with considerable diminution of visual power. Finally, with regard to common sensibility,—M. Notta reports three cases in which *anæsthesia* was observed. *Hyperæsthesia* of the surface only occurred in the latter stages of the disease.

Various other observers have added to this list of the secondary affections which may occur in facial Neuralgia the following: Iritis, glaucoma, corneal clouding, and even ulceration; periostitis, unilateral furring of the tongue, herpes unilateralis, &c.

All the above complications of facial Neuralgia, excepting glaucoma, have been under my own observation, and most of them I have seen in a great many cases. Moreover, my own attention had been called independently to the subject by my own unlucky personal experience. I began, at the age of about fourteen, to suffer from attacks of unilateral facial Neuralgia in the right side (chiefly supra-orbital), which very soon assumed the type of severe migraine, such as it has already been described. A year or two later, the pains being at this time severe and frequent, there occurred a painful thickening and tumefaction of the periosteum round the brow, and also the formation of one or two dense white patches on the cornea, in the centre of which small phlyctenular ulcers appeared. About the same time, probably, there occurred a great thickening of the fibrous tissue, surrounding the upper end of the nasal duct, which caused a dense stricture of that canal. Some years later, when the attacks had become much less frequent, they recurred with great severity during the prostration brought on by choleraic diarrhœa. I then first noticed that the hair of the eyebrow was whitened opposite the supra-orbital notch, and that gray hairs were thickly strewn over the right side of the head for some time after the attack; and this phenomenon has occurred after every severe attack since that time. It only lasts in intensity for a few days, and the color soon becomes partially restored to its original tint, but *without any falling off of the hair.* The latter fact seems at first difficult of belief; but I have most closely observed the phenomenon, and have since witnessed the same thing in several patients, both of my own and other practitioners. Another nutritive modification which I have seen in my own case is the formation of a dense epithelial fur on one-half of the tongue.

There is another complication which, so far as I am aware, was first identified by myself as having a definite relation to facial Neuralgia: viz. erysipelatoid inflammation of the tissues to which the painful nerve is distributed. Some years ago I was much surprised at observing, in a woman aged thirty-two, a patient of the Chelsea Dispensary, a most acute attack of unilateral erysipelas of the face and head, supervening on some severe and frequently recurring attacks of Neuralgia, which affected all three divisions of the trigeminus, but was most violent in the branches of the ophthalmic division. On the recurrence of the erysipelas, the acute pain subsided, but the most intense tenderness remained for some days, and pressure anywhere in the track of the nerves would re-excite a momentary spasm of pain. Since that time I have been constantly on the look-out for similar cases, and have observed a good many either in my own practice or that of others. In several instances I have seen Neuralgia of the fifth actually terminate in an affection undistinguishable from ordinary erysipelas, limited to the painful parts: in four of these cases it was limited to the side of the nose, the infra-orbital and frontal regions. But the facts bearing on a connection between facial neuralgia and erysipelas are by no means limited to this. In twenty-two cases which have come under my care, of patients suffering either from typical facial tic, from migraine, or from clavus hystericus, I have discovered, by inquiry, the existence of a strong tendency to erysipelatoid inflammation of the parts then affected with Neuralgia. An attack of erysipelas would be brought about in these patients, by the most trivial causes, by a slight exposure to cold winds, or, on the other hand, by unusually depressing fatigue or emotion. The majority of these patients give me a

family history which showed a marked inherited disposition to neurotic affections, a circumstance which, as we shall hereafter see, is of importance.

Perhaps the most striking of all the cases which have come under my notice is one which was obligingly sent to me by Mr. Ernest Hart, and which I have already published[1] in detail. The exciting cause of the whole train of phenomena was apparently fright from an accident which there was no reason to suppose inflicted any direct physical injury. The sequence of events was: (1) abrupt cessation of menses, with hysteric depression; (2) severe neuralgia of the first and second divisions of the fifth, quickly producing iritis, with effusion of lymph; (3) erysipelas, exactly limited to the skin of the painful parts, and as it were supplying the Neuralgia.

The concurrence of *iritis* with the erysipelas, in this case, is a most interesting fact, as showing a general tendency to paralysis of the vessels in the affected district, which will be much dwelt on in the section on pathology. The connection of iritis with Neuralgia is a subject which, though only quite recently mooted, already assumes an extraordinary magnitude, and may yet lead to pathological and therapeutical discoveries of first-rate importance. For my own part I do not hesitate to express the belief that the very vague and ill-defined disease known, in common phrase, as "Rheumatic iritis," is destined to be almost, if not quite, banished to limbo; for, that careful observation will prove the cases so denominated to be nearly all capable of classification as "Neuralgic iritis."

The symptoms which characterize this malady are as follows. The patient first of all complains (usually after exposure to cold wind, or damp, or both) of pain round the orbit, which gradually increases to a pitch of great severity, but which exhibits marked intermissions or at least remissions. The vessels of the conjunctiva, but more particularly of the sclerotic, then become injected. Last of all the iris itself becomes cloudy, and, in severe cases, actual deposits of lymph take place. I cannot hesitate to say, from careful inquiries into the past history of such patients, that this kind of affection occurs quite as frequently in persons who have never shown any distinctive rheumatic tendencies as in those who have. On the other hand, there is nearly always a recognizable history of tendencies towards neuralgic affections of one sort or another. And indeed with regard to the whole series of so-called chronic rheumatic affections of fibrous membranes, it must be remembered that there is reason to doubt whether, on careful analysis, their local symptoms can be grouped into any intelligible unity. It seems far more likely that, as the consequences of spinal irritation become more perfectly known, the whole group of such affections will be resolved into particular cases of centric nervous irritation.

And finally it may be noted that this variety of iritis is greatly more amenable to the influence of quinine than to that of any other remedy; in fact, beyond the use of belladonna to prevent pupillary adhesion, no other treatment is required.

Herpes, as a complication of dorso-intercostal Neuralgia, has been already referred to. Although not so commonly, it may probably attend Neuralgia of any superficial nerve. For instance, the occurrence of a regular facial herpes zoster has been considered by many authors not so much a rarity as an impossibility. But various single cases have been recorded by individual observers of late years; and in a very valuable paper on unilateral herpes in the London Hospital Reports for 1866, Mr. Jonathan Hutchinson reckons up fourteen cases, including several which came under his own observation: some of them are mentioned to have been accompanied by Neuralgia of the fifth. In one of these cases, in which the Neuralgia was particularly severe, the herpetic vesicles were followed by ulcers, which left considerable scars on the forehead. I have myself seen herpes the attendant of two cases of cervico-brachial Neuralgia, in one of which the ulcerations following the vesicles were a cause of severe suffering; and in one instance of sciatica in my practice there occurred enormous vesicles, or rather bullæ, on the back of the calf, which formed most troublesome and exquisitely painful ulcers. Barensprung[1] records a similar case, in which the irritation of the sciatic was secondary to psoas abscess.

The tendency of deeper tissues to be affected in an inflammatory manner as a consequence of Neuralgia, which is specially shown in the cases of neuralgic iritis, receives every-day illustration. In fact, the painful points so universally observed in severe or inveterate cases are probably produced by a subacute inflammation, first of the fibrous membranes (periosteum or fascia) in contact with the nerve at points where it comes out from a deeper to a more superficial position, and further (in some cases) to all the subcutaneous tissues for an inch or two round. In one of the cases of cervico-brachial Neuralgia already referred to, a bright red painful spot, as large as half-a-crown, appeared on the outer side of the arm; there was dense thickening of tissues in

[1] Lancet, 1866, vol. ii. p. 548.

[1] Loc. cit.

this situation, and the resemblance to an inflamed syphilitic node was remarkable. The neuralgic origin was, however, unmistakable. Among the cases of facial herpes collected by Hutchinson, there are several in which serious or even irremediable damage was inflicted on the eye by general inflammation of its tissues.

Diagnosis.—The diagnosis of neuralgic affections from others which may involve pain is, on the whole, not difficult, if we are able to extract from the patient a full account of his history. The essential points for observation are:—1. The situation and direction of the pain, whether this is unilateral, whether it corresponds to the course of a recognizable nerve branch or branches. 2. Whether it is intermittent or markedly remittent. The points of history which are most important are:—1. Whether the patient has suffered Neuralgia before, and if not, whether Neuralgias, or neurotic diseases of any kind, have prevailed in his family. 2. Whether the attack was preceded by nervous disposition, or was ushered in by distinct numbness or tingling. 3. Whether the immediate excitant appeared to be cold or damp or both, or a severe nervous shock, or a direct physical injury. 4. (If the affection has lasted some time) whether there has occurred any development of secondary tender points in the situations where, as above described, they might be expected. 5. Whether the patient has suffered from secondary affections of glands (*e. g.* lachrymation, in the case of facial pain) during the attacks, or of temporary congestion of surfaces (*e. g.* of the conjunctiva) in the same case, or from alterations of epithelium or hair, or herpetic eruptions, or erysipelatoid inflammation of the skin corresponding to the distribution of the affected nerves.

The affirmative answer to any of these questions is, *pro tanto*, in favor of the genuinely neuralgic character of the disorder; and, indeed, the union of features 1 and 2, under the heading of "observation," with one, or still more with two or three, of the "historical" facts, would be pretty well decisive in this sense.

The main source of embarrassment, in difficult cases of diagnosis, is the impossibility which we sometimes encounter of getting a clear history. This is especially apt to occur when we are called to the patient not so much on account of the primary neuralgic affection as because of severe secondary consequences that happen to have arisen. For instance, in a case of severe Neuralgia of the fifth, attended with periosteal inflammation round the orbit, or with intense conjunctivitis, and, it may be, corneitis, or even iritis, the history related is likely enough to lack explicit details of the primary affection. It is necessary to inquire very strictly whether the pain, when it first occurred, was, or was not, accompanied by tenderness on pressure; and whether this simple pain markedly preceded the organic lesions.

Another serious difficulty arises, not unfrequently, in distinguishing between true Neuralgia, and that form of pain which is vaguely called hysteric; and also between the former, and Myalgia not associated with the hysteric diathesis. The great characteristic of true Neuralgia is the limitation of the pain to the course of recognizable branches of nerves, as opposed to the diffused character both of hysteric and neuralgic pains. A history of intense hysteric predisposition may help the diagnosis in some cases, and a history of overwork done by under-nourished muscles may clear it up in others. But hysterical persons may, and sometimes do, suffer from true Neuralgia. And again, it is very common for hysteric patients to develop tender points in certain situations (especially beneath the left mamma, in the epigastrium, and at various situations along the vertebral fossæ which lodge the great muscles of the back), which bear a superficial similarity to the tender points developed in long-standing Neuralgia. The more generalized hyperæsthesia of the skin which usually accompanies these symptoms, when they are due to hysteria, will seldom be observed, however, in true Neuralgia; and the remarkable affections of volition which mostly accompany the hysteric diathesis rarely occur in Neuralgia pure and simple. A means of diagnosis between hysteric hyperæsthesia and the true Neuralgia which I have found most useful is the use of Faradization. It has a strikingly inactive effect in the former, but acts much more slowly, or not at all, in true Neuralgia.

It is almost impossible to lay down rules of diagnosis, in this place, between Neuralgia pure and simple, and that which accidentally occurs from a nerve becoming squeezed, or otherwise damaged, in the progress of tumors or other organic diseases external to it. The reader must be referred to the diagnostic characters mentioned in the treatises on such diseases for the means of distinction.

The neuralgic pains which usher in locomotor ataxy, are highly peculiar, and their diagnosis from ordinary Neuralgia must be learned by studying the article on the former disease.

Prognosis.—The prognosis of Neuralgia is nearly always an uncertain matter. The simplest case is when a clearly malarial history can be made out, and when the blood infection has not lasted too long: here we may expect a speedy cure

by appropriate treatment. The least complicated varieties of traumatic Neuralgia—those in which the irritation is only kept up by some mechanical irritation (*e. g.*, a foreign body lodged, or a tight cicatrix making pressure)—of course offer a good chance of cure by surgical interference. Among the Neuralgias which are more purely of internal origin, those are chiefly to be regarded as benign which occur in young subjects: and next to youth in favorable influence on the prognosis comes the fact of otherwise unbroken health. Neuralgia becomes progressively less curable in each successive decade of life, and more especially after the commencement (at whatever nominal age) of the symptoms of organic degeneration. Very formidable, in all cases, is the fact that the patient's family have been liable either to severe Neuralgias, or to other grave neuroses. And when a patient with such a family history is first attacked with a Neuralgia after he has already entered on the period of organic degeneration, his chances of complete recovery must be reckoned very small. Moreover, such a Neuralgia is not unfrequently the first warning of degeneration of the centres, which will end with softening of the brain.

These are the fundamental points in prognosis. A less essential, but still important, class of momenta are the circumstances of the patient's life; how far, for instance, he is likely to be exposed to the hostile influences of cold, damp, and privation, with the disorders which they tend to engender; and how far there may be unavoidable exposure to the influences of mental distress, or of "the weariness of an objectless life."

PATHOLOGY AND ETIOLOGY.—These two subjects, in the case of Neuralgia, are inextricably mixed; nor is it possible to discuss the one without constant reference to the other. They are so mixed, firstly, because there is no sufficient basis of anatomical fact to support a "pathology," in the ordinary sense; and secondly, because, in addition to the philosophical difficulties which always beset the construction of an etiological system, there are, in the case of Neuralgia, special obstacles to the decision as to what is "cause" and what "effect," arising from the necessity of regarding a neuralgic person as a mere offshoot of a certain family beset with peculiar tendencies, rather than as an individual who forms his own physical destiny by the manner and circumstances of his life.

Of facts tending to elucidate the morbid anatomy of Neuralgia there are very few. This necessarily follows from the rarity with which neuralgic patients die under circumstances which lead to any careful examination of the nerves and nerve-centres. Among the very few recorded cases which show anything positive is the remarkable one related by Romberg.[1] The patient was a victim to the severest form of facial Neuralgia, "of the period of bodily degeneration," such as I have described it. The Gasserian ganglion of the painful nerve was almost destroyed by the pressure of an internal carotid aneurism, the trunk and posterior root of the nerve were completely degenerated, and the atrophic process had extended, in less degree, to the nerve of the opposite side.

This case, alone, of course *proves* nothing as to the general question of the pathology of Neuralgia. But it teaches a notable fact, that the extremity of pain can be suffered in a nerve in which sensation would soon have become extinct by dissolution of the connection between centre and periphery. It is imaginable that a not less real, but less advanced and less coarsely obvious atrophic change may have been present in every case of Neuralgia, even where dissection has failed to reveal anything amiss. It must be remembered that the microscopic study of morbid changes in nerve tissues is even now only in its infancy. It would be vain to occupy a large space in a practical treatise, with disquisitions on a subject at present so obscure as the pathology of Neuralgia; I shall therefore content myself with stating the hypothesis which appears most probable to me, and the mere outline of the reasons which incline me to adopt it.

I think it most probable that in *all* cases of Neuralgia there is either atrophy, or a tendency to it, in the posterior or sensory root of the painful nerve, or in the central gray matter with which it comes in closest connection. The following are the heads of the argument:—

1. Neuralgia is eminently hereditary. It is constantly observed to prevail in particular families, breaking out in successive generations and various individuals. But what is even more important to notice is the fact that these neuralgic families are almost invariably also distinguished by a tendency to the severer neuroses—insanity, cerebral softening, paralysis, epilepsy, hypochondriasis, or an uncontrollable tendency to alcoholic excess; and very often in the various members of the same family we may observe the alternation of all these affections with Neuralgia. [The importance of the hereditary *gouty* diathesis is quite great in some instances.—H.]

2. Such hereditary tendencies in a race seem strongly to suggest a tendency to

[1] Diseases of Nervous System, Syd. Soc. Trans. vol. i.

imperfection in the congenital construction of the central nervous system; so that we may imagine that certain cells and fibres of this system are, in a large proportion of that race, built, as it were, only to live with perfect life for a short term. The weak spot may be in one place in this person, in another place in that.

3. Given such a weak spot, congenitally present, all hostile influences will tell more heavily on it than on the rest of the organs. The depressing influence of cold applied to the periphery, of a wound of the trunk or branches of a nerve, of a severe shock (mental or physical) to the nervous centres generally, or of continued alcoholic excesses, will suffice to throw the imperfectly constructed cells into a state of positive disease, which may end in decided atrophy. Even in the absence of any special external cause, the depressing influence on the nervous centres produced by the great crisis of puberty, child-bearing, the involution of the female organs at the grand climacteric, and still more the partial failure of nutrition which the arterial degeneration of advanced life would cause—any of these may suffice to start the local morbid process.

4. A very weighty argument in favor of the idea that central mischief is a factor in all cases of Neuralgia is the great frequency of complications, such as have been described, in which various nerve-fibres, quite distinct from those which are the seat of pain, and connected with these only through the centre, are secondarily affected.

5. Those cases in which a localized peripheral lesion is the immediate excitant also require for their explanation the assumption of a peculiarity in the individual, as one factor, and that the most important, in the production of the Neuralgia. For of hundreds of persons to whom exactly similar lesions happen every year, not more than two or three, perhaps, experience any Neuralgia; and these two or three will, I believe, be invariably found to belong to neurotic families.

6. The only cases in which the theory of congenital central imperfection appears neither applicable nor necessary are those in which a pressure, ulceration, or other lesion extending from neighboring tissues towards the nerve, maintains a constant depressing centripetal influence which it is not difficult to suppose might impair the vitality of the posterior root, or of the central gray matter.

7. Certain influences, especially that of excessive drinking, which notoriously tends to produce degeneration of the nervous centres, are powerful predisposers to the production of Neuralgia of the inveterate type. Moreover, the descendants of drunkards, among other evidences of an enfeebled nervous organization, are decidedly prone to Neuralgia. So frequently have I made the discovery that neuralgic patients have had drunken parents, that I cannot suppose the coincidence to be accidental.

TREATMENT.—The treatment of Neuralgia may be classified under three heads. The first division includes all remedial measures which are intended to improve the general nutrition, including that of the nervous system, or to remove any vicious condition of the blood which may impair nervous function. The second division includes the narcotic stimulant remedies. The third division comprises all the remedies which are destined to exert a direct influence upon the affected nerve.

1. Constitutional treatment.

(*a*) Under the head of *nutritive* remedies for Neuralgia, by far the most important sub-class is the series of animal fats. There is a theoretical basis for the use of these substances which it is impossible to ignore, although I have no desire, in the present state of our knowledge, to insist too absolutely upon it. In some way or other, fat must undoubtedly be applied to the nutrition of the nervous system, if this is to be maintained in its organic integrity; since fat is one of the most important, if not the most important, of its organic ingredients. But if our theoretical ideas on this point be as yet deficient in the exactness which is to be desired, there can be no doubt, I think, that the practical lessons which they would teach are abundantly verified in experience. If we take, for instance, the class of Neuralgias which are most plainly and indubitably connected with impaired nutrition—those of advanced life, and particularly the inveterate forms of facial tic douloureux—there is the strongest ground, in the results of experience, for insisting upon the value of this class of remedies. To Dr. Radcliffe belongs the merit of having been chiefly instrumental in bringing forward this therapeutical fact in this country, and it is one which I have had repeated occasions to verify. It is a very singular circumstance, which also was first pointed out by Dr. Radcliffe, that neuralgic patients are, in the majority of instances, found to have cherished a dislike to fatty food of all kinds, and to have systematically neglected its use. I have also obtained strong evidence that this is the general rule, and the reverse a rare exception. And it has several times occurred to me to see patients entirely lose neuralgic pains, which had troubled them for a considerable time, after the adoption of a simple alteration in their diet, by which the proportion of fatty ingredients in it was considerably increased.

Cod-liver oil occupies the highest rank among fatty remedies; where it does not immediately disagree with the stomach, this oil is the best fat to employ. But in other cases butter, and especially cream, may be employed with great advantage; and in fact one of the most successful examples of the treatment of Neuralgia which I record was treated solely by the administration of Devonshire cream in increasing, and finally in very large quantities. Even the vegetable olive oil, though far inferior to animal fats as a general rule, may occasionally be used with good effect. It is necessary in many cases to make a series of trials, before we arrive at the particular form of fatty food which is best suited to the particular patient.

(*b*) The various preparations of iron are of use, so far as I know, only in cases which are marked by the existence of actual anæmia. For patients who possess well-globulated blood (as indicated not merely by the color of the face, but by that of the mouth and tongue, especially by the freedom of the latter from teeth-markings, and by the absence of the drowsiness, *muscæ volitantes*, &c., which indicate defective blood-nutrition of the brain) I do not believe that iron treatment has any value. The carbonate, in large doses, is the best form, when iron is needed at all. [Obstinate neuralgia is one of the *signs* of anæmia, itself so generally positive, that, when nothing contra-indicates iron, the carbonate may be used with confidence. Its effects are not unfrequently admirable.—H.]

(*c*) The employment of the so-called special nerve tonics is of great use in some cases, of none at all in others. Quinine, arsenic, and zinc (in various preparations) are the only medical substances of this class which possess any solid claims to efficacy.

With regard to the efficacy of quinine there are the most conflicting opinions, except in one respect. No one doubts that in the Neuralgias which are of malarious origin this medicine, though not infallible, is extremely efficacious. It should be administered, in all cases which from their regular intermittence leave room for a suspicion that this may be their nature, in full doses (five to twenty grains) shortly before the time at which the attack of pain is expected; in fact just in the way which proves most effective in the treatment of regular ague. If after three or four doses a decided improvement is not effected, the probability is great that the Neuralgia is not malarial. Nevertheless, arsenic may subsequently be tried if other means (to be presently described) prove ineffectual.

In a certain number of non-malarial cases, also, quinine produces good effects; but there is no need, nor is it advisable, to employ it in such large doses. From two to three grains, three times a day, is the largest quantity which is likely to be of any use, if my own experience is worth anything. I know of no circumstances which indicate beforehand that quinine will be useful in non-malarial cases, *except that it seems always much more effective in Neuralgia of the ophthalmic branches of the fifth, than in other non-malarial Neuralgias.*

With regard to other non-malarial Neuralgias I share Valleix's opinion, that it is far from being frequently useful.

Arsenic is a more widely applicable remedy: for it is useful in many cases both of the malarial and of the non-malarial type. In the former it should be given, probably, in full doses, of ten minims, increasing to thirty, of Fowler's solution, three times a day. In the non-malarial forms, the ordinary tonic dose of five minims of liq. arsenicalis, three times a day, or $\frac{1}{12}$ grain of arseniate of soda in pill, with extract of hop,[1] will effect all the good which this medicine can produce. The ordinary precautions must of course be observed, as in any other case where we employ arsenic. There is one form of Neuralgia, however, which merits special mention in relation to arsenical treatment; I mean the specially neurotic form of angina pectoris. In France this remedy is extensively used for cardiac Neuralgia. I have myself seen most remarkable relief afforded by arsenic in this complaint, and an extraordinary tolerance of the system to large doses of it. Very recently, Dr. Philipp has put on record a most interesting case of the kind.[2] There are, indeed, some patients whose alimentary canal is too irritable to bear this remedy at all; but it is usually well borne, and often extremely efficacious. Arsenic may also be effectively administered by subcutaneous injection.

The preparations of *zinc*, and more especially the valerianate, enjoy a high reputation with some practitioners. It is necessary to record this fact; but I cannot say that I have ever seen any good result, which could be confidently attributed to these remedies, in Neuralgia.

(*d*) Last, among the constitutional remedies, we have to mention those which are directed against a real or presumed depravation of the blood by some special poison. Neuralgia may certainly arise from *syphilis;* but then it is probably always due to a local deposit somewhere in the course of the affected nerve. Where this can be suspected, iodide of potassium should be administered in large doses;

[1] Dr. Radcliffe tells me he finds that extract of hop enables arsenic to be better tolerated than when given alone.

[2] Berlin. Klin. Wochensch. 4, 1865.

and if this fail, the bichloride, or biniodide of mercury, in small doses. Neuralgia is said to have frequently a *gouty* origin: but the facts on which this statement rests, perhaps hardly warrant a decided opinion. They scarcely amount to more than this, that in a certain ill-defined group of cases, the subjects of which are perhaps more often than not of a gouty constitution, a form of Neuralgia occurs which yields more speedily to treatment with colchicum than to any other remedy. Twenty to thirty minims of the tincture or the wine, three times a day, will be sufficient; and if a marked good effect be not produced in two or three days, the medicine should be abandoned, or even earlier, if any tendency to weakness or irregularity of the heart's action be perceived.

"Rheumatic" Neuralgia is a phrase which, under the precautions above indicated, must still be retained, as signifying a class of cases in which inflammation of circumjacent fibrous tissues seems to cause the neuralgic pain by producing mechanical damage to the nerve. Iodide of potassium in five to ten grain doses twice or thrice daily is often useful; causing the absorption of local deposits, or rather of local proliferations of fibrous tissue. Even in cases where the Neuralgia was the primary affection, and the fibrous hypertrophy secondary to it, the local tenderness and swelling appear to be often diminished by the use of this remedy. I have never seen *colchicum* produce the slightest benefit in these cases, in which local tenderness is a prominent symptom. [In *gouty* cases, however, as above said, colchicum is the most efficacious of remedies. —H.]

2. We have now to consider the large group of narcotic-stimulant remedies for Neuralgia. In this class, I include not only the substances generally recognized as belonging to it, such as opium, belladonna, alcohol, &c., but also many others, such as ammonia, turpentine, &c., which are commonly spoken of merely as "stimulants;" and also substances which, like aconite, are ordinarily ranked either as pure "sedatives" or as "acro-narcotics." I shall not retrace here the arguments which I have given at large, in my work on "Stimulants and Narcotics,"[1] to prove that all these substances possess the common property of assisting nerve function when given in small doses, and of paralyzing it when given in excess.

The narcotic-stimulant group of remedies, when administered internally or by subcutaneous injection, may be said to hold an intermediate position between the constitutional and the local agencies which we may employ against Neuralgia. On the one hand, they enter the general circulation, and pervade the organism. On the other hand, it may be suspected that in many cases their effect is produced mainly by a local action, either upon the central nuclei of affected nerves, or perhaps upon their spinal ganglia.

Indisputably, at the head of all this class of remedies stands opium. And we may consider opium, as used against Neuralgia, to be fully represented, for every useful purpose, by morphia. But the gastric administration by opiates can, after all, be only considered as *palliative*. The invention of the subcutaneous injection (which was imperfectly forestalled by the *endermic* method) has thrown quite a new light on the capabilities of opium as an anti-neuralgic. It may be confidently said that in the right use of this remedy, we possess the means of permanently and rapidly *curing* very many cases, and of alleviating, to a degree quite unknown before, the suffering caused by even the most inveterate forms of Neuralgia.

The *local* injection of alkaloids, as first systematically employed by Dr. Alexander Wood, is a proceeding which is specially applicable, in my opinion, only to a few cases. In many instances the nature of the integument at or near the point of severest pain, is such as to render the local operation inconvenient or even impossible. In the great majority of cases, especially those which are seen early, the injection may be more advantageously performed in some indifferent place, such as the loose skin over the front of the biceps muscle, or, in fact, in any place where a fold of skin can be conveniently picked up. The substance injected, if properly dissolved in a convenient quantity of fluid, quickly enters the general circulation, and, in a large majority of instances, produces just as decided an effect on the local nerve pain, as if it had been locally injected. I cannot doubt that, in the greater number of cases, the "local" injection is such only in name; the injected substance producing no effect till it has entered the absorbent vessels or the veins, and thence travelled all round the circulation to the small arteries, either of the spinal and ganglionic centres, or, perhaps, to the arteries which supply the peripheral branches of nerves. The discovery of the great utility of the plan of general, as opposed to local injection, is due to Mr. Charles Hunter, and is of the highest importance, not merely as a practical fact, but in the suggestions which it gives as to the general subject of the place of origin of Neuralgia. There is, however, a class of cases in which the local injection of morphia becomes desirable. In advanced cases, in which very great local hyperæsthesia exists, and there is reason to think that thickening and hyper-

[1] London: Macmillan. 1864.

trophy of the structures round the nerve has taken place, I have several times known injection at a distant point to fail, when local injection of the same substance, in the same dose, has immediately produced a marked effect; and the same thing has been recently pointed out to me by several medical men. It happens sometimes, however, that in the very cases which seem most to demand the local injection, the local tenderness makes the operation intolerably painful: in such a case I should recommend a plan which Mr. Hart introduced to my notice, viz.: that of first rendering the skin insensible with ether spray, and then injecting. As the freezing process renders the tissues quite hard, a *steel* canula to the syringe is needed to penetrate them.

As regards the dose to be employed, I cannot but think that the received ideas are much in fault. One hears constantly of as much as half a grain or one grain, even, of morphia being employed, even at the outset. That such quantities are necessary, sometimes, where the cellular tissue injected into is already irritated and thickened, I have no doubt; and I explain it by the hypothesis that a good deal of the injected substance never enters the general circulation, nor even the vessels of the part, but lies encysted, just as is undoubtedly the case when one injects an irritant substance like pure chloroform into the cellular tissue anywhere. But I am quite certain that when injection of any non-irritant solution of morphia into a healthy cellular tissue is *neatly performed*, it is unnecessary, and *even unsafe* to commence with larger quantities than $\frac{1}{6}$ gr. Both in my own practice and in that of a friend, I have known so little as $\frac{1}{4}$ gr. produce dangerous symptoms of poisoning in a person not especially sensitive to opium; and I am convinced that the activity of remedies hypodermically used is generally much underrated. I have produced all the desired effects by injection of not more than $\frac{1}{10}$ gr. in slight cases, and very rarely indeed (where the morphia is injected at an indifferent spot) do I increase the dose beyond $\frac{1}{3}$ gr. The best medium dose is $\frac{1}{6}$ gr.; and the injections should be repeated, if possible, daily, or even twice a day in severe cases. In visceral Neuralgia, it need hardly be said, we are obliged to be contented with injection at an indifferent spot; yet (as *e. g.* in ovarian Neuralgia) we sometimes produce excellent effects.

Next to opium in value, amongst the stimulant narcotics, is *belladonna* and its alkaloid *atropia*. The value of belladonna, as given by the stomach, is confined pretty much, according to my experience, to painful affections of the pelvic organs, on the sensory (as notoriously in the motor) nerves, on which it seems to have a special influence. In doses of $\frac{1}{6}$ gr. to $\frac{1}{2}$ gr. of the extract, it will frequently relieve ovarian dysmenorrhœa, as also some forms of superficial lumbo-abdominal Neuralgia. But by far the most important use of belladonna is by the subcutaneous injection of atropia. From the $\frac{1}{150}$ up to the $\frac{1}{30}$ of a grain is about the range of doses for adults; and I can confirm the statements of Mr. Hunter that by repeated applications of this treatment, even very severe and inveterate Neuralgias are often greatly relieved and sometimes cured. It is a question whether there is not less tendency to relapse after this treatment than after that by morphia. On the other hand, I have met with more than one person in whom it has been found impossible to give a dose sufficient to relieve the pain without producing distressing head symptoms.

Next in value to morphia and atropia comes Indian hemp, which has been especially brought forward by Dr. Reynolds. A *good* extract of this, in doses of from $\frac{1}{4}$ to $\frac{1}{2}$ grain or (rarely) 1 grain, given in pill, is very effective in some forms of Neuralgia, particularly in clavus hystericus and migraine. Even in the severest and most intractable forms it often palliates greatly. It should be given every night, whether there be then pain or not.

Muriate of ammonia is an excellent stimulant remedy in migraine and clavus, and in some cases of intercostal Neuralgia. It should be given in 10 to 20 gr. doses. In cases of suspected hepatic Neuralgia I have also found it very useful; and I believe that its action on the liver (in disorders of secretion) is through the nervous system entirely.

Sulphuric ether, which in the severer forms of superficial Neuralgia is of little or no effect, is supremely useful in certain visceral Neuralgias. It sometimes relieves gastralgia, and Neuralgia of uterine or ovarian origin, with magical rapidity. But it is still more valuable in the most purely nervous form of angina pectoris. I have now under my care a case of this latter affection, which I am convinced would have ended fatally long since, in one of the agonizing attacks of spasmodic heart-pain, but for the discovery that, by taking a spoonful of ether immediately on its commencement, the patient can greatly mitigate the attack. This patient had tried arsenic, but from the irritability of his intestinal canal, could not take it. The same dose of ether has continued to produce the same happy effect on each occasion of its use for the last three years.

Aconite, in the form of Flemming's tincture, is of very great use in some forms of Neuralgia, especially in that

kind of ocular Neuralgia, with secondary inflammation, which is so frequently called rheumatic iritis. But, unfortunately, it is a very uncertain remedy in one respect: with some persons it produces nausea, burning in the throat, and a sense of cardiac depression, with doses which are quite harmless to other patients. In a case where I recently employed it, in only three-minim doses every six hours, I was compelled to abandon it after the third dose, from the intensely depressing effect which it produced.

The oil of turpentine is a remedy which enjoys, or enjoyed, considerable reputation for its effect in a certain class of cases. In the more obstinate forms of sciatica it is at least worth a trial, although it is commonly very disagreeable to the patient; ten minims, three times daily, is the proper dose.

Still, after the enumeration of all the narcotic-stimulant substances which have been, and many more that might be, named, it would be idle to pretend that any of them are to be compared, for wide and general efficacy, to the subcutaneous use of morphia and atropine, and the internal use of Indian hemp in small doses.

I have reserved to the last, under the head of Stimulant Narcotics, what must be said about alcoholic drinks. There can be no question about the power of alcohol to relieve neuralgic pains; it is as distinct as that of opium. But the dangers of prescribing it as a remedy are very great, since the patients cannot always be induced to use it in the strictly medical manner in which alone it is safe. Too often, instead of employing it in the moderate stimulant doses which really are of service, they accustom themselves to drowning the pain with a large narcotic dose, and thus they contract a liking for the oblivion of drunkenness. It is of much consequence, where this is possible, that they should be forbidden to take alcohol otherwise than at meal-times. If once they are induced to take it for the mere relief of acute pain, there is great danger that they will drink to excess. I am, nevertheless, convinced that a fixed daily allowance of wine or brandy (beer more rarely agrees), which shall contain not more than one ounce of absolute alcohol, is a decided help to recovery from every form of Neuralgia; and in the case of persons of firm character, who can be trusted to exercise self-control, a larger quantity than this may sometimes be allowed. Without pretending to speculate on the physiological reason for it, I must add my testimony to the fact, which has been observed by Dr. Radcliffe, that *saccharine* liquors and saccharine foods, except in very moderate quantities, decidedly disagree with neuralgic patients. [For many women (the most frequent subjects of neuralgia), an ounce of alcohol daily, however divided, will be too disturbing to the system to be beneficial; and will endanger the tippling habit. Less than half an ounce of alcohol (equal to two tablespoonfuls of whisky) daily, will be ample in most cases.—H.]

3. We now come to consider the external remedies for Neuralgia. Incomparably the most valuable of these is the use of so-called counter-irritation; that is, the application of various irritants to the skin. Valleix comes to the conclusion that there is no one remedy which approaches *blistering* in value, and (putting aside the recently discovered hypodermic treatment) that saying remains absolutely true at the present day. It is to be observed that Valleix latterly always employed the milder form of the flying blister. Such an application as this to the foci of pain must, if we consider it, be supposed to excite a directly stimulant effect upon the painful nerve. This kind of blistering, and the analogous use of mustard plasters, have always yielded good results, in my experience, solacing even when they did not cure. And in numerous early cases one or two flying blisters, applied successively over different points in the course of the painful nerve, have at once and permanently arrested the disease. It is a remedy which ought always to be tried in cases of any severity, especially if the subcutaneous injection of morphia and of atropine has failed. There is one method of blistering which I have recently tried with great success, viz. the application of a blister close to the spine, as nearly as possible opposite the intervertebral foramen from which the affected nerve issues. The effect produced is, I suppose, a reflex stimulation through the posterior branches. This method is of course not so applicable to Neuralgias of the fifth as to those of spinal nerves. Yet even in these, blistering of the nape has sometimes appeared to do marked good—through the occipital nerve, I presume.

The application of various stimulating liniments and ointments to the skin of the painful parts is sometimes very useful. Of these the use of chloroform diluted with seven parts of oil or soap-liniment is by far the most efficacious. This produces no anæsthesia, but a mild stimulation. Strong *counter-irritation* may be produced by the use of tartar-emetic or of veratrine ointment.

Electricity. — The efficacy of various forms of electricity in Neuralgia is a large subject, and as yet, it must be owned, only very partially cleared up. The comparative merits of Faradization and of the continuous current are hardly settled. But the weight of testimony is now in favor of the belief that in the majority of

instances the continuous current is the most valuable.

As regards one or two points, one may speak with some confidence. In the first place I may say, after extensive trials of the ordinary rotatory (magneto-electric) machine for the *induced* current, that this method of treatment is most unsatisfactory. I have never seen it produce, indisputably, good effects. Secondly, as regards that form of *continuous* current which is generated by Pulvermacher's chains, I am reluctantly obliged to give up the hope of doing any real service with it in Neuralgia, however great its utility is in other diseases. As is remarked by Dr. Althaus, the current generated by these chains is too irregular, and their activity is too soon exhausted for us to get a sufficiently uniform dose of electricity applied continuously for a definite period by their means.

It appears probable that we shall ultimately find that for neuralgic affections of all kinds the most useful form of electrical element is by the continuous current generated from a Bunsen's or a Daniell's battery; and that the three principles on which we must act in its use are:—1. The maintenance of the current, with only a very few breaks, for a considerable time. 2. The application of the positive pole over the seat of pain. 3. The employment of a very low-tension current. I am informed by Mr. J. N. Radcliffe, whose experience in this matter is very large, that the use of this mode of electrization in Neuralgia is as yet, in his opinion, only beginning to be developed, but that it promises to effect great things. In short my present opinion as to the value of electricity in Neuralgia may be thus expressed: that as used, up to the present time, it has achieved no results which entitle it to more than a third or a fourth rate place among remedies; but that if the desideratum of a low-tension continuous current, which can readily be applied for long periods together, can be obtained by means of apparatus of moderate portability and cheapness, it is probable that we may obtain that which will equal or exceed in value any of the remedial measures which are at our disposal.

A few words must be given to the rather uninviting subject of the surgical treatment of inveterate Neuralgia. The section of a neuralgic nerve, or rather the excision of a piece, is still, I suppose, to be reckoned among the measures which it may be occasionally justifiable to employ. Nothing, however, either in the two cases of its use which I have seen, or in the records of similar operations, would lead me to recommend it in any case. The relief given is nearly always very transient; and, indeed, the nearly infallible certainty with which the pain returns in the central end of the divided nerve is only what I should expect from the many considerations which point to the central origin of the nerve as the most peccant part. With such remedies in our hands as the subcutaneous injection of morphia, &c., I cannot see that we need to be tempted to perform such an operation for the sake of a temporary alleviation. [*Nerve-stretching* is an operation recently somewhat in vogue, of which the same remarks may be made.—H.]

The removal of any distinct source of peripheral irritation by surgical means is quite another matter, and may be highly proper and necessary. Yet even here it is always necessary to calculate whether the shock of the procedure itself may not be injurious; and it will be desirable before inflicting it to fortify the system, as far as possible, with tonics; and sometimes to diminish the shock, not merely by giving chloroform, but by prolonging the chloroform narcosis by subcutaneous injection of a large dose of morphia. This precaution is especially advisable where we extract one or more carious teeth, which may seem to be keeping up neuralgic pain. Too often we find that the extraction has been in vain; and then, unless some such precautions have been taken, it may be discovered that the shock has aggravated the Neuralgia.

A most important subject, with which I may conclude these remarks on treatment, is the employment of suitable *prophylactic* measures. First, as regards nutrition; it is absolutely necessary that this should be as abundant as may be possible without deranging the digestion. It must also contain a liberal allowance of fatty matters; no amount of dislike on the patient's part—and they often show great dislike—should induce the physician to give up this point. If one form of fat cannot be tolerated, another must be tried; perseverance will, I believe, always bring success; and the effect of an improvement of this kind in the diet will rarely fail to tell upon the constitution, rendering the nervous system less sensitive to the ordinary exciting causes of neuralgic pain. Equally important is the avoidance of exposure to cold and damp air with insufficient clothing, for cold is much the most frequent immediately determining cause of neuralgic attacks. Flannel underclothing, thick veils for the face, &c., are quite as important as any direct remedies. It cannot be doubted that everything which tends to set up the habit of pain, directly tends also to aggravate that obscure vice of the organism on which the disposition to Neuralgia depends, and *vice versâ*. Physical exercise must be so regulated that it may improve nutrition without inflicting severe fatigue. And as regards mental influences, which,

unfortunately, are often beyond control, one can only say, that the two extremes, of a specially laborious and exciting life, and an existence spent in the dreary monotony of idleness, are equally hurtful.

[*Sunshine* is, usually, very beneficial. Let the neuralgic patient live in the light as much as possible. I have repeatedly known a severe attack of hemicrania to be relieved by the patient sitting or lying directly in the rays of the sun.—H.]

In the foregoing article I have followed the plan also adopted in my article on Alcoholism; namely, of stating my own view of the subject connectedly, and without pausing to answer all the statements and opinions of the numerous writers who differ from me. The necessary limits of a work like this "System of Medicine," makes it almost impracticable for an author to follow any other course with success, if he happens to hold a view of his subject which conflicts with, or differs from, the views of well-known authors on a considerable number of points. But the following selected list of the more important treatises will enable the reader to study the questions connected with this disease from every point of view. It has been my purpose to bring out clearly and consistently that view of Neuralgia which seems warranted by the majority of the facts recorded by others or observed by myself; and the result has been that I have given much prominence to the arguments for the existence of an element of organic change in the *centres* in all true Neuralgias. Those who desire, however, to hear all the arguments which can be urged for a chiefly or solely peripheral origin of Neuralgia will find abundant material in the undermentioned treatises: Trousseau, "Névralgie Epileptiforme," vol. i. of his "Clinique Médicale," 2me Edit.; "Névralgies," vol. ii. of the same work (Trousseau's insistance on the constant presence of a painful "point apophysaire," seems to me an overstatement; but it is still more strange that this author should think its constant presence could consist with a peripheral origin of Neuralgia); Beau, Traité des Névralgies, Arch. de Méd. 1847; Brown-Séquard, Lectures on the Therapeutics of Nervous Diseases, Lancet, 1866, vol. i. (see also his Lectures on the Physiology and Pathology of the Central Nervous System, 8vo. Philadelphia, 1860). Of authors who allow at least a large share in the production of many cases of Neuralgia to the centres, are Teale, Treatise on Neuralgic Diseases, &c., London, 1829; C. Handfield Jones, on Functional Nervous Disorder, London, 1864; also Lumleian Lectures, Med. Times and Gaz. 1865, vol. ii. But the most suggestive and important treatise, and one which has been unaccountably neglected, is the Observations on the Functional Affections of the Spinal Cord, by William and Daniel Griffin, London, 1834. I have, in the text, given Valleix just credit for laying the foundation of the current knowledge respecting Neuralgia; but it must be allowed that in the work of the Griffins, which is little known, there are the germs of a great improvement of that knowledge. Of essays which illustrate the serious secondary complications which may attend Neuralgia, the following may be mentioned, besides the treatises of Barensprung, of Notta, the work of the Griffins, and the other papers already specified: Schiff, Hyperæmia of the Eye, Ulceration of Cornea, &c., after a Wound of the Superior Maxillary Nerve; Untersuch, p. 116; Allcock, Disease of the Eye from Injury to the Infra-orbital Nerve; Todd's Cyc. of Anat. and Physiology, vol. ii. p. 132. A great many cases also are quoted in Handfield Jones's Lectures on Functional Nervous Disorders, already cited.

It is only just to Dr. Handfield Jones to acknowledge that he has long advocated the opinion that nerve-pain is invariably, and in all its phases and consequences, an expression of debility of function; an opinion which has been strongly expressed also by myself not only in the present article, but in many other papers.

LOCAL PARALYSIS FROM NERVE DISEASE.

By J. Warburton Begbie, M.D., F.R.C.P.E.

There can be no doubt that for a lengthened period, and till a comparatively recent date, the attention of pathologists was too exclusively directed to the great nervous centres in explanation of the causes of nearly all nervous disorders, including paralysis. So much so was this the case as fully to justify the language employed by the late Dr. Graves, of Dublin. "If," says he, "you examine the works of Rostan, Lallemand, Abercrombie, and those who have written on diseases of the nervous system, you will find that their inquiries consist in searching after the causes of functional changes, either in the cerebrum, cerebellum, or

spinal marrow, forgetting that these causes may be also resident in the nervous cords themselves or their extremities, which I shall call their circumferential tracts."[1] Since 1843, however, when the first edition of Graves's Lectures appeared, it has been satisfactorily determined by physiological investigation and by the careful observation of disease in numerous examples, that paralysis, or the loss of the power of motion, may result from one or other of two causes. It may depend either on a central nervous lesion, that is, a lesion of the Brain or Spinal Cord, or on an abnormal condition of a particular nerve in some part of its course. It is with the latter, as giving rise to a local form of paralysis, that we are now exclusively concerned. We are abundantly familiar with the effects of mechanical injury as applied to nerves. When a nerve is cut across, there results immediately a paralysis of the parts below the section supplied by that nerve. Further, if a nerve be included in a ligature, or subjected from any cause to much pressure, a similar result is produced. The paralysis of the arm caused by pressure on the axillary plexus of nerves, is an excellent and familiar illustration of injury so occasioned. It is thus described by Dr. Todd:—"A man gets intoxicated, and falls asleep with his arm over the back of a chair; his sleep under the influence of his potations is so heavy, that he is not roused by any feelings of pain or uneasiness, and when at length he awakes, perhaps at the expiration of some hours, he finds the arm benumbed and paralyzed. It generally happens that the sensibility is restored after a short time, but the palsy of motion continues. Cases of this kind sometimes derive benefit from galvanism, but if the pressure which caused the paralysis has been very long continued, they seldom come to a favorable termination. Nerve-tissue is one which never regenerates quickly, and seldom completely, so that great or long-continued lesion of its structure is not likely to be removed."[2] Although by no means so distinctly witnessed as the result is, in the class of cases now referred to, there seems no reason to doubt that, equally with mechanical injury, interference with the proper nutrition of nerves may lead to forms of local palsy. Illustrations of such occurrences will be adduced, more especially when directing attention to one of the most interesting of all the varieties of local paralysis, namely facial palsy. Again, familiar as we are with the action of various poisons—such as alcohol, opium, chloroform—on the great nervous centres, and on the same portions of the nervous system of certain poisons formed in the living body, as urea, and the morbid materials in rheumatism and gout; having also important knowledge regarding the influence which is exerted on the nervous and muscular systems generally, but especially on the nerves and muscles of the upper extremities by the poison of lead, we cannot hesitate to account, in a manner closely similar, for the other forms of local paralysis which from time to time present themselves to our notice.

Dr. Todd alludes to cases of local paralysis occurring in states of the constitution which, if not rheumatic, are at least allied to it, and associated with imperfect action of the kidneys. "Of this," he says, "the following affords a good example:—A medical man, ætat. 53, extensively engaged in practice in the county of Bucks, applied to me in August, 1847, with complete paralysis of the deltoid muscle. He was a stout, full man, tall, of large build, and very active in his habits; fed well, and drank beer, but not to excess. He had been subject to a shifting neuralgia of the scalp, and to a discharge from the right ear, where he thought the tympanic membrane was destroyed; he was deaf on that side. Six weeks before he came to me he suffered from pain in the left side of the neck and shoulders, followed by complete paralysis of the left deltoid muscle and weakness of the whole arm. On examining, I found a total inability to raise the left arm to a right angle with the trunk, or to perform any of those actions which are usually effected by the deltoid muscle, which was very much wasted. He could, however, grasp perfectly with the left hand, and execute all the other movements of the arm and forearm. There was some degree of numbness of the arm. There were no symptoms distinctly referable to the head. His tongue was coated; appetite good; the discharge from the ear had ceased. The urine was pale, of low specific gravity, and contained albumen in small quantity. I viewed the case as one of local palsy, connected with a deranged state of system, rheumatic or gouty. I regulated his diet, and gave him small doses of the mineral acids. After a fortnight of this treatment he improved considerably, and could raise his arm slightly. The albumen in the urine had much diminished: and crystals of lithic acid were precipitated. He was now ordered three grains of iodide of potassium, with ten minims of liquor potassæ thrice daily. He only followed this treatment for ten days, as the iodide of potassium purged him. Still, he was improving. I continued the liquor potassæ, and advised galvanism to the

[1] Clinical Lectures on the Practice of Medicine, Lecture xxxiii.

[2] Clinical Lectures on Paralysis, certain Diseases of the Brain, and other Affections of the Nervous System, Lecture i.

muscle. This plan was diligently pursued for a fortnight, at the end of which time he had so far improved that he could raise his arm nearly to a right angle,—he could put on his coat, and tie his cravat; and in three weeks more he was quite well. All signs of albumen had disappeared from his urine."[1] The writer's experience has furnished cases bearing a remarkable resemblance to the one now quoted. He calls to remembrance more especially that of a young and plethoric as well as highly rheumatic female, who suffered from paralysis, succeeding severe pains of the left lower extremity, and in whom a plan of treatment which secured the copious discharge of urine, previously much diminished as well as disordered, and free action of the skin, proved eminently successful in removing the palsy of the limb. Besides the gouty and rheumatic poisons, it is well to keep in view the very decided action of the syphilitic in inducing this among other local disorders. No one calls in question the injurious effects which are capable of being produced on the nervous centres by the syphilitic poison; there is, however, good reason to believe that some local palsies are thus created. The writer has been able to trace the occurrence of paralysis of the portio dura, of paralysis of the third pair, as shown by a marked ptosis; and also of palsy of the limbs, slight although threatening, to the same cause, when neither brain nor spinal cord appeared to be implicated. And it is probable that the experience of many physicians has not been dissimilar to his own, in finding the iodide of potassium administered in large doses, and steadily persevered with, a most useful remedy in such cases, relieving the palsy as effectually as it is so frequently the means of doing, the neuralgic and wearing-out headache, or the painful node on the shin bone, which are evidently due to the same cause. Allusion has been made to the influence of direct pressure external to the body, in producing such injury of nervous structure as leads to a form of local paralysis. Palsy thus induced is generally merely temporary in duration. Tumors within the body, involving nerves, are frequently the direct occasion of local palsies. No more interesting variety of such palsy exists than that which is due to the interference with the recurrent or motor laryngeal nerve produced by an aneurism of the arch of the aorta, or by a cancerous mediastinal tumor. Well-marked atrophy of the muscles of one side of the larynx has under such circumstances been found. The dyspnœa, which is induced by the implication of the vagus, or as sometimes happens of the phrenic nerves in strumous or tubercular tumors, is abundantly recognized since the writings of Risberg and Ley. There seems reason to believe likewise that pressure upon or other injury of some parts of the sympathetic nervous system may occasion local palsies. Of this the paralysis of the radiating fibres of the iris caused by cutting the sympathetic in the neck in Budge and Waller's experiments, but especially a similar contraction of the pupil to that physiologically produced, due to the pressure of an aneurism projecting into the neck or malignant tumor similarly situated, are now quite familiar to the physician.

Attention will now be directed to some of the more important varieties of local palsy dependent on nerve disease, and first to *Facial Palsy*. This most interesting local paralysis is known under different names, of which the more commonly employed are *Facial Hemiplegia*, *Histrionic Paralysis*, *Bell's Palsy*, and *Paralysis of the Portio dura*. Occurring as it usually does on one side of the face only, nothing can be more striking than the peculiar features of the disease. This is owing to the palsied condition of a few or all of the superficial muscles—the muscles of expression—on the affected side, and the heightened antagonism of muscular action on the unaffected side. The patient cannot knit the forehead,[1] neither can the eyebrows be raised or drawn together. The eye remains open, as the power of closing the lids is lost, and their blinking movement no longer exists. This open condition of the eye, seen both in waking and sleeping, and which is due as well to the increased action of the levator palpebræ muscle as to the palsy of the orbicularis palpebrarum, is a characteristic, it has indeed been styled a pathognomonic, feature of facial palsy.[2] The ala nasi is dependent, and on full inspiration on smelling or blowing the nostrils there is no expansive movement. The angle of the mouth hangs down. Further, the patient cannot whistle, for he is unable to purse up his mouth for that purpose, and for the same reason he can neither spit,

[1] Loc. cit. p. 72.

[1] In alluding to the smoothness of the brow in the aged, who are affected by facial palsy, owing to the disappearance of all wrinkles, Romberg facetiously observes, "für alte Frauen kein wirksameres Cosmeticum existirt."

[2] "The leading character of cases of facial palsy," writes Dr. Todd, "is the inability to close the eyelids from paralysis of the orbicularis palpebrarum: this is the pathognomonic sign which determines the peculiar nature of the palsy, and distinguishes it from the most serious form of facial palsy, which is dependent on diseases of the brain and palsy of the fifth or third nerve." (Clinical Lectures, Lecture iv.)

nor can he distend the buccal cavity with air, or blow wind from the mouth. Pronunciation of labials is notably impaired. The saliva and fluids frequently trickle from the mouth. In mastication portions of food are apt to collect between the cheek and gums, as the support of the lips and cheeks necessary for its proper performance is lost. Let the patient laugh, cry, sneeze, yawn, or be the subject of any violent emotion, and the distortion of the features becomes much more conspicuous, the face being forcibly drawn to the sound side. Motionless and void of expression is the one side, contrasting in a very remarkable manner with that on which intelligence remains visible and power of movement unaltered. Trickling of the tears down the cheek, owing to the immobility of the lower eyelid, with consequent dryness of the corresponding nostril, and redness of the conjunctiva, it may even be severe conjunctivitis, determined by the operation of cold, dust, or other external influences on the constantly exposed eye, are among the accompanying phenomena of this palsy.

To Sir Charles Bell we are indebted for pointing out the true nature of this affection. He showed that one nerve only was involved, that the muscles governed by the portio dura of the seventh pair were alone affected, that strictly it is a local palsy. The sensibility of the face is usually unimpaired; a slight affection of the filaments of the fifth may, however, cause a little facial pain, but that is to be accounted rare. In instances of long-standing facial palsy, Romberg has drawn attention to the relaxed and flaccid condition of the skin covering the affected muscles, while Dr. Todd has insisted on increasing flaccidity of the cheek, and especially a rapid development of that condition, as a symptom of unfavorable omen as regards the patient's prospects of recovery. But while this form of local palsy is clearly dependent on lesion of one nerve only, there is reason to believe, as Romberg has more particularly shown, that its features are subject to modification, according to the precise seat of the disease. That may be *peripheral* or *central*. Not only so, but the diagnostic marks may vary under the former head, according as the superficial distribution of the portio dura, or the nerve as it passes through the temporal bone, or the nerve within the cranium and near its central origin, is affected. Viewing these very briefly in their order, it may be remarked —that, *facial palsy, due to an affection of the superficial distribution of the nerve*, is generally met with as the result of exposure to cold.[1] "A very common cause of this palsy," writes Dr. Todd, "is the influence of cold; as by exposure at an open window, in a coach or railway carriage, to a current of cold air."[1] "A blast of cold air on one side of the face," remarks Dr. Graves, "has been known to cause paralysis and distortion of several months' duration."[2] External injuries, such as blows on the cheek and surgical operations on the face, have been followed by this form of local palsy. Of the cases which occur, there are not a few in which no traumatic cause can be found, neither can any marked exposure to cold be traced. In such circumstances it is proper to make a very careful inquiry into the condition of the general health of the sufferer, when it is not unlikely that the connection of the palsy with a gouty or rheumatic taint may be satisfactorily established. Dr. Todd, alluding to the dependence of periodic neuralgic affections on the determination of some poison to a particular nerve, as the paludal poison or some matter generated in the system, expresses the opinion that morbid matters may affect a motor nerve just as they affect a sensitive, causing in the former case paralysis, as in the latter they determine neuralgia.

Facial Palsy caused by an affection of the portio dura in its passage through the temporal bone.—The connection of this paralysis with local strumous affections in children is well known. These may be simple and easily remediable, as for example the parotid and more general glandular enlargements consequent on measles, scarlatina, and other disorders; but of much more serious nature is the otitis resulting in caries of the petrous portion of the temporal bone. Here the palsy is associated with deafness, and very probably also with purulent discharge from the meatus. Direct violence, likewise, as in a case related by Sir Charles Bell, in which a pistol-shot through the ear had splintered the bone, and torn the nerve in its osseous canal, may of course determine the palsy. The diagnosis of the disease or injury affecting the nerve, in its passage through the bone, rests, according to Romberg, not only on the coexistence of such phenomena as otorrhœa, removal of necrosed portions of bone, perhaps of one or other of the small bones of the ear, and deafness, — symptoms which are not likely to occur in cases of

[1] Some writers speak of facial palsy as specially a disease of northern climates. Thus Joseph Frank, after alluding to the collection of cases by various authors, remarks, "Nosque plurima exempla vidimus. Morbus iste in regionibus septentrionalibus tam communis est, ut spatio quindecim annorum viginti duo mihi obvenerint exempla." (De Paralysi, Praxeos Medicæ Universæ Præcepta.)

[1] Loc. cit. p. 69.

[2] Loc. cit. p. 380.

simple peripheral facial palsy,—but, further, upon certain peculiarities in the observed paralytic phenomena. One of these is the diminution of taste on the side of the tongue corresponding to the palsy, another is a unilateral paralysis of the velum palati. On the latter point the statements of writers have been very contradictory. Romberg remarks that in four patients afflicted with facial palsy he has noticed the paralyzed condition of the velum palati, the uvula, having a slanting direction, being arched and the tip pointed to the paralyzed side. While failing to offer any explanation of the peculiar position of the uvula, Romberg evidently attaches very great importance, in a diagnostic point of view, to the palsied condition of the velum, and the marked curving of the uvula; concluding from their existence, that the seat of the disease *must* be in the petrous portion of the temporal bone. And he again emphatically repeats when the disease is in the peripheral distribution of the nerve, the velum is not affected, "wovon ich mich in vielen Fällen überzeugt habe." It is the implication in the diseased condition, of whatever nature that may be, of the nervus petrosus superficialis major, of Arnold—which takes its origin from the knee-shaped bulb on the trunk of the portio dura as it lies in the Fallopian aqueduct, and which communicates with Meckel's ganglion, whence the muscles of the palate derive their nerves,—that in the view of Romberg causes the displacement of the velum and uvula. Dr. Todd, while admitting the occasional occurrence of this phenomenon, combats the notion of Romberg, and maintains that undoubted instances of disease of the aqueduct, causing paralysis of the nerve, are met with, in which affection of the velum does not exist. In his own experience the symptom in question was of very rare occurrence, and he regarded it as a coincidence. Since the publication of the views of the authors now referred to, the paralysis of the palate in facial palsy has received renewed attention from M. Davaine and Dr. Sanders. The former recorded one case of unilateral paralysis of the palate, in connection with facial palsy of right side, observed by himself, and has commented on several instances furnished by Romberg and others. His description of the phenomena he observed is given as follows: "The velum palati is not regular; the arch formed by the right anterior pillar is less elevated than the left. The posterior pillar of the same side descends directly downwards, without being curved like that of the other side. The uvula is bent like a bow; its point is directed forwards and towards the paralyzed side, while its base is carried a little towards the sound side. The patient's voice is slightly nasal."[1] Dr. Sanders, in a valuable paper,[2] gives an interesting case of paralysis of the velum in connection with facial palsy of the right side, and enters at some length into a consideration of the mechanism of the deviation of the palate. Dr. Sanders is satisfied that a partial hemiplegia of the palate does exist in connection with facial palsy, and, like it, is dependent on affection of the portio dura. He believes that this form of palatal palsy consists in a vertical relaxation or lowering of the corresponding half of the velum palati, with diminished height and curvature of the posterior palatine arch, on the paralyzed side, and that it is due to paralysis of the levator palati,—that muscle and the azygos uvulæ, also supplied by the seventh pair, being the only muscles affected. Among several conclusions at which Dr. Sanders has arrived, the following appear to be specially important: that the partial paralysis of the velum in facial palsy, due to implication of the levator palati muscle, is by no means so rare as palsy of the velum (hitherto not accurately described) has been generally supposed, and that the prognosis is not necessarily rendered more unfavorable in facial palsy when the palate is implicated.

The lesion in facial palsy may exist at the cerebral origin of the seventh pair of nerves. We are not, however, called upon to consider this variety of facial palsy: suffice it to say, that its existence may be determined, and the differential diagnosis between it and the other forms—already briefly considered—established, by the occurrence, sooner or later, of symptoms due to the implication of other nerves, such as deafness, strabismus, ptosis and anæsthesia. While either the presence of inflammatory products, or apoplectic extravasations in the vicinity of the pons Varolii, may be the precise lesion which gives rise to the palsy, the probability is that, in such cases, a tumor of one nature or other, and subject to gradual extension, exists.

The duration of facial palsy is subject to considerable variety, according to the precise seat and nature of its determining lesion. Dr. Todd remarks that "it rarely, if ever, lasts a shorter time than ten days, whilst it very often extends to as many weeks; perhaps three or four weeks may be assigned as an average duration for the non-traumatic cases;" and Romberg warns us not to expect its duration to be brief. It is in those cases which have been evidently connected with rheuma-

[1] Gazette Médicale De Paris. 1852.

[2] Edinburgh Medical Journal, August, 1865.

tism that he has found the paralysis least enduring.[1]

The writer has seen simple cases of the disease, in so far as their cause was concerned, lasting a very lengthened period, many months, and even a year.

It is incumbent on the physician to be very careful in offering an opinion as to the prognosis in cases of facial palsy: that must always be founded on a consideration of the probable cause. Those cases are nearly certain to terminate favorably in which cold or rheumatism is to be looked upon as the determining agent. On the other hand, when the palsy has been due to mechanical injury the prognosis cannot be favorable, and this very specially in those instances where a division of the nerve has been caused. We cannot be too careful in the expression of our opinion in cases characterized by nerve disease within the temporal bone. The records of medicine contain reports of such, which have given rise to meningeal inflammation, intracranial, even cerebral and cerebellar, abscess and death.

If prognosis is to be guided by a just consideration of the causes, so also is the treatment of facial palsy when amenable to cure. The remedial measures at our disposal may be conveniently classed under the heads of internal and external agents. In the use of the former, regard should always be had to the diathetic condition of the patients—rheumatic, gouty, strumous, syphilitic, anæmic, or suffering from the injurious influence of a paludal poison. We are disposed to think that this is one of the forms of local palsies in which the loss of power may be due to changes in nerve structure determined by neuritis. In such examples, and still more so if there be reason to conclude that a syphilitic taint is in existence, iodide of potassium will prove a most serviceable remedy. We have ourselves found it to be so. The iodide should be administered in doses of five grains twice or thrice daily, simply dissolved in distilled water. The efficacy of the remedy is secured by its being administered while the stomach is empty, but food may be taken very shortly thereafter. Should a rheumatic or gouty habit be found in connection with the palsy, alkaline remedies, colchicum, and lemon-juice may exert a beneficial influence, and so probably will quinine or arsenic in the not unknown examples of the disease allied to intermittent fevers. Mercury in the form of blue pill has been extolled by several practitioners. Sir Thomas Watson counsels the exhibition of mercury "so as just to touch the gums," adding, "I should always take this precaution, lest any effusion of lymph should cause abiding pressure on the nerve."[1] Iron is likely to be useful when an anæmic condition of the system exists. The muriate of lime, the iodide of iron, and cod-liver oil, are available remedies when a strumous cachexia obtains. The writer can bear a decided testimony to the therapeutic value of strychnine as an internal remedy in one long-existing instance of the disease, which had bid defiance to the more ordinary remedies; he cannot, therefore, coincide in the observation of Dr. Todd, that "strychnine is of no use in such cases."

As to external remedies. Blisters, strongly recommended by some physicians, are discountenanced by others, on the ground that they sometimes cause enlargement of the neighboring glands, which by pressure may in their turn injuriously influence the nerve twigs. Local hot fomentations and the application of leeches are very useful remedies at an early part of the disease, the employment of the latter being generally limited to persons of full habit, and otherwise in the enjoyment of fair health. The endermical application of strychnine—over a blistered surface—the use of various stimulating liniments, and particularly, in the writer's opinion, galvanism, are the more approved remedies in cases which have lasted for a little time.

Before concluding our notice of facial palsy, we must add a few remarks on the occasional occurrence of the disease on both sides of the face, and very briefly refer to the statements of Dr. Todd respecting the integrity of the seventh pair in cases of cerebral hemiplegia, a view which has recently been ably controverted by Dr. Sanders.

Double Facial Paralysis.—This is unquestionably a rare affection, and especially rare when the double palsy is solely dependent on nerve disease. Romberg and Dr. Christison[2] refer to cases of what may be styled simple bilateral paralysis of the face, while the seventeenth case in Dr. Todd's lectures is a very remarkable example of paralysis of the portio dura on both sides connected with affection of the portio mollis; for the patient was "perfectly deaf in both ears;" and the loss of function of both branches of the seventh pair evidently resulted from disease in the temporal bone. In addition to the writers already named, M. Davaine has especially directed attention to the

[1] "Die Dauer der mimischen Gesichtslähmung ist selten kurz. Am kürzesten fand ich sie bei der rheumatischen: doch habe ich sie auch hier in günstigen Fällen nur selten unter sechs Wochen wahrgenommen, einmal sah ich die Heilung innerhalb acht, ein andermal in vierzehn Tagen."—P. 664.

[1] Lectures, vol. i. p. 563.

[2] Monthly Journal of Medical Science, 1850.

subject in a valuable memoir, the title of which is given below,[1] and to which Professor Gairdner,[2] of Glasgow, in giving an account of a very interesting case of double facial palsy, has referred. Dr. Gairdner considered the paralysis to be due to cold, and connected with rheumatism of the external branches alone; and in the course of his paper he alludes to another case of double paralysis of the portio dura, evidently connected with syphilis. In the latter case iodide of potassium, with iodide of mercury and corrosive sublimate, were employed in alternate doses, and the result was an excellent recovery. One example of double facial palsy has occurred under the writer's observation; it was associated with tubercular disease within the chest, and the patient, a man of thirty years of age, subsequently died of what appeared to be strumous meningitis. Unfortunately an examination of the body after death was not permitted. This is scarcely the opportunity for entering on a consideration of the view which was so strongly entertained and expressed by the late Dr. Todd, that the seventh nerve was very rarely involved in facial palsy depending on cerebral disease, and that the affected facial muscles were those governed by the fifth pair. It will, however, tend to complete the brief exposition of facial paralysis now given, if we state in this connection, that there is, in our opinion, no reason to doubt that the view taken by Dr. Todd, and in which several systematic writers in this country have closely followed him, is erroneous, and that, on the other hand, the current doctrine on the Continent, and which has been recently ably unfolded and extended by Dr. Sanders, is correct; viz. "that in cerebral hemiplegia, as in peripheral face-palsy, it is the motor seventh nerve which is affected.[3]

Disease of other of the motor cerebral nerves than the portio dura may likewise determine local palsies. A short reference to such may be made here.

Paralysis due to disease of the third pair of nerves (oculo-motor).—Ptosis or blepharoplegia, the falling down of the upper eyelid, is the notable feature of this affection. When this is due to a cause seated within the cranium, such as an inflammatory exudation, or a tumor, it is almost invariably accompanied by palsy of those muscles of the eyeball, and those fibres of the iris which are likewise governed by the motor oculi. Hence in such cases, and they are far from being uncommon, external squint and dilatation of the pupil are associated with the ptosis. Not only so, but other adjacent cerebral nerves are for the most part implicated, while the indications of the existence of some formidable cerebral lesion are under such circumstances not likely to be absent. On the other hand, when the determining cause of the local paralysis is peripheral in its seat, the ptosis exists alone. Romberg remarks that rheumatism may be the cause of paralyzing the palpebral branch of the motor oculi, although not so frequently as is the case with the facial nerve; and he distinctly states that when so induced, the ptosis occurs without the participation of the muscles of the eyeball, and the contractile fibres of the iris.[1] The writer remembers to have seen this dependence of ptosis on rheumatism illustrated in the case of a young lady, who, after having frequently suffered from distinct rheumatic affections, became within a limited period the subject of facial palsy and ptosis, the immediate peripheral impression on both the seventh and third nerves being evidently due to severe cold. A complete and speedy recovery occurred after the local application of warmth and the use of anti-rheumatic remedies. M. Marchal de Calvi has directed attention to the occurrence of oculo-motor paralysis, consequent on very severe tic of the face. M. Marchal, and likewise the late M. Jobert de Lamballe, found the muscles of the eyeball affected as well as dilatation of the pupil, the vision[2] disordered, and insensibility of the conjunctiva in this affection. Such cases, however, are rather illustrative of the reflex form of paralysis, our knowledge of which has been of late greatly increased by the observations of M. Brown-Séquard and others.

In the same way as peripheral affection of the oculo-motor nerve exists, so may local paralysis result from disease of the *fourth pair* (*trochlear*), and of the *sixth pair*

[1] Mémoire sur la Paralysie générale ou partielle des deux Nerfs de la septième paire: lu à la Société de Biologie (Mars, 1852) par M. C. Davaine. See also Gazette Médicale de Paris, 1852.

[2] Clinical Observations, Lancet, May 18, 1861.

[3] On Facial Hemiplegia and Paralysis of the Facial Nerve, by Wm. R. Sanders, M.D. Lancet, 1865. See on the same subject Dr. Hughlings Jackson in Clinical Lectures and Reports of the London Hospital, 1864.

[1] "Der rheumatische Anlass paralysirt, obgleich nicht in solcher Frequenz wie den Facialis, den Ramus palpebralis des Oculomotorius und hat eine einfache Blepharoplegie ohne Theilnahme der Augenmuskeln und der contractilen Irisfasern, nach der Norm der isolirten Leitung, zur Folge." (Augenmuskellähmung.)

[2] Mémoire sur la Paralysie de la troisième paire consécutive à la Névrose de la cinquième. (Archives Générales de Médecine, Juillet, 1846.)

(*abducens*). Such are, however, much less frequent in their occurrence, and especially so, as Romberg has observed, that resulting from affection of the abducens. The author just named has made reference to a case seen by Dr. Dahling, and published by Stromeyer, in which the facial and abducens nerves on the left side were paralyzed in consequence of a sudden cooling of the heated face.

Palsy of the tongue from affection of the hypoglossus nerve in its distribution is of great rarity, offering a marked contrast to the frequency with which a central lesion gives rise to the same form of local palsy.

The lesser branch of the fifth pair may be the seat of disease and consequently give rise to masticatory palsy. The movements of the face in mastication on one or both sides, as the case may be, are thus arrested or impeded. The temporal and masseter muscles are readily recognized to be inactive; and their condition when the disease is unilateral offers to the touch a marked contrast with the firmness of the same muscles on the unaffected side during the process of mastication. This variety of local palsy, when due to disease of the nerve, is generally caused by tumor of the dura mater, or disease of the sphenoid bone, or such a morbid condition of the gasserian ganglion as compresses the nerve itself.

LOCAL SPASMS.

By J. Warburton Begbie, M.D., F.R.C.P.E.

The term *Spasm* (*spasmus*, from σπάω, I draw) is used to indicate the sudden and involuntary contraction of muscular fibres or of muscles. *Hypercinesis* (ὑπέρ, in excess, κίνησις, motion) is likewise employed in a sense precisely similar. This peculiar vital phenomenon may be general or local, involving apparently all, or nearly all, the muscles of the body, or, on the other hand, limited to a few muscles—it may be, to one.

In every occurrence of Spasm there is increased action of the motor nerve, the result of which is the sudden contraction of muscular fibres, the act itself being wholly removed from the control of the will. The expressions *clonic* and *tonic* are used, the former to denote a Spasm which is characterized by rapidly alternating contraction and relaxation of muscular fibres, while the latter implies the existence of the contractions for a certain time, and of this condition rigidity of the affected muscles is also an invariable feature.

Attention is now to be directed to *local* as distinguished from *general* or universal spasms. To the latter, the term *convulsions* is correctly applied.

Local Spasm is not necessarily attended by pain, but it generally is so, and as expressive of painful Spasm we find a suitable term in *cramp* (Saxon *kramp*). The term *cramp* is most frequently applied to painful muscular contraction in the extremities, and to the same phenomenon affecting the stomach or intestines, and also the heart. Such pain as occurs in connection with Local Spasm is in all probability due to injury done to the sensory nerves supplying the muscle during its violent contraction.

Both kinds of muscular fibre, both orders of muscles, the voluntary and involuntary, are liable to be affected by Spasm. Of the former the most familiar illustration is cramp in the extremities. Of the latter are cardiac and intestinal Spasms. Romberg has pointed out that, as a general rule, when the muscles of animal life, those under the control of the cerebro-spinal nerves, are affected by Spasm, the fibres exhibit a uniform contraction throughout their whole extent; while, on the other hand, the muscles of organic life, over which the sympathetic system is dominant, when similarly affected manifest successive contractions moving like waves.[1]

It need scarcely be observed that, although the abnormal condition now described as Spasm is evidenced by a disorder of muscular fibres or muscles, the cause of this disturbance is always resident in the nervous system. There is a very important and interesting variety in the connection which subsists between the nervous stimulus and the phenomenon of muscular contraction. The former may be central, that is, operating directly on the great nervous centres, the brain, or spinal cord; or, and in the case of Local

[1] Romberg, Lehrbuch der Nervenkrankheiten des Menschen: Hypercineses, Krämpfe.

Spasm this is far the more frequent, the irritation is peripheral, and consequently the induced action is reflex.

Our knowledge of the causes of Local Spasms is as yet far from being perfect, and in not a few instances the attempt to determine these, notwithstanding the most careful inquiry, signally fails. The etiology of general convulsive disorders is indeed more advanced, and may serve to elucidate doubtful points in relation to the more limited and less serious affection.

The late Dr. Graves of Dublin was one of the earliest to direct attention to the frequency with which various nervous affections, of which Spasm is one, and not the least interesting, are dependent on reflected nervous irritation. He has graphically described the sudden and complete relief afforded to a young lady, who had suffered most severely from spasmodic cough, after the discharge of a tapeworm, which had been effected by a large dose of oil of turpentine with castor oil.[1] The subject thus adverted to by Graves has more recently attracted the attention of several competent observers, more especially of M. Davaine in France,[2] and Dr. Heslop[3] of Birmingham. Their statements show that the presence of worms in the intestinal canal is a frequent cause of remote nervous phenomena, including Spasms, and throw doubt on the assertion of Romberg, that the influence of worms in producing convulsions has been formerly over-estimated. Again, a careful study of the whole phenomena in that most interesting disease, *spasmodic asthma*, has led to the conclusion that the spasmodic affection in it, seated in the smaller bronchial tubes, may be induced by an irritation of the nervous system, which is either centric or eccentric. In the former case the irritation is in the nervous centres themselves, the brain, or spinal cord. In the latter, and it is by far the more common in its occurrence, the irritation is applied at a distance from the nervous centres. This subject has been very fully and ably illustrated by Dr. Hyde Salter, in whose work examples the most interesting and conclusive as to the essentially nervous origin of asthma are to be found.[1]

In treating of what may be styled central asthma, Dr. Salter gave, among others, the following case:—A man about fifty was subject to epilepsy. His fits had certain well-known premonitory symptoms, and occurred with tolerable regularity about once a fortnight. On one occasion his medical attendant was sent for in haste, and found him suffering from violent asthma. The account given by his friends was, that at the usual time at which he expected the fit he had experienced the accustomed premonitory symptoms, but instead of their being followed as usual by the convulsions, this violent dyspnœa had come on. Within a few hours the dyspnœa went off, and left him as well as usual. At the expiration of the accustomed interval after this attack, the usual premonitory symptoms and the usual epileptic fit occurred. On several occasions this was repeated, the epileptic seizure being as it were supplanted by the asthmatic. Nothing seemed to be amiss with the lungs either before or after the attack. Dr. Salter truly observed, that such a case as this appears to admit of only one interpretation, that the particular state of the nervous centres that ordinarily threw the patient at certain times into the epileptic condition, on certain other occasions, from some unknown cause, gave rise to bronchial Spasm; that the essential diseased condition was one and the same, but that its manifestation was altered, temporary exaltation and perversion of the innervation of the lungs in the asthmatic paroxysm supplanting unconsciousness and clonic convulsion in the epileptic seizure. It has occurred to the writer to witness in one instance an alternation of phenomena bearing a close resemblance to that observed by Dr. Salter. The patient, a young man, was admitted to the Royal Infirmary of Edinburg, on the recommendation of Dr. Turner of Keith. He had for several months previously been subject to cerebral attacks, attended by loss of consciousness, and occasionally by convulsive movements of the muscles of the face and extremities. These continued to occur during the patient's residence in the hospital, observing

[1] Clinical Lectures, Lecture xl., Bronchitic Asthma, Cough.

[2] Traité des Entozoaires. Paris, 1860. M. Davaine remarks: "Tous les organes, pour ainsi dire, peuvent ressentir l'influence sympathique des vers du canal intestinal; la fausse perception des odeurs, la dilatation de la pupille, l'amaurose permanente ou passagère, l'exaltation de l'ouïe, la perversion du goût, le prurit et les fourmillements à la peau, témoignent de l'action sympathique des vers sur les sens; d'un autre côté, la somnolence ou les vertiges, les rêves fâcheux, *les spasmes*, les douleurs vagues, la toux, la dyspnée, les palpitations, les intermittences du pouls, la faim insatiable ou l'anorexie, la salivation, la qualité des urines, l'amaigrissement, témoignent également de leur action sur le système nerveux, sur les organes de la respiration, de la circulation, de la digestion, sur les sécrétions, enfin sur la nutrition."—Page 48.

[3] The Cerebro-spinal Symptomatology of Worms, especially Tapeworms: Dublin Quarterly Journal of Medical Science, vol. xxvii. 1859.

[1] On Asthma: its Pathology and Treatment. London, 1860. See also article on Asthma, Vol. II.

for a time the same periodicity which had antecedent to that time always distinguished them, when, on three separate occasions, and in the most distinct manner, an attack of asthma took the place of the more manifest cerebral disorder. The loss of consciousness and convulsive movements again recurred in a modified form: and after the lapse of several weeks, during which various remedies were employed, the patient left the Infirmary to return home, his condition having materially improved. Besides instances of the nature just alluded to, there are other examples of asthma, which, although in by no means so distinct a manner, must be held as caused by some impression taking origin in the nervous centres, and responding in a mysterious manner with certain feelings or emotions of the mind; such are the cases in which fear, excitement, and fatigue operate.

Now, passing to a very brief consideration of *bronchial* Spasm, dependent not on centric but *peripheral* irritation. Dr. Salter speaks of three degrees of remoteness of the application of the stimulus producing asthma, and consequently of three groups into which the reflex cases of the disease may be divided:—1st. Those in which the source of irritation is alimentary, and chiefly gastric. 2d. In which the irritation is more remote, but still confined to the organic system of nerves; as, for example, asthma produced by a loaded rectum, by the presence of tapeworm, or ascarides. 3d. Cases in which the cerebro-spinal system is the recipient of whatever irritation is the cause provocative of the attack, as, for example, was illustrated in a most remarkable instance recorded by Dr. Chowne, where the application of cold to the instep produced in the most direct manner the asthmatic paroxysm. Looking to the first, and by a long way the largest, of these three classes of cases, the nerve irritated is the gastric portion of the pneumogastric; through it the stimulus reaches the medulla oblongata, and from that portion of the nervous centre it is again transmitted to the bronchiæ by the pulmonary filaments of the same nerve. It is indeed of the highest importance in a therapeutical point of view to notice this chain of connection. We are thus called to recognize in the paroxysm of asthma a disease not unfrequently originating in disorder of the stomach; and it may be assumed, as a correct conclusion, that a large proportion of the sufferers from this severe spasmodic affection are to be relieved by attention being given to their diet and regimen. But even here we should be adopting too limited a notion of the influence of the digestive and assimilating processes in the production of asthma, did we conclude that those cases alone are examples of this nature, in which bronchial spasm is induced by reflex stimulation directly through the important nervous trunk—the pneumogastric. There are, over and above, numerous instances in which this direct communication of the influence exerted will not apply. In such the occurrence of the Local Spasm does not so speedily follow the introduction of food into the stomach as in many of the former cases, and therefore we must look for a somewhat different explanation. We find it in the disordered condition of the blood; the faulty assimilation is no doubt the primary cause of this, but the unhealthy blood is in such instances the direct irritant; by its operation on the nervous distribution through the lungs the bronchial spasm is caused. This humoral origin of asthma affords in all probability the most satisfactory explanation of the frequent occurrence of this nervous disorder in persons who are gouty. The accuracy of the view thus expressed is further evidenced by the circumstance that such sufferers are benefited by a plan of treatment which tends to eliminate the essential poison of gout from the system; often, indeed, are benefited by such a plan of treatment only. In these cases remedies need scarcely be directed to the chest: it may be possible to relieve, it is impossible to subdue, by antispasmodics a bronchial spasm so induced; but on the other hand, by acting freely on the great emunctories of the body, on the skin and kidneys, the disease is to be met and overcome.[1]

Allusion has been made to the production of bronchial Spasm as determined by reflex irritation, and also by an impure condition of the blood. The same precisely holds true of *cardiac* Spasm. The

[1] Laennec, who, while strongly insisting on the connection between asthma (asthme spasmodique) and catarrh, admitted the existence of a purely nervous asthma (sans aucune complication de catarrhe), has acknowledged the great difficulty there is in the satisfactory treatment of the disease. "Beaucoup de moyens," he remarks, "peuvent être opposés aux troubles de l'influence nerveuse qui constituent principalement l'asthme: *mais ici, comme dans toutes les affections nerveuses*, rien n'est si variable que l'action des médicaments: les remèdes qui réussissent le mieux chez un grand nombre de sujets sont sans efficacité pour beaucoup d'autres; et chez le même individu tel moyen qui avait produit d'abord des effets héroïques, et d'une promptitude surprenante, devient tout à fait inefficace au bout d'un petit nombre de jours. Il faut successivement en essayer plusieurs, et souvent de très-disparates: nous allons, en conséquence, parcourir les diverses séries de moyens dont on a tiré le plus d'avantage dans l'asthme."—*Traité de l'Auscultation Médiate: Affections Nerveuses du Poumon.*

irregular, unrhythmical, and painful contractions of the heart known under the name of *palpitation*, are found in close connection with various derangements of the general health, and of special organs. Among the latter, those of the alimentary canal, but particularly of the stomach, and of the uterus, occupy the chief place. Perhaps the most painful of all the forms of cardiac palpitation is that resulting from either an imperfect depuration of the blood, or from a regular blood impoverishment, or anæmia, as is so frequently observed in cases of amenorrhœa and chlorosis.

We pass to a brief consideration of Spasm as occurring in the muscular organs which constitute the alimentary canal. It affects *the stomach and intestines* as well as the *œsophagus and pharynx*, while the severe pain determined by its occurrence in any part of the alimentary tract is very generally accompanied by other and various symptoms which cannot with any propriety be referred to now. Painful peristaltic spasm of the intestines is usually known under the name of *colic*. During its occurrence, and as affording proof of its occasional violence, *intussusceptio*, and prolapsus of the rectum may take place. Foremost among the determining causes of colic is to be placed the presence of indigestible articles of food and morbidly altered secretions in the intestinal canal. But, besides this, the influence of the emotions, and more especially of fear and fright, is well known; just as bronchial Spasm may be due to reflex nervous irritation, so may intestinal-spasmodic stricture (as it is called, to distinguish a temporary and functional from an enduring and organic contraction, similarly produced) have its seat in any part of the alimentary canal. In some instances the direct exciting cause is seated at a great distance from the induced disorder: of this nature no more common or manifest example can be given than that of colic, often very severe, resulting from the exposure of the lower extremities, it may be of the feet only, to cold and damp. *Spasm of the pharynx and œsophagus* is one of the most interesting of all the varieties of Local Spasms. It is of common occurrence, particularly in females, in whom it shows itself either as a reflex phenomenon dependent on uterine irritation, or—and this still more frequently—as one of the most striking features in a paroxysm of hysteria. It is not always an easy task to distinguish between spasm of the œsophagus due to organic disease and that which is simply the result of a nervous irritation. The cautious introduction of the probang or œsophageal bougie is the most ready and certain means for establishing the diagnosis.

An irritation of the pharynx or œsophagus, of the stomach, bowels, or liver, is sometimes the direct cause of *hiccup* or *singultus*, a spasmodic affection extremely interesting in its nature. Sudden powerful jerking inspirations, accompanied by a peculiar noise, and succeeded by a brief expiration, interrupting speech, distinguish hiccup. It is essentially a reflex phenomenon; in the vast majority of instances depending on some peripheral irritation, but occasionally, as its presence in apoplexy, meningitis, and hydrocephalus testifies, determined by a central cause. There seems to be some difficulty, in accounting for the occurrence of hiccup from an irritation of the phrenic nerve, as has been suggested by various writers; nevertheless it is consistent with the writer's observation in several instances of long-continued and distressing hiccup, that firm pressure exerted for a brief period over the lower part of the neck, corresponding to the situation of the scaleni muscles, so as to probably compress the phrenic, has led to its temporary and even entire arrestment. In singultus and in yawning, which resembles it in being of the nature of inspiratory convulsion—also in *sternutatio* or *sneezing*, where the expiratory function is involved—what is of consequence to notice is, as Romberg has pointed out, that the spasmodic action does not affect a single muscle, but, on the contrary, groups of muscles; and that these Local Spasms, more particularly the former, hiccup, while occurring as independent affections, are still more prone to assume the symptomatic character, affording evidence of the existence of some other malady, or distant irritation.[1]

Spasms of the *urinary bladder* and of the *urethra*—the latter commonly styled spasmodic stricture—are familiar to the surgeon. Vesical Spasm is not unfrequently a truly reflex phenomenon: this is witnessed on the introduction of the catheter or bougie, when violent and most painful efforts are made to evacuate the organ, even when at the time empty. Romberg insists on the action of the vesical muscles being due to an irritation of the neck of the bladder, that particular part being, as Sir Charles Bell demonstrated, the most vascular and the most sensitive portion of the viscus. It is when the catheter reaches, or the calculus touches, the neck of the bladder, that the ischuria is produced; and the intense pain is seen to subside whenever the irritating body is removed from that particular portion of

[1] "Häufiger als auf einzelne Nervenbahnen beschränkt, kommen die krampfhaften Athembewegungen zu Gruppen associirt vor, entweder selbständig, oder was öfter der Fäll ist, abhängig, und in Begleitung von andern Affectionen." (Loc. cit. p. 354.)

the organ. The irritation upon which vesical spasm depends may, as we have seen to hold true of other forms of Local Spasm, be distant from the induced phenomenon. It may be resident in the kidneys, or in any part of the intestinal canal, but very specially in the rectum. Hemorrhoids are a frequent cause of vesical spasm; and it is well known in how distressingly severe a degree that is apt to occur after the operation of their deligation. Exposure of the surface of the body, especially of the feet, to cold and wet, and depressing mental emotions, act in the same way.

As our object in this article has been, not to illustrate every example of Local Spasm, but rather to indicate the nature of this special morbid action by a brief consideration of some of its more important and most frequently occurring varieties, we shall now take a very rapid survey of a few other forms, and bring our remarks to a conclusion by offering some general observations with a special reference to treatment. There is a peculiar variety of Local Spasm affecting certain muscles of the face, and giving while it lasts a very strange aspect to the individual. In the *histrionic spasm of the face*, by which title this affection is known, there are, in the language of Romberg, "grimaces, alternating or lasting, on one side, seldom on both sides, of the face."[1] Pain is occasionally, but by no means necessarily, an accompaniment of the disordered muscular action. A local malady essentially, because affecting the muscles governed by *one* nerve, the seat of the spasm is in some instances still further localized by there being only one of the branches of the seventh pair involved. Of the latter are *blepharospasmus*, or spasm of the eyelids, and the *risus caninus*. The peculiar convulsive grin thus named is caused when the molar and labial branches are affected. To it the terms *spasmus cynicus* and *sardonic laugh* are likewise applied. The relation of facial spasm to chorea must not be overlooked; this association has been frequently noticed: and it is also a matter of not unfrequent observation that the Local Spasm lasts in some cases for a considerable period after the disappearance of the general nervous disorder with which it had been in the first instance connected.

Masticatory Spasm is witnessed in its most formidable degree when, as trismus, it accompanies, or is itself the chief element in, tetanic convulsions. In a much milder degree spasm in the muscles which are supplied by the motor division of the fifth pair is seen as a reflex action, determined, as in children, by the presence of worms in the intestinal canal, or by the progress of dentition. The spasm of the muscles is sometimes associated with a grinding of the teeth. To the occurrence of the latter symptom in persons of the gouty diathesis attention was called by the late Dr. Graves. Such grinding of the teeth continued for years as a daily habit, and produced very remarkable changes in the conformation of these organs, affecting sometimes one side of the jaw, sometimes both; so that in confirmed cases the teeth were frequently found ground down to the level of the gums.[1]

Spasm of the muscles of the eye, dependent on an irritation of the third, fourth, or sixth nerves, is seen in *strabismus*—which is to be distinguished from the paralytic form by the movement of the eyeball in other directions being in the former case possible—and in *nystagmus*. These spasmodic affections equally with others acknowledge a peripheral or central origin. Both are of common occurrence in connection with intestinal and dental disorders, but they are also not unfrequently the indications, sometimes among the very earliest, of mischief, inflammatory or otherwise, commencing at the base of the brain.

Painful Spasm of the muscles of the extremities are of very frequent occurrence; and with this affection, more especially seated in the lower limbs, and then in the calves, we are especially familiar under the name of *cramp*. The attack of cramp is usually sudden; and it frequently occurs at night, the person in bed being awakened from sleep by the seizure. During its continuance the muscular fibres are gathered up into a hard knot, which is always easily felt by the touch, and may often be seen. The pain is very severe, and produces a feeling of sickness and depression, which may even lead to syncope. The patient not unfrequently gives utterance to an irrepressible exclamation or scream. Cramp usually lasts only for a few moments; it may, however, continue for minutes, and even hours. A sudden cessation of the spasm may occur, or a more gradual relaxation of the muscular fibres ensue; but in either case, if the attack have been at all severe, sufficient injury during its continuance has resulted to the sensory nervous filaments as to cause a feeling of soreness, always increased by touch, and frequently an inability fully to exert the affected limb or other parts for some time. The irritation of the sciatic nerve, upon which the painful spasm of the muscles of the calf depends, is intimately connected with disorder of the stomach and bowels, and is also particularly prone to occur in persons of the gouty and rheumatic habits. [See *Athetosis*, in this volume.—H.]

[1] Loc. cit. "Mimischer Gesichtskrampf."

[1] Clin. Med., "Gout."

In Asiatic cholera the occurrence of intensely painful cramps contributes, as is well known, largely to the sufferings of its victims. Again, in persons of intemperate habits there is sometimes observed a tendency to the development of severe spasmodic action in the muscles, of the extremities, more especially, but likewise of other parts of the body. In one instance which fell under the writer's observation, a patient, having recently recovered from an attack of delirium tremens, was seized with most violent and painful spasm of the muscles of both upper and lower extremities, during which the fingers were powerfully flexed and bent inwards on the palms of the hands, as in the carpal contractions of children. So severe was this case, that a syncopal depression very threatening in its character, occurred. After lasting for several hours, and exhibiting for many days a marked tendency to recur, the affection passed off, and the patient entirely recovered both health and strength.

In the *treatment* of local, as of general, Spasms, the great object is to remove the cause on which they depend. In the brief consideration of the different varieties of Local Spasm now offered it has been shown that in a large proportion of cases the excited muscular action is induced by reflex action; that the direct exciting cause is a distant nervous irritation. Fortunately the removal, or at all events the lessening, of this irritation is in many instances within the power of our art. Again, in those cases, of the frequent occurrence of which proof has been afforded, which are characterized by a morbid state of the blood, *e. g.*, gouty or rheumatic, we may often be successful in our treatment by paying due attention to the therapeutical indications—in other words, by the employment of an alterative or eliminating plan, suggested by the peculiarity of each individual case. We may as effectually subdue the morbid action of Spasm as we are constantly enabled, by the use of suitable remedies, to relieve that of pain in neuralgia. In addition, we possess in various agents a power of controlling or completely removing such excited nervous action as induces Local Spasm: not, indeed, one upon which we can invariably rely, because we are often disappointed in the results; nevertheless the remarkable therapeutical effects which succeed the exhibition of various of the antispasmodic and calmative remedies is such as to convince us of their efficiency. Our knowledge, moreover, regarding the action of such remedies is on the increase. It is only quite recently that a valuable addition has been made in the bromide of potassium, the operation of which in removing the painful cramps of cholera, not less than in many instances averting the convulsive seizure of epilepsy, has been witnessed by numerous observers.[1]

Pressure firmly exerted on the thigh relieves a violent cramp of the calf. while, according to Dr. Wise, the application of a tourniquet so as to compress the blood-vessels will banish the exhausting muscular contractions in cholera.

Finally, in the treatment of such exalted nervous action as determines Local Spasm, as in the proper management of every form of derangement of the nervous system, however slight or severe, let the potent influence of peculiarity in psychical constitution, and of the ready susceptibility in some to the operation of all manner of external impressions, not be lost sight of.

TORTICOLLIS.

By J. Russell Reynolds, M.D., F.R.S.

Definition.—A spasmodic condition of the muscles of the neck—generally clonic, but rarely tonic—whereby the head is displaced to one side, or towards one shoulder, or is thrown backwards; occurring almost exclusively in adult life, and characterized by great obstinacy and chronicity.

Synonyms. — Wry-neck; spasmodic wry-neck; spasm in the muscular distribution of nervus accessorius Willisii, and of the superior cervical nerve (Romberg).[2]

[1] See Note on the Therapeutical Effects of Bromide of Potassium, by James Begbie, M.D.; Edin. Med. Journ. 1866. Also, The Actions of Bromide of Potassium upon the Nervous System, by J. Crichton Browne, M.D.; Ibid. 1865.

[2] Syd. Soc. Transl. of Manual of Nervous Diseases of Man, vol. i. p. 316.

CAUSES. — So far as I have seen, the male sex has been slightly more frequently affected than the female; but the difference is so small, that its existence is of no diagnostic value. The affection has sometimes originated, and recurred, or been exaggerated, during pregnancy. With only few exceptions, the cases that I have seen have first presented symptoms after thirty years of age; and the majority after forty. There has been no one thing, nor any combination of circumstances, which has occurred with such frequency as to warrant a belief in its operation as an *exciting cause*. Once a strained position, maintained for a long time; occasionally exposure to cold; sometimes a sudden shock, either mental, moral, or physical; and at other times the presence of long-continued anxiety, or the recurrence of pregnancy, has been referred to by the patient as the cause of symptoms; but, in regard of such modes of causation, we can see distinctly that which might lead to disturbance of the nervous system of any kind whatsoever, but we fail to see anything which should conduce to this special form of derangement.

In one case that I have seen the symptoms were preceded by hemiplegia; in another by paralysis agitans of the side from which the head was turned; in a third, and fourth, and fifth, there was previous "writer's cramp;" in a sixth there was histrionic spasm of the face; but in the majority of cases the nervous system had exhibited no prior derangement, and had continued free from ulterior disturbance for a long period of years.

The position in life and the occupations of those who have suffered from Torticollis have varied widely, and I have not been able to attribute the malady with anything like constancy to that common cause of nervous disease—overwork.

SYMPTOMS.—There is great similarity in the symptoms presented by different individuals, when once the disease is established, and is free from accidental complications. Sometimes the *commencement* is sudden, but much more commonly it is gradual, and often so insidious at first that the real nature of the malady is overlooked. The patient feels uneasy in the neck, thinks that something is wrong with his cravat, or with his pillow, and only after several months discovers for himself, or is told by others, that his head is not straight. There is with this want of symmetry some uneasiness in the neck, extending from the occipital protuberance downward to one of the shoulders, and sometimes onwards into the arm, or even forearm. As the malady *advances* the uneasiness becomes greater, and sometimes amounts to definite pain, felt usually in the same direction. The pain is increased by voluntary efforts to bring the head into the middle line, but sometimes attains its maximum when the head is carried round to the furthest point possible by the spasmodic movement. The pain is not severe, but generally of dull, aching character; and often is relieved by lying down, and keeping the head still by resting it upon a pillow.

Observed casually, a case of medium severity would give the impression to a bystander that the patient's cravat was uncomfortable, and that he was trying to make it less so by moving the head, in a somewhat restless manner, towards one side; or that he was making some attempt to look at an object on one side of him, which object he could not "get his head round" sufficiently far to see conveniently.

Upon more careful examination it is seen that the head is constantly being moved, by a succession of jerks, in such manner that the occiput is depressed, and the chin raised, and that the movement is in a definite direction, hour after hour, and month after month. Early in the case the individual is able so far to antagonize the spasm, by a simple voluntary effort, as to bring the head into the middle line, or even beyond it; but as time passes on this often becomes impossible, and the hands are used to pull the head back again into its proper position.

When Torticollis has existed for a few months only, the head presents a constant series of movements — the spasm and the voluntary effort so balancing one another that the effect is that described above. But when it has lasted for a longer period, the head is habitually "carried on one side;" for the voluntary interference with the spasm, although frequent—if not constant—does not suffice to bring the head into a central position, being overcome by the spasmodic contraction. Sometimes, even under these circumstances, a very strong voluntary effort may restore momentary equilibrium; but the effort is attended by distress, if not by pain, and is often followed by an exaggeration of the spasm.

The muscles of the neck on the side from which the chin is turned are found hard, contracted, and often hypertrophied; those on the opposite side are frequently soft, and sometimes wasted. Early in the history of Torticollis it would seem—so far as my experience extends—that the deeply-seated muscles at the back of the neck are the most affected: the sterno-mastoid, at such time, being often free from spasm. At a later period the sterno-mastoid is found hard, frequently hypertrophous.

Occasionally the muscles of the shoulder are so involved that the acromion is raised;

more rarely the muscles of the face present histrionic spasm; and not unfrequently there is some difficulty in controlling the movements of the arm. I have noticed sometimes difficulty of deglutition, and in a very few cases some morbid condition of motility in the leg: but these symptoms must be regarded as complications rather than conditions of the disease now under consideration; for it more frequently happens that the muscles of the neck are alone involved in the morbid contraction.

As a rule, to which the exceptions are very rare, the spasms cease during sleep; and not only so, but when the patient lies down and supports the head. They are increased by all that lowers or disturbs the general health, and by emotional excitement.

The electric irritability of the contracting muscles I have found much increased when tested by faradization: the electric sensibility is sometimes so greatly augmented that an interrupted current, not in the least degree painful on the healthy side, was perfectly intolerable when passed through the seat of spasm. It has appeared often that the relaxed muscles, on the side opposite to the contraction, have exhibited less than their normal contractility; but I have never found them so defective that it was impossible to restore the head to equilibrium by their direct faradization. The battery current, when continuous, and passed through the contracting muscles, relaxes the spasm and allows of temporary equilibrium; but, when interrupted, its action is similar in kind to that exerted by the induced current of faradization: there is, however, less intensity of contraction, and much less display of electric sensibility. The effects of either the battery current or of faradization appear to be singularly transient, in whatever manner they may have been produced. It has often happened to me to see that a head which had been maintained *in equilibrio* for many minutes, and that day after day for a considerable number of days, returned at once to its spasmodic jerkings the moment that the application was suspended. Sometimes it has been obvious that the spasm was subsequently increased by the electricity.

The side to which the twisting occurs has been sometimes the right, sometimes the left. There appears to be no special proclivity to the affection of one side rather than the other in either sex; but when once the malady has shown itself, its pertinacity is remarkable: it remains in exactly the same position, with slight tendency to extend; or it may in rare instances disappear for eight or nine years, and then return to the muscles that it had previously affected. In many cases *progress* is so slow that no change is observable after several years—*i. e.* no change as to locality—whereas in others the malady seems to extend either upwards or downwards, and involve muscles not implicated at the first. In this manner the face may be distorted or the arm may be rendered partially useless by either rigidity or weakness; the head becomes more or less fixed in an oblique position, the ear of one side being drawn down to the shoulder, and the chin thrown upwards and outwards in the opposite direction. When left entirely to itself—*i. e.* when not interfered with by either the will, the ideas, or emotions of the patients, or by any influence from without—the spasm is tonic, and the head may remain for hours drawn to one side, but motionless. This is rarely, if ever, noticed early in the history of a case, and sometimes it is never observed; but, even when it occurs after several years' duration of the spasm, the slightest emotional disturbance or attempt at voluntary movement brings back the clonic contraction: and the only difference to be recognized between the early and the later stages of the malady is, that in the latter the head is never brought back to the position of exact equilibrium, and that there is less obvious movement of the head; for, as it seems, the habitual struggle between volition and clonic spasm is given up, and the latter, having gained the day, allows tonic spasm to take its place.

The mental faculties, the sensibility of the skin, the special senses, and the general health undergo no necessary changes in Torticollis, but I have often observed great mental depression. In some highly-marked instances there has been complete integrity of function in every direction; the one thing that has been wrong has been the disease itself. Sometimes the general health has been impaired, the patient has been anæmic and weak; but this has been frequently the result of the annoyance occasioned by the spasm, and very rarely the supposed cause of its development.

Numbness and anæsthesia may occur in the arm, together with œdema, when the scaleni are so much affected as to press upon the brachial plexus and its adjacent veins.[1] Insomnia is by no means unfrequent.

Diagnosis.—The symptoms that have been now described are sufficient when carefully regarded to enable the practitioner to distinguish this disease from every other. An accidental exposure to cold may produce "stiff-neck;" but here the head is permanently fixed in one position, and maintained therein, not by spasmodic rigidity of muscle, but by the fear

[1] Romberg, loc. cit. p. 317.

of pain which, as the patient knows, any movement may occasion. Such malady has its relations to pleurodynia, lumbago, and "muscular rheumatism;" it is sudden in its development, and temporary in its duration, and could only be accidentally mistaken for Torticollis. The opposite error is sometimes made—viz. that of regarding genuine spasmodic Torticollis as a simple "stiff-neck from rheumatism or cold." In its earliest stage, however, genuine Torticollis should be at once distinguished by the clonic character of the spasm, and freedom from pain on movement.

Injuries to the spine occasionally produce stiffness of the neck, and this to such a degree that the head may be permanently placed in some awkward position. In such cases the spasm is tonic; there is marked tenderness of the spinous processes, and with this some fulness or hardness around or behind the vertebral column; and there is also some impairment of the motor or sensory properties in the arms and legs.

In certain *organic diseases of the brain* accompanied by hemiplegia, there is sometimes Torticollis, just as there is synergic movement of the eyeballs; but the mode of onset of symptoms is such that a case of cerebral apoplexy cannot well be confounded with the malady now under consideration. The opposite mistake has, however, sometimes been made, and an individual who is beginning to suffer from Torticollis spasmodica has been supposed to be the subject of organic disease of the brain. For the distinction between these two very different conditions it is sufficient to bear in mind that in the one the disease is limited to the neck, in the other it occurs in combination with marked hemiplegia; that in the former the spasm is clonic, in the latter tonic; and that in the first the development of symptoms is insidious, gradual, and local, whereas in the second it is sudden, and of wide distribution.

It is enough to mention the existence of cases in which *growths*, benignant or malignant, may affect the position of the head, in order to prevent the occurrence of any errors in diagnosis.

PATHOLOGY.—Anatomical inquiry has not yet shown the locality or existence of any special lesion of the nervous centres with which Torticollis is necessarily associated. Physiological experiment has proved that it may exist when the spinal accessory nerve is irritated at its passage through the foramen lacerum,[1] or when injury is inflicted on certain muscles, upon the olivary body, or the auditory nerve.[1] The disease would appear to be one of those curious conditions—not yet fully understood—in which some "centre" of associated movements is so altered that there follows a disturbance of the normal equilibrium; a disturbance exhibiting itself at first by dynamic change, but subsequently leading to structural alterations in the affected muscles. It has its analogies in writer's cramp and histrionic spasm, and its peculiar and intimate pathology is a question as yet reserved for further investigation.

PROGNOSIS.—When once established,—*i. e.*, when fully developed and of three or four months' duration,—Torticollis is one of the most obstinate of maladies. It has sometimes yielded to treatment, under favorable circumstances; but it has almost invariably recurred, and has proved capable of resisting all efforts made for its relief.

When the case departs widely from the ordinary type,—as, for example, when the chin is drawn either backwards and upwards, or downwards and forward, by bilateral contraction of the muscles at the back of the neck, or at its front,—the prognosis is more favorable. Such cases frequently improve, and sometimes get well by rest, and other measures.

Unfortunate as the prognosis is with regard to the cure of this special malady, there is one ground for consolation,—viz. that it is not by any means necessary, nor is it at all highly probable, that the victim of Torticollis should suffer from any other nervous disease. Sometimes it forms but part of a general nervous disorder; but, as a rule, it exists alone; and although it may continue for many years, the source of great but measurable annoyance, it does so without entailing any danger to life, or any high probability of ulterior change. Prognosis, therefore, is based upon the duration of the disease, and its complication with other signs of nervous malady. When it exists alone, the patient may look forward to a troublesome and obstinate affection; but he may, at the same time, know its limits, and be directed to go on without fear of further mischief.

TREATMENT.—In its early stages Torticollis has yielded to various plans of treatment; iron, setons, moxæ, rest, mercurials, electricity, pressure on the cervical sympathetic, and the division of nerves or of muscles, have each been followed by a cure: but in the advanced stages no one, nor any combination, of these modes of treatment has availed to cure, or even to modify, the disease.

I have used all kinds of soothing appli-

[1] Volkmann, quoted by Romberg, loc. cit. p. 316.

[1] Brown-Séquard, Lectures, p. 194.

cations, have employed electricity in every form, and have failed to influence the disease when once it has become fairly fixed; but have found that the continuous current has been useful when the malady has existed for a few months only, and have also at that period seen notable advantage from the continued application of morphia by the method of hypodermic injection.

It would seem desirable to enjoin rest; to secure the regulation of the general health; to apply a moderate continuous current to the muscles which exhibit spasm, and a mild induced current to their antagonists; and to inject morphia, hypodermically, for a lengthened period. It is not essential that the morphia should be injected into the neck; it may be introduced into the arm or thigh, or any other convenient locality: but it is important that its use be steadily continued, and that the quantity injected be gradually increased until a definite effect is produced upon the spasm. Beginning with the tenth part of a grain, the quantity may be increased, if necessary, until two, or even three, grains are injected twice daily; and when the patient can bear this amount, the spasm has sometimes yielded. But it often happens that morphia, even by hypodermic administration, cannot be borne, from the fact of its producing nausea, constipation, and an amount of malaise that is greater than the evil it is intended to relieve; and in such cases the Torticollis is positively increased by the injection. Several patients whom I have known with Torticollis have positively refused to continue the injection of morphia from the misery which it has thus occasioned.

Mechanical contrivances have been employed in order to force the head into position; but these, although so managed as to be borne for a short time—*e. g.*, to enable a clergyman to get through a service, or a doctor to visit two or three patients in succession—are often found to be productive of so much annoyance, or even pain, that the patient would rather trust to his own hands or to the "chapter of accidents" in order to get through his work. The most simple, and at the same time most effective, appliance that I have seen for mild cases is that devised by Dr. Hearne of Southampton; but it has failed to be of service when the disease has been of long duration. Mr. Heather Bigg has constructed several machines which meet the difficulty for short periods of time; but I have not yet seen any apparatus which a patient with confirmed Torticollis could bear habitually.

Division of the nerves has been useless,[1] and division of the sterno-mastoid worse than useless, for it has led to an exaggeration of the spasm in the deeper-seated muscles at the back of the neck, as I had occasion to observe in a well-marked case that came under my notice some years ago.

LOCAL ANÆSTHESIÆ.

By J. Warburton Begbie, M.D., F.R.C.P.E.

The term Anæsthesia (*a* privitive, ἀΐσθησις, sensibility) indicates deprivation or loss of sensibility, and was first employed by the distinguished Cappadocian physician, Aretæus.[1] There exist three abnormal modifications of the function of sensation: *first*, it may be lost; *second*, it may be exalted (hyperæsthesia); *third*, it may be perverted.

By Local Anæsthesia we understand a morbid state of sensibility, in which the normal physiological sensation of a part is abolished entirely, or nearly so.

Since the introduction of ether and chloroform inhalation, for the purpose of destroying pain, it has been customary to describe these valuable agents as anæsthetics, and the condition of insensibility into which the person is thrown by their action as Anæsthesia. With this interesting phenomenon we have at present no concern.

In Local Anæsthesia the want or failure of the due impression must arise from a morbid state of the extremities of nerves, or of an afferent nerve ceasing to convey the impression to the sensorium, or of the sensorium itself.

Thus we are entitled to limit the seat of the morbid influence, because these three organs, or classes of organs, are

[1] ἦν δὲ ἀφὴ ἐκλείπη μούνη κοτέ—σπάνιον δὲ τὸ τοιόνδε—ἀναισθησίη μᾶλλον ἢ πάρεσις κικλήσκεται.—Περὶ Παραλύσεως. Περὶ Αἰτιῶν καὶ Σημείων Χρονιῶν Πάθων. Βίβλιον Πρῶτον.

[1] Romberg, loc. cit. p. 319.

concerned in the production of each sensation.

With precisely the same signification as Anæsthesia, the expression paralysis of sensation, or of the nerves of sensation, has been employed. It were better, however, to abandon the use of paralysis in this sense altogether, and to restrict it to the loss of power of motion. The intimate connection of paralysis and Anæsthesia is abundantly conspicuous: the latter is very frequently noticed as an antecedent phenomenon of the former, or they occur simultaneously; and while paralysis lasts Anæsthesia may continue, or sensation may be restored long before the recovery of the power of motion.

The special situations in which Anæsthesia is met with, or may be considered apt to occur, are various. For convenience of illustration the following classification may be made, and to the forms now to be mentioned attention will be very briefly directed: (*a*) Anæsthesia of the skin (cutaneous Anæsthesia). (*b*) Anæsthesia of muscular nerves. (*c*) Anæsthesia of sensorial nerves. (*d*) Anæsthesia of the fifth pair of nerves. (*e*) Anæsthesia of mucous surfaces. (*f*) Anæsthesia of the viscera.

(*a*) *Anæsthesia of the Cutaneous Nerves.*—The notable and lasting diminution, or the entire loss, of the tactile sense of the skin is what is understood by cutaneous Anæsthesia. It is by a careful examination as to the delicacy of tactile sensibility, and the perception of degrees of temperature, that we are enabled to determine the extent to which Anæsthesia of the surface exists. For the former purpose the mere statements of the patient will not suffice. Besides measuring the degree and determining the precise seat of Anæsthesia by the point of the needle, recourse must be had to the method of experiment suggested by Weber, testing the consciousness of the patient, while blindfolded, to the two points of a pair of compasses, placed at different parts upon the skin, or, which is still more satisfactory, employing the delicate little instrument known as the æsthesiometer of Dr. Sieveking. The ready and accurate determination by the patient of degrees of temperature, heat and cold, is impaired or destroyed: it is not uncommon to find hot things styled cold, and cold things hot. In marked instances of the cutaneous Anæsthesia the power of resisting the injurious influence of temperature is lost: and not only so, but, owing to a similar defect, superficial sores are readily formed on parts of the body exposed to even a slight degree of pressure. Evidence of the derangement of the circulation is afforded by a change in the color of the affected part; it is apt to become livid or blue in appearance, and extravasations of serum, and even of hæmatin, occur. Distressing sensations are experienced by the patients,—chiefly numbness and pricking; also formication.

In alluding to the treatment of cutaneous Anæsthesia, the distinguished German writer on nervous diseases truly observes, "Die Behandlung der Anæsthesia cutanea war bisher eine *oberflächliche*, im wahren Sinne des Wortes;" but while this is to be regretted, we may reasonably anticipate an increase of our knowledge, owing to the much more satisfactory manner in which the causes and seat of disease have of late, and are at the present time, being investigated.

(*b*) *Anæsthesia of Muscular Nerves.*—The loss of the power of motion is usually unassociated with any marked degree of muscular Anæsthesia. On the other hand, instances are on record in which a very perfect insensibility to pain has existed in muscles, while the power of moving them has been retained. It is of the utmost importance to distinguish between the loss of tactile sensation (cutaneous Anæsthesia) and the definition of sensation in muscles, for without carefulness in examination these two are capable of being, and in some instances have no doubt been, confounded. Romberg makes the interesting observation, that muscular Anæsthesia, without the loss of or any damage done to tactile power, exists in tabes dorsalis.[1]

(*c*) *Anæsthesia of Sensorial Nerves.*—The nerves of special sense which thus suffer are the optic (Anæsthesia optica), the Auditory (Anæsthesia acoustica), the Olfactory (Anæsthesia olfactoria; Anosmia), and the Gustatory (Anæsthesia gustatoria; Ageustia). To the many interesting affections included under these terms—for example, amblyopia and amaurosis under optic Anæsthesia—it is not desirable to make any reference now. Such important diseases demand a separate and detailed consideration not contemplated in this System of Medicine.

(*d*) *Anæsthesia of the Fifth Pair of Nerves* (Facial or Trigeminal Anæsthesia).—Physiological experiments have demonstrated the remarkable effects produced by section of the fifth pair; of these, insensibility of the face, eye, nostrils, cavity of the mouth and tongue, is the most conspicuous; while the extent of the Anæsthesia is of course determined by the nervous injury being limited to one or more branches, or, on the other hand, involving the trunk before division. Experimental inquiry, as well as clinical observation, have further shown that when injury or lesion of the nerve exists within the cranium, the resulting phenomena are not such as are included in Anæsthesia merely, but paralysis and impairment or

[1] Muskelanästhesie.

loss of special sense are also induced. Romberg,[1] in directing attention to the different diagnostic symptoms, has indicated certain very important particulars, as follows: (*a*) The more the Anæsthesia is confined to single filaments of the fifth pair, the more peripheral the seat of the cause will be found to be. (*b*) If the loss of sensation affects a portion of the facial surface, together with the corresponding facial cavity, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium. (*c*) When the entire sensory tract of the fifth nerve has lost its sensation, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease. (*d*) If the Anæsthesia of the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be assumed that the cause is seated at the base of the brain. Thus facial Anæsthesia, as a phenomenon of disease, may be in itself a simple, really trivial, affection, or it may be the indication of serious organic disease. In the former case it will be apparently independent and isolated; in the latter, linked with other striking features, its significancy will as little escape observation as its existence.

Facial Anæsthesia in some instances comes on gradually; in others its occurrence is sudden. Neuralgic pain, or a condition of local hyperæsthesia, may precede its development; while facial palsy and facial Anæsthesia are occasionally associated.

(*e*) and (*f*) *Anæsthesia of Mucous Surface, and of the Viscera.*—The morbid condition in such circumstances must depend on a failure of the sympathetic to conduct the impression to the brain; but, as a general rule, impressions made on the ganglionic nervous system are not thus conveyed, and it requires a powerful irritation, or condition of notable hyperæsthesia, in order that a consciousness of their existence should be established. The inquiry into the operation of the organic nervous system is one of very great difficulty, and Romberg has truly remarked in regard to it, "Von vorn herein bekennen wir unsere unbekanntschaft mit diesen Zuständen, die bisher nicht einmal zur Sprache gekommen sind, und deren Forschung mit grossen Schwierigkeiten verbunden ist."

[1] Anästhesie des Quintus, Lehrbuch der Nervenkrankheiten.

INDEX OF VOL. I.

ABDOMEN, enlargement of, in rickets, 476, 482
pain and tenderness of, in measles, 107
in typhoid, 204
pain in, in cerebro-spinal meningitis, 297, 299
in tetanus, 971
retraction of, in meningitis, 810, 821
swelling of, in dysentery, acute, 376
tenderness of, in dysentery, mild, 375
acute, 376
Abdominal diseases, connection of, with melancholia, 590
Abortion, in influenza, 43, 45
malignant smallpox, 132, 137
relapsing fever, 279
smallpox generally, 138
syphilis, 430
typhoid fever, 208
typhus, 261
yellow fever, 289
under influence of malaria, 354
Abortive epilepsy, 772
Abscess of the brain, a cause of convulsions, 760
article on, 934
diagnosis of, from tubercular meningitis, 831
Abscesses, ecchymotic, in yellow fever, 289
gouty, 517
in glanders, 189
in pyæmia, 333, 335, 346
in scarlet fever, 86, 90
intracranial, in tertiary syphilis, 433
in typhus, 262
of brain, in pyæmia, 334
of cavity of abdomen, in typhoid, 208
of ear, in smallpox, 133
of heart, in pyæmia, 332
of intermuscular cellular tissue, in smallpox, 133
of kidney, in pyæmia, 333
of liver, in dysentery, 380
of lungs, in pyæmia, 332, 345
of lymphatic glands, *see* Bubo.
of muscles, in pyæmia, 334
of pharynx, in diphtheria, 76
of prostate, in pyæmia, 324
of spleen, in pyæmia, 333
of subcutaneous cellular tissue, in dengue, 101
of testicle, in pyæmia, 334
of tongue, in pyæmia, 334
of tonsils, in diphtheria, 76
Abscesses—
post-pharyngeal, in diphtheria, 76, 91
treatment of, 97
secondary, in pyæmia, 331, 332, 345, 346
sub-periosteal, in syphilis, 433
Absorption of fluids from eruption, a probable cause of secondary fever in smallpox, 133
Abstinence from drink, essential in treatment of alcoholism, 684
relation of, to the induction of delirium tremens, 671
Accelerated cow-pox, 160
Acephalocysts, in the brain, 899
in the spinal cord, 1016
Acetic acid, uselessness of, in scurvy, 456
Acids, mineral, in syphilis, 437
Acne, in gout, 520
in interval between secondary and tertiary stages of syphilis, 439
Acne of the face, diagnostic value of, 682
due to alcohol, 677
Aconite, external application of, in gout, 541
to rheumatic joints, 572
internal administration of, in measles, 115
in rheumatism, 570
Aconitine, external application of, in gout, 541
Acute alcoholism, 678
Acute mania, 596
treatment of, 619
Acute softening of the brain, 857
Acute specific diseases, the, a cause of convulsions in children, 745
of insanity, 591
[Acute spinal paralysis of adults, 1007]
Acute tuberculosis in the child, symptoms of pulmonary form, 828
of typhoid form, 828
Adventitious products in the brain, article on, 883
in the meninges, 843
Affective, or pathetic insanity, 603
Age, as a cause of disease, 22
at which rickets appear, 475
for vaccination, 182
Age, influence of, on diagnosis of hypochondriasis, 626
on mortality in influenza, 43
in relapsing fever, 260
in typhus, 266
on vaccination, 160
on occurrence of cerebro-spinal-meningitis, 308
of cholera, 390
of diphtheria, 62
of erysipelas, 322
of gout, 527, 536
of heart disease in rheumatism, 563
of hooping-cough, 48
of muscular rheumatism, 574
of parotitis, 120
of purpura, 463
of pyæmia, 344
of relapsing fever, 274
of rheumatism, 564
of rheumatoid arthritis, 551, 554, 555
of scarlet fever, 84, 95
of typhoid fever, 235
of typhus fever, 252
of varicelli, 125
on prognosis in cerebro-spinal meningitis, 304
in erysipelas, 327
in gout, 538
in rickets, 494
in smallpox, 137, 145
in typhus, 252, 265
of epilepsy, 774, 779
of insanity, 618
of chronic mercurial poisoning, 804
of neuralgia, 1030, 1041
of softening of the brain, 882
Age of parents, influence of, in production of rickets, 473
Age, predisposing to chorea, 697
to cerebral hemorrhage, 905
to chronic hydrocephalus, 836
to congestion of the brain, 848
to convulsions, 738
to cysticerci in the brain, 897
to epilepsy, 764
to essential paralysis of children, 1004
to general paralysis of the insane, 607
to hydatids in the brain, 899
to hypochondriasis, 626
to hysteria, 631
to insanity, 588
to laryngismus stridulus, 741

Age, predisposing—
to locomotor ataxy, 988
to neuralgia, 1029
to paralysis agitans, 725
to sciatica, 1035
to simple meningitis, 814
to softening of the brain, 872
to tubercular meningitis, 818
to wasting palsy, 786
to writer's cramp, 734
Agminated glands, *see* Peyer's patches.
Ague, *see* Intermittent fever, 354
Air, change of, after cholera, 419
[beneficial in hooping-cough, 54]
in gout, 549
in hooping-cough, 56
in rheumatoid arthritis, 558
in rickets, 496
in syphilis, 438
condition of, in relation to cholera, 386
impure, as a cause of cholera, 388
of dysentery, 375
of purpura, 463
of pyæmia, 344
of rickets, 473
of typhoid fever, 241
of typhus, 252
Aix-à-Savoy waters in gout, 547
in muscular rheumatism, 575
Aix-la-Chapelle waters, in gout, 547
in muscular rheumatism, 575
presence of lithia in, 544
Albumen, increase of, in blood of cholera, 406, 414
in urine, in acute rheumatism, 563
in cerebro-spinal meningitis, 298, 302
in diphtheria, 65, 66, 67, 73
influence of, on prognosis in diphtheria, 77
in erysipelas, 324
in gout, 518, 523, 534, 537
pathology of, 534
prognosis of, 538
in intermittent fever, 353
in measles, 111
in purpura, 462
in pyæmia, 346
in remittent fever rare, 367
after scarlet fever, 91
in scarlet fever, 91
treatment of, in scarlet fever, 97
in the urine in typhoid fever, 208
an occasional sequel of typhoid fever, 209
in typhus, 260
treatment of, 268
an unfavorable symptom in typhus, 266
in yellow fever, 285, 286, 290, 292, 368
Albuminoid degeneration, *see* Amyloid.
Albuminuria, a cause of convulsions, 745, 759
chronic, resulting from cholera, 415
see also Bright's disease.
Alcohol, effects of, on the system, 671
Alcohol, effects of—
on the pulse, 671
elimination of, by the urine, 672
oxidation of, in the system, 684
test for, in the urine, 928
useful in treatment of chorea, 715
of neuralgia, 1046
of tetanus, 979
when taken in excess, a cause of cirrhosis of the liver, 677
Alcoholic beverages as a cause of gout, 527
no influence on rheumatoid arthritis, 554, 558
Alcoholic stimulants, in cerebro-spinal meningitis, 313
cholera, 416, 417, 418
chronic dysentery, 383
diphtheria, 78, 81
dysentery, 376
erysipelas, 327
gonorrhœal rheumatism, 579
influenza, 45
gout, 548, 549
intermittent fever, 361, 363
measles, 114
parotitis, 122
pyæmia, 352
relapsing fever, 280
rheumatism, 573
scarlet fever, 96
smallpox, 142
the plague, 317
typhoid, 249
typhus, 267
yellow fever, 294
Alcoholism, article on, 670
definition, 670
synonyms, 670
history, 670
etiology, 671
symptoms of the chronic form, 675
of the acute, 678
diagnosis, 681
prognosis, 682
complications, 683
pathology, 684
treatment, 684
Alcoholism, acute, diagnosis of, from apoplexy, 927, 928
Alcoholism, chronic, relation of, to hypochondriasis, 627, 630
Algide stage of cholera, 400
Alkalies, in acute gout, 539
in chronic gout, 543
in diphtheria, 82
acute rheumatism, 569
hooping-cough, 56
rheumatoid arthritis, 556
rickets, 495
scarlatinal rheumatism, 97
Alum, in diphtheria, 80, 82
in hooping-cough, 56
lotion for otorrhœa, 116
topical use of, in hemorrhages in purpura, 468
Amaurosis, a common symptom in locomotor ataxy, 985
a rare sequel of convulsions in children, 744
in cerebro-spinal meningitis, 300
in hereditary syphilis, 429
Amaurosis—
in relapsing fever, 279
in tertiary syphilis, 440
Amenorrhœa, a cause of purpura, 463
cure of, by influenza, 43
Ammonia, in the air of marshes, 353
in stage of collapse in cholera, 417, 418
in diphtheria, 82
in erysipelas, 328
in influenza, 45
in pyæmia, 351
in rheumatoid arthritis, 557
in rickets, 496
in scarlet fever, 96
in syphilis, 438
in typhoid fever, 247
in typhus, 268
salts of, in chronic gout, 545
Ammonia, acetate of, in gout, 540
in measles, 115
in muscular rheumatism, 575
in smallpox, 143
in typhoid fever, 247
in uræmia after cholera, 419
in yellow fever, 294
Ammonia, carbonate of, in chorea, 715
Ammonia, hydrochlorate of, in muscular rheumatism, 575
Ammonia, phosphate of, in gout, 545
Ammonia, muriate of, in neuralgia, 1045
Ammoniacum plaster in rheumatoid arthritis, 557
[Amyl, nitrite of, in epilepsy, 782]
Amyloid corpuscles in the brain, 844
numerous in the insane, 615, 616
Amyloid degeneration of organs in purpura, 463
of organs in rickets, 494
Anæmia after cholera, 415
after intermittent fever, 358
after rheumatism, 572
after scarlatinal dropsy, 92
a predisposing cause of rheumatoid arthritis, 554, 556
in hereditary syphilis, 430
in purpura, 462
in rickets, 493
in mother, a cause of rickets, 473
Anæmia, predisposes to insanity, 590
to neuralgia, 1027, 1030
Anæmic bruit in ague, 358
Anæsthesia, hysterical, 636
complicating facial neuralgia, 1038
sciatica, 1036
paraplegic, in myelitis, 958
Anæsthesia, in yellow fever, 292
Anæsthesia, local, article on, 1064
definition, 1064
cutaneous anæsthesia, 1065
muscular, 1065
facial, &c., 1065
Anæsthesia, muscular, article on, 783
definition, 783
nomenclature, 783

Anæsthesia, muscular—
symptoms, 783
causes, 784
diagnosis, 784
pathology, 785
prognosis, 785
treatment, 786
Anasarca, *see* Œdema.
Aneurisms, intra-cerebral, 895
a cause of fits, 759
of death, 759
rupture of, a cause of cerebral hemorrhage, 904, 910
Aneurisms, miliary, in the brain, found after death from chronic cerebral congestion, 852
from chronic softening of the brain, 877
from general paralysis, 615
pathology and morbid anatomy of, 894
predispose to cerebral hemorrhage, 907
Angina, a consequence of scarlet fever, 90
in scarlet fever, 86
Angina pectoris, treatment of, by arsenic, 1043
by sulphuric ether, 1045
Angular curvature of the spine, 1017
Ankylosis of the fingers after dengue, 103
of joints in gout, 514, 516, 525
favored by local bleeding, 540
from gonorrhœal rheumatism, 576, 577, 578
Anomalous smallpox, 129, 132
Anterior columns of the spinal cord, functions of the, 943
[Anterior cornua of spinal cord, affected in wasting palsy, 796
in infantile paralysis, 1005]
Anterior pyramids of the medulla oblongata, functions of the, 943
Anterior roots of the spinal nerves, functions of the, 942
atrophy of, in wasting palsy, 791
Antimony, in acute mania, 621
in acute rheumatism, 568
in chorea, 714
in delirium tremens, 688
in dengue as an emetic, 104
in hooping-cough, 56
in influenza, 46
in purpura, 467, 468
in relapsing fever as an emetic, 280
in rickets, 495
in scarlatinal dropsy, 97
in typhus, 268
[Antiphlogistic treatment, 31]
Antiscorbutics, 458 *et seq.*
Anus, a frequent seat of erysipelatous inflammation, 323
condylomata of, in syphilis, 424, 441
Anxiety, excessive, a cause of writer's cramp, 734
Aphasia, after infantile convulsions, 744
Aphasia—
an occasional symptom of meningitis, 869
of abscess of the brain, 938
causes of, 861, 911
following epilepsy, 753
varieties of, 861
Aphemia, 861
Aphonia, hysterical, diagnosis of, 642
treatment of, 645
Aphthous ulcers of mouth, in dengue, 101
in measles, 110
Apnœa, in diphtheria, 67
in erysipelas, danger of, 327
treatment of, 329
Apoplexy, a consequence of cerebral congestion, 846
of cerebral hemorrhage, 920
of epilepsy, 776
of mercurial poisoning, 804, 805
of softening of the brain, 858
article on, 902
definition of the term, 902, 920
diagnosis of, from alcoholism, 928
from concussion of the brain, 929
from opium poisoning, 929
from uræmia, 881, 930
differential diagnosis, 880
the capillary form (of Cruveilhier), 863, 874
the serous, 930
simple, 931
without local paralysis, 927
Apoplexy, cerebral, in gout, 518
in purpura, 461, 464
pulmonary, in diphtheria, 76
in purpura, 461
in pyæmia, 332
Apoplexy, congestive, relation of, to cerebral hemorrhage, 851
[Apoplexy in hooping-cough, 52]
[Apyretic remedies in typhoid fever, 250]
Apyrexia, period of, in intermittent fever, 355
incomplete in remittent fever, 355
Arachnitis, in hereditary syphilis, 430
Arachnoid hemorrhage, 841
a cause of chronic hydrocephalus, 836
Arachnoid membrane of cord, supposed inflammation of, in yellow fever, 291
Arachnoid, opacity of, after chronic mania, 614
thickening of, in cerebro-spinal meningitis, 305
Areola, surrounding pustules in smallpox, 130
of vaccine vesicle, 159
Arcolar tissue, infiltration of, in erysipelas, 323
Arsenic, in cerebro-spinal meningitis, 313
in glanders in the horse, 192
in hooping-cough, 55
Arsenic—
in intermittent fever, 362
in muscular rheumatism, 575
in purpura, 468
in pyæmia, 350
in relapsing fever, 280
in rheumatoid arthritis, 557
Arsenic, value of, in treatment of chorea, 712
of neuralgia, 1043
Arteries, atheroma of, in general paralysis, 614
a cause of cerebral hemorrhage, 907
Arteries, morbid anatomy of, in pyæmia, 335
supposed paralysis of, in cholera, 412
Arthritis, rheumatoid, *see* Rheumatoid arthritis.
[Arthropathy of ataxic patients, 987]
Articular rheumatism, *see* Rheumatism.
Articulation, impairment of, in general paralysis of the insane, 605
in abscess of the brain, 938
in softening of the brain, 860
see also Speech, changes in.
Articulations, *see* Joints.
Arytenoid cartilages, gouty deposits in, 525
Ascarides, a cause of roseola, 106
Ascarides lumbricoides, vomiting of, in cerebro-spinal meningitis, 301
Ascites, as the result of syphilitic disease of the liver, 434
Ash-leaves in chronic gout, 545
Asphyxia, in diphtheria, 67
treatment of, 82
[Assafœtida in hooping-cough, 52]
in rheumatoid arthritis, 557
enemata, in typhoid fever, 248
Asthenia, in diphtheria, 66, 63
Asthma, a species of, dependent on gout, 515
spasmodic, causes of, 1056
Astringents, in cholera, 416
[Athetosis, article on, 731]
Atrophy, muscular, diagnosis of, from muscular rheumatism, 575
Atrophy, muscular, progressive, 786
Atrophy, muscular, from injury to nerve-trunks, 1050
from essential paralysis, 1006
from lead palsy, 790
Atrophy, of the spinal cord, 1015
found after death from paralysis agitans, 727
from wasting palsy, 791, 792
Atropia, external application of, in gout, 540
to rheumatic joints, 572
subcutaneous injection of, in neuralgia, 1045
Auditory nerve, irritation of, a cause of vertigo, 694
Aura epileptica, 752, 770

BADEN-BADEN waters, presence of lithia in, 544
use of, in gout, 547
Bael fruit, in scorbutic dysentery, 382
Bandaging of the head, in chronic hydrocephalus, 839
Barberry in intermittent fever, 262
Barèges waters, in muscular rheumatism, 575
Barometric pressure, influence of, on spread of cholera, 387
Bath waters, in gout, 547
Baths, in rickets, 495
in rheumatoid arthritis, 558
cold, after measles, 116
hip, in chronic dysentery, 383
hot air, in gout, 540, 545
in hydrophobia, 200
in acute rheumatism, 570
hydrochloric acid, in rickets, 495
mercury, for syphilis, 438
salt, in rickets, 495
Turkish, in chronic gout, 545
in gonorrhœal rheumatism, 579
vapor, in gout, 540, 545
warm, in cerebro-spinal meningitis, 313
in cholera, 418
in mild dysentery, 380
in parotitis, 122
in pyæmia, 350
to prevent profuse sweating, 123
at the end of smallpox, 145
Baths, in treatment of catalepsy, 658
of chorea, 716
in acute mania, 620
in paralysis agitans, 730
Bathing after measles, 116
Battley's solution in hooping-cough, 55
Bed-clothes, kicking off, in rickets, 476
Bedsores, in cerebro-spinal meningitis, 303
in cholera, 404
in typhus, 257
tendency to, in myelitis, 961
[Beef-tea without filtration, 114, 470]
Belladonna, in cerebro-spinal meningitis, 313
external application of, in gout, 540
in muscular rheumatism, 575
to rheumatic joints, 572
pericarditis, 572
in rheumatoid arthritis, 558
internal administration of, in gouty affections of the bladder, 547
in hooping-cough, 54
supposed preventive of scarlet fever, 95
in treatment of myelitis, 964
of neuralgia, 1045
[Bell's disease, 508]
palsy, 1050
Benghazi, epidemic of plague at, 318, 319
Benign smallpox, 129, 132
Bibeerine, sulphate of, in intermittent fever, 362
Bile, absence of, in stools of typhoid, 207
alterations of, in typhoid fever, 215
condition of, in typhoid fever, 215
in blood in pyæmia, 346
in stools of rickets, 476
in urine in pyæmia, 346
in relapsing fever, 273
in scarlet fever, 88
vomiting of, in cerebro-spinal meningitis, 301
in dengue, 100
in relapsing fever, 273
in remittent fever, 367
in yellow fever, 285
Bilious fever, *see* Remittent fever, 365
Bismuth, in typhoid fever, 247
Bismuth, trisnitrate of, in diarrhœa in scurvy, 458
Bites of rabid animals, treatment of, 200
Black bile, the, 623
Black death, *see* Plague.
Black measles, 107
Black pock, 131
Black vomit, 285
chemical analysis of, 289
microscopical examination of, 289
in relapsing fever, 273
Bladder, hemorrhage from, in purpura, 461
implicated in gout, treatment of, 546
inflammation of, in gout, 520
irritability or paralysis of, in dysentery, 70
irritability of the, in spinal irritation, 994
paralysis of, in myelitis, 960
in spinal meningitis, 95
Blebs in purpura, 460
[Bleeding at the nose in typhoid fever, 202]
Bleeding, in treatment of acute mania, 619
of catalepsy, 656
of cerebral hemorrhage, 933
of infantile convulsions, 751
of meningeal hemorrhage, 842
of simple meningitis, 815
of sunstroke, 669
of tubercular meningitis, 835
[for yellow fever, 293]
[Blending of types of fever, 236]
Blindness, during erysipelas, 524
from syphilitic disease of the optic nerve, 435
in hydrophobia, in wolves, 197
in keratitis of congenital syphilis, 442
in scurvy, 453
Blisters, in acute rheumatism, 572
in gout, 541
in intermittent fever, 360
in muscular rheumatism, 575
in rheumatic pericarditis, 571
in rheumatoid arthritis, 557
in treatment of chronic meningitis, 817
of hysterical paralysis, 646
Blisters, in treatment—
of neuralgia, 1046
of spinal irritation, 998
to epigastrium for vomiting in cholera, 418
to the head in typhus, 268
uric acid in fluid of, in gout, 523
Blood, abdominal conditions of the, relation of, to insanity, 611
effusion of, in malignant measles, 108
in purpura, 461
in scurvy, 452, 456
examination of, in diagnosis of gout, 520, 537
in stools in cholera, 405
in dysentery, mild, 375
acute, 376
malignant, 377, 378
in urine after scarlet fever, 91
in cerebro-spinal meningitis, 302
in diphtheria, 68
in intermittent fever, 358
in malignant measles, 108, 111
smallpox, 131
in Pali plague, 318
in purpura, 461
in pyæmia, 346
in remittent fever, 367
in the plague, 315
in yellow fever, 290
less alkalinity of, in gout, 534
loss of salts from, in cholera, 414
odor of, in yellow fever, 288
state of, in cerebro-spinal meningitis, 306
in cholera, 400, 405, 410
in diphtheria, 72, 73
in gout, 520
in influenza, 43
in intermittent fever, 358
in intervals between attacks of gout, 521
in purpura, 463, 464
in pyæmia, 335
in relapsing fever, 279
in rheumatism, acute, 562
chronic, 562
sub-acute, 562
in rheumatoid arthritis, 553, 554
in rickets, 485
in scurvy, 455
in the plague, 317
in typhoid fever, 218
in typhus, 264
in variola maligna, 131
in yellow fever, 288
uric acid in, in lead poisoning, 530
Bloodletting, in dengue, 103
in diphtheria, 82
[for inflammation, 31]
in influenza, 45
in intermittent fever, 356, 360
in smallpox, 141
in the plague, 317
in typhus, 268
general, in acute rheumatism, 567
in cerebro-spinal meningitis, 313
in cholera, 419
in choleraic diarrhœa, 421

Bloodletting, general—
in dysentery, 381
in erysipelas, 327
in gonorrhœal rheumatism, 579
in gout, 540
in purpura, 467
in relapsing fever, 280
in remittent fever, 369
in rickets, 495
local, in cerebro-spinal meningitis, 313
in cholera, 419
in dysentery, 381, 384
in gonorrhœal rheumatism, 579
in gout, 540
in muscular rheumatism, 575
in parotitis, 121
in rheumatic pericarditis, 571
in typhoid fever, 248, 249
Bloodvessels, degeneration of, in purpura, 463
ulceration into, in scurvy, 465
Blows on the head, a cause of abscess of the brain, 934
of cerebral hemorrhage, 904, 929
of chronic hydrocephalus, 836
of congestion of the brain, 848
of meningitis, 814
Boils, in cholera, 404
in chronic dysentery, 377
in dengue, 102
in farcy, 184
in smallpox, 133
treatment of, 142
Bones, affections of, in scurvy, 453, 456
arrest of growth of, in rickets, 493
chemical constitution of, in rickets, 491
condition of the, in chronic hydrocephalus, 837
disease of, a cause of pyæmia, 330
eburnation of articular ends of, in rheumatoid arthritis, 553
extremities of long, enlargement of, in rickets, 478, 481, 488
gouty deposits in, 525
microscopic characteristics of, in rickets, 488
morbid anatomy of, in pyæmia, 334
in rickets, 487
necrosis of, in pyæmia, 334
occasionally involved in erysipelas, 326
pains in, in rickets, 476, 482
in syphilis, 425
suppuration of, a frequent cause of pyæmia, 367
tubercle of, diagnosis of, from rickets, 486
Borax in diphtheria, 79
Bowels, state of the, in mania, 597
in simple meningitis, 809
in tubercular meningitis, 821, 828
Bowels—
see, also, Diarrhœa, Constipation.
Brain, abscess of the, article on, 934
diagnosis, 940
etiology, 934
morbid anatomy, 936
pathology, 938
symptoms, 937
treatment, 941
Brain, adventitious products in the, article on, 883
diagnosis, 886
morbid anatomy, 889
prognosis, 901
symptoms, 883
treatment, 901
Brain, affection of, in gout, 518
cancer of the, 891
chronic softening of, predisposes to cerebral hemorrhage, 908
compression of, by hemorrhage, 920
compression of, by osseous nodes in tertiary syphilis, 433
concussion of, causing abscess, 934
condition of, in hydrophobia, 199
in typhus, 265
in yellow fever, 291
degeneration of, in chronic alcoholism, 684
in insanity, 616
hemorrhage into, in purpura, 461
membranes of, state of, in cerebro-spinal meningitis, 305
morbid anatomy of, in cholera, 412
in pyæmia, 334
in scurvy, 456
softening of, in pyæmia, 334
after typhus, 259
syphilitic deposits in, 435
Brain, congestion of the, article on, 844
causes, 848
diagnosis, 848
morbid anatomy, 851
prognosis, 853
symptoms, 845
treatment, 853
Brain fever, essential, in children, 832, 847, 849
Brain, hypertrophy of, 888, 899
inflammatory softening of, secondary to abscess, 939
malformation of, in idiocy, 603
melanosis of, 891
œdema of, in tubercular meningitis, 833
syphilitic tumors in, 890
tubercular masses in, 889
tumors of, a cause of hemorrhage, 904
wounds of, causing abscess, 934
Brain, softening of the, article on, 856
definition, 856
causes of, 857
diagnosis, 880
Brain, softening of the—
morbid anatomy, 873
pathology, 865
prognosis, 882
symptoms of the acute form, 857
of the chronic form, 864
treatment, 882
Break-bone fever, *see* Dengue.
Breath, coldness of, in cholera, 400
odor of, in pyæmia, 345
peculiar smell of, in alcoholism, 677
Breathing, difficulty of, *see* Dyspnœa.
Bright's disease, predisposes to cerebral hemorrhage, 905
to cerebral softening, 935
Bromide of potassium, as an antaphrodisiac, 766
value of, in treatment of acute mania, 622
of chronic alcoholism, 685
of delirium tremens, 688
of epilepsy, 780
of hysteria, 645
of infantile convulsions, 750
of muscular cramps, 1060
of tumor of the brain, 902
Bromide of sodium, in epilepsy, 781
Bromism, symptoms of, 781
Bronchi, dilatation of, in hooping-cough, 53
implication of, in diphtheria, 67
morbid anatomy of, in diphtheria, 75
in pyæmia, 332
plugging of, in hooping-cough, 51
smallpox eruption in, 131
[Bronchial cough in typhoid fever, 202]
spasm, 1056
Bronchitis, a predisposing cause of measles, 106
a sequela of measles, 111
concealing the commencement of rickets, 476
in cerebro-spinal meningitis, 303
in chronic glanders, 190
in gout, 519
in hooping-cough, 51
treatment of, 56
in influenza, 42
in relapsing fever, 273
in rickets, 482
in scarlet fever, 90
in smallpox, 143
in typhus, 258
treatment of, 268
Broncho-pneumonia, a sequela of measles, 112
in hooping-cough, 50
in relapsing fever, 273
treatment of, 56
Brow-ague, 1029
Bubos, in chronic farcy, 199
in Pali plague, 318
in plague, 314
in relapsing fever, 279
in scarlet fever, 90, 95
in syphilis, 424
morbid anatomy of, in plague, 317

Bubos—
not pathognomonic of plague, 315
Bubo, parotid, a sequela of typhus, 262, 265
diagnosis of, from parotitis, 120
Buffy coat in acute rheumatism, 562
[Bulbo-nuclear paralysis, 1010]
Bullæ, in congenital syphilis, 441
in erysipelas, 324
in glanders, 189
in smallpox eruption, 130
Burial, ante-mortem, 655
Bursæ, effusion into, in gout, 514
gouty deposits in, 516
syphilitic inflammation of, 432
Buxton waters in gout, 547

CÆCUM, perforation of, in typhoid fever, 211
Calcareous deposit in urine in rickets, 477
Calcareous masses in the brain, 896
Calculi, urinary, common in rickety children, 484
diagnosis of, from gouty inflammation of kidney, 520
in gouty diathesis, 520
Calomel, in cerebro-spinal meningitis, 313
in cholera, 415
in choleraic diarrhœa, 420, 421
in dengue, 104
in diphtheria, 82
topical use of, in diphtheria, 81
in gout, 540
in influenza, 45
in intermittent fever, 360
in purpura, 467
in remittent fever, 369
in acute rheumatism, 568
in smallpox, 141
in syphilis, 438
in typhoid fever, 247
in yellow fever, 293
vapor bath for syphilis, 438
Calvaria, affections of, in tertiary syphilis, 433
Camel, traditional origin of smallpox from, 128
Camphor, in cholera, 417
in scarlet fever, 96
in typhus, 268
Cancer, simulation of, by syphilitic nodes, 433
by syphilitic tumors of muscles, 433, 440
Cancer of the brain, 891
of the spinal cord, 1016
Cancrum oris, a rare sequela of scarlet fever, 93
Capillaries, condition of, in cholera, 410, 412
of skin, degeneration of, in purpura, 464, 465, 466
Capillaries in the brain, degeneration in the walls of, a cause of cerebral softening, 871
Capillaries in the brain—
aneurismal dilatation of, 851, 877
obstruction of, a cause of cerebral softening, 870
Capsular ligaments of joints, thickening of, in rheumatoid arthritis, 553
Carbolic acid, glycerine of, in diphtheria, 80
Carbonic acid, in the air of marshes, 353
Carbuncles, in cerebro-spinal meningitis, 304
in the plague, 314, 315
not pathognomonic of plague, 315
Carburetted hydrogen in the air of marshes, 353
Cardiac disease, connection of, with chorea, 699, 705
Cardialgia, treatment of, by arsenic, 1043
Caries of bones in scurvy, 453
Caries of the vertebral column, 1017
Carlsbad waters, in gout, 547
danger of, in rheumatoid arthritis, 557
presence of lithia in, 544
Carotid, internal, hemorrhage from, a rare sequela of scarlet fever, 90
Carpus, deposit in joints of, in gout, 525
Cartilage, excess of water in, in rickets, 491
Cartilage cells, changes of, in rickets, 488
Cartilages, articular, absorption of, in rheumatoid arthritis, 553
deposit of urate of soda into, in gout, 524
destruction of, in gonorrhœal rheumatism, 577
in pyæmia, 335
opacity of, after rheumatism, 564
inter-articular, destruction of, in rheumatoid arthritis, 553
Cascarilla, its use in relieving thirst in parotitis, 121
in measles, 114
Castor-oil, in dysentery, 380
Casts, of gastric tubuli, in vomit in scarlet fever, 94
of tubuli uriniferi, in urine, in cerebro-spinal meningitis, 302
in cholera, 401
in diphtheria, 68
in gout, 523
in intermittent fever, 358
in purpura, 462
in scarlet fever, 91
in yellow fever, 290
Catalepsy, article on, 652
definition, 652
description, 652
causes, 654
treatment, 658
Catalepsy, connection of, with epilepsy, 654
with hysteria, 657
Catarrh, diagnosis of, from diphtheria, 70
earliest symptom of hooping-cough, 49
Catarrh, liability to, after influenza, 41
in varicella, 126
occasional absence of, in measles, 107
Catarrhal fever, diagnosis of, from influenza, 44
Catechu, in chronic dysentery, 383
in typhoid, 248
Causes of disease considered generally, 21
predisposing, 21
exciting, 23
Causes of alcoholism, 671
of catalepsy, 654
of cerebritis, 855
of cholera, 386
of chorea, 709
of congestion of the brain, 848
of dengue, 103
of diphtheria, 61
of dysentery, 373
of epidemic cerebro-spinal meningitis, 308
of epilepsy, 763
of erysipelas, 321
of glanders in the horse, 184
in man, 188
of gonorrhœal rheumatism, 576
of gout, 526, 534
of hooping-cough, 48
of hypochondriasis, 628
of hysteria, 631
of influenza, 34
of insanity, 587
of intermittent fever, 356
of hydrophobia in animals, 195
in man, 197
of locomotor ataxy, 989
of simple meningitis, 814
of tubercular meningitis, 817
of metallic tremor, 801, 807
of miliaria, 123
of mumps, 120
of muscular anæsthesia, 784
of myelitis, 963
of neuralgia, 1027
of paralysis agitans, 725
of purpura, 463
of pyæmia, 338
of remittent fever, 366
of rheumatism, articular, 564
muscular, 574
of rheumatoid arthritis, 554
of rickets, 473
of roseola, 105
of scarlatina, 84
of scarlatinal dropsy, 92
of scurvy, 445
of smallpox, 138, 139
of cerebral softening, 857
of somnambulism, 658
of spinal congestion, 968
of spinal irritation, 997
of spinal meningitis, 955
of sudamina, 122
of sunstroke, 664
of tetanus, 977
of torticollis, 1061
of typhoid fever, 235
of typhus fever, 252
of varicella, 125
of vertigo, 691
of wasting palsy, 786

Causes—
of writer's cramp, 734
of yellow fever, 281
Celibacy, a predisposing cause of insanity, 588
of hysteria, 632
Cellular tissue, gangrene of, in erysipelas, 326
morbid anatomy of, in pyæmia, 335
nodes of, in tertiary syphilis, 432
Cellulitis, diffuse, diagnosis of, from erysipelas, 325
Cephalodynia, 574
Cerebellum, symptoms of hemorrhage into, 915
of tumor in, 887, 990
Cerebral fever (of Trousseau), 808
Cerebral hemorrhage, and apoplexy, article on, 902
definition, 902
morbid anatomy, 902
etiology and pathology, 905
predisposing constitutional state, 905
localization of lesions, 910
the apoplectic condition, 916
diagnosis, general, 922
special, 927
prognosis, 931
treatment, 932
Cerebritis, article on, 854
causes, 855
symptoms, 855
diagnosis, 855
pathology, 855
prognosis and treatment, 856
Cerebro-spinal meningitis, epidemic, article on, 293
bibliography, 314
complications, 303
definition, 293
description of disease, 297
diagnosis, 304
duration, 303
etiology, 303
geographical distribution, 306
history, 306
mode of death, 304
morbid anatomy, 305
nature, 311
prognosis, 305
synonyms, 296
terminations, 304
treatment, 312
Cerebro-spinal meningitis, epidemic, fulminant, 293
purpuric, 293
simple, 297
Cerebrum, *see* Brain.
Cervico-brachial neuralgia, 1032
Cervico-occipital neuralgia, 1032
Cerviodynia, 574
Cesspools, gases from, a cause of cholera, 338
Chalk, in typhoid fever, 247, 248
Chalk-stones, in chronic gout, 516, 538
analysis of, 517
influence of, in prognosis, 538
treatment of, 545
Chancre, hard, description of, 424
Chancre—
soft and hard, relation, 424
soft, the result of syphilitic inoculation on a syphilized person, 427
Change of scene, importance of, in treatment of insanity, 618
Charcoal in sloughing wounds, 349
[Charcot's account of pathology of paralysis agitans, 728]
Chemosis in pyæmia, 334
Chest, paralysis of the muscles of, from hemorrhage into corpus striatum, 912
in wasting palsy, 789
Chicken-pox, *see* Varicella.
Child-bearing, too rapid, a cause of rheumatoid arthritis, 554
Chloral, in delirium tremens, 688
in mania, 621
Chloric ether in parotitis, 123
[Chloride of iron in cerebro-spinal fever, 313]
Chlorides, diminution of, in urine of acute rheumatism, 563
erysipelas, 324
intermittent fever, 353
scarlet fever, 87, 91
typhoid fever, 208
typhus, 269
Chlorinated soda, in diphtheria, 80
Chlorodyne in yellow fever, 294
Chloroform, in treatment of chorea, 715
of infantile convulsions, 669, 751
of delirium tremens, 688
of epilepsy, 780
of sunstroke, 669
of tetanus, 979
Chloroform, inhalation of, for cramps, in cholera, 413
for hiccup in cholera, 410
in influenza, 47
in hooping-cough, 55
in remittent fever, 370
internally, in yellow fever, 294
in typhus, 268
[tolerance of, in hydrophobia, 231]
[Chlorosis, article on, 433]
causation of, 459
history, 463
pathology, 470
prognosis, 470
symptoms, 463
treatment, 470]
Chlorosis, diagnosis of, from scurvy, 454
[Choked disk with brain tumors, 884]
Cholera, epidemic, article on, 396
cryptogamic theories of, 397
definition, 384
departure of epidemics of, 391
diagnosis of, 409
diffusion of, 393
duration of, 408
etiology, 336
Cholera, epidemic—
history, 385
limitation of areas of, 391
mode of invasion of localities by, 391
morbid anatomy, 410
mortality of, 408
mortality to populations from, 392
pathology during life, 412
portability of poison of, 395
prognosis, 415
prophylaxis, 421
symptoms, 398
synonyms, 384
treatment, 415
limitation of areas of, 391
resemblance of, to enteric fever, 231
spasmodic, 407
[Cholera, since 1866, 385
antispasmodic treatment of, 416
contagion of, disputed, 396
pathology of, 411, 414
venesection in, 421]
Cholera fever, 407
Cholera pills, 417
Cholera typhoid, 403
Choleraic diarrhœa, 407
treatment of, 420
Cholerine, 407
[Cholesteræmia a cause of vertigo, 693, 695]
Chondrine, absence of, in rickety bones, 491
Chorea, article on, 696
symptoms, 696
exceptional forms, 704
pathology, 709
causes, 709
prognosis, 711
treatment, 711
Chorea in acute rheumatism, 560
[Chorea from subacute rheumatic meningitis, 767]
Chorea senilis, synonym of paralysis agitans, 718
Choreic mania, 590
Choroid membrane of the eye, tubercle in the, 824
Choroid plexus, abnormal formations in the, 844
Chronic cerebral softening, 864
Chronic hydrocephalus, article on, 836
Chronic meningitis, 816
Chronic pyæmia, 342
Chronic rheumatic arthritis, *see* Rheumatoid arthritis.
Cicatrix after vaccination, 159
in prognosis of smallpox, 157
the sign of efficiency, 168
Cicatrization after cerebral hemorrhage, 904
Cicatrization of ulcers of intestine in typhoid, 215
Cicuta virosa, poisoning by, resemblance to typhoid fever, 244
Cider as a cause of gout, 528
useless as an anti-scorbutic, 459
Ciliary muscles, paralysis of, in diphtheria, 74
Cinchona bark in erysipelas, 328
in chronic gout, 543

Cinchona bark—
in purpura, 468
in acute rheumatism, 569
in rickets, 495
Circulation, cerebral, peculiarities of the, 908
Cirrhosis, of the liver, connection of, with alcoholism, 677
Citric acid, influence of, on scurvy, 456
Civilization, high, a cause of insanity, 587
Clairvoyance, 660
Classification of diseases, 32
Classification of diseases generally, 581
of nervous diseases, 582
of convulsions, 737
of the varieties of insanity, 585, 603
Clavicles, deformity of, in rickets, 479
a frequent seat of nodes in syphilis, 433
Clavus hystericus, 1031
Claw-shaped hand, the, of wasting palsy, 788
Climacteric insanity, 593
Climate, change of, a cause of increased liability to smallpox, 156
a predisposing cause of recurrence in smallpox, 139
essential in chronic dysentery, 382
Climate, in relation to hysteria, 632
to insanity, 587
predisposing to tetanus, 973
influence of, as a cause of cerebro-spinal meningitis, 303
cholera, 388
diphtheria, 62
dysentery, 372
gout, 520
rheumatism, 565
typhus fever, 253
yellow fever, 284
Clitoridectomy, in epileptics, 767, 782
Clots in the brain, changes in, 904
size of, 903
in heart during death from diphtheria, 68, 74, 76
from yellow fever, 291
in small arteries in pyæmia, 336
in veins in pyæmia, 335
disintegration of, 335, 340
suppuration of, 335, 340, 341
Clothes, disinfection of, after cholera, 422
dissemination of cerebro-spinal meningitis by, 309
of cholera by, 394, 395
of scarlet fever by, 84
of smallpox by, 139
of typhus fever by, 254
of yellow fever by, 282
Clothing after acute rheumatism, 572
in muscular rheumatism, 575
in rheumatoid arthritis, 558
in rickets, 495
Coagulation of the blood imperfect in cholera, 406, 410
in malignant smallpox, 131
in purpura, 463
in pyæmia, 335
in typhus, 264
in yellow fever, 288
Cod-liver oil, after measles, 116
in rheumatoid arthritis, 557
in rickets, 496
in smallpox, 143
in treatment of chronic alcoholism, 686
of chorea, 717
of hypochondriasis, 629
of neuralgia, 1042
of spinal irritation, 993
Coffee in scarlet fever, 96
in typhus, 268
supposed prophylactic power of, for gout, 549
Colchicum, in dengue, 104
in acute gout, 539, 540, 541
chronic gout, 542
mode of action of, in gout, 539
in acute rheumatism, 568
[in gouty rheumatism, 568]
in muscular rheumatism, 575
rheumatic pains in influenza, 47
in rheumatoid arthritis, 556
poisoning by, resemblance of, to typhoid fever, 243
Cold, a cause of dysentery, 374, 375
of erysipelas, 322
of gonorrhœal rheumatism, 576
of intermittent fevers, 356
of muscular rheumatism, 575
of rheumatism, 559, 565
of rheumatoid arthritis, 551, 554
of rickets, 473
of scarlatinal dropsy, 92
of scurvy, 450
Cold affusion in influenza, 45
in remittent fever, 369
in scarlet fever, 96
danger of, in erysipelas, 328
influence of, on diphtheria, 62
in remittent fever, 369
[Cold baths in typhoid fever, 250]
Cold exposure to, a cause of congestion of the brain, 848
of facial palsy, 1051
of muscular anæsthesia, 784
of neuralgia, 1032
of sciatica, 1036
of tetanus, 977
of torticollis, 1061
of wasting palsy, 787, 790
Cold, external application of, in epilepsy, 782
in mania, 620
in meningitis, 815
in sunstroke, 669
Cold water, immersion in, for hydrophobia, 200
Collapse, a cause of death in rheumatic pericarditis, 560
in cerebro-spinal meningitis, 298
in malignant scarlet fever, 89
Collapse—
from perforation in typhoid fever, 208
stage of, in cholera, 400
pathology of, 413
treatment of, 417
Collapse, fatal, in mania, 621
stage of, in meningitis, 810
sudden, in delirium tremens, 680
Collodion in erysipelas, 328
Colloid corpuscles in the brain in insanity, 615
Colon, morbid anatomy of, in cholera, 411
Coma, from acute alcoholism 672
from cerebral hemorrhage, 920
from cerebral congestion, 846
from simple meningitis, 810
from tubercular meningitis, 823
from sunstroke, 667
from softening of the brain, 859
from uræmia, 930
Coma, in cerebro-spinal meningitis, 298, 299, 301
in cholera, 400, 401, 402
in erysipelas, 327
in glanders, 189
in hydrophobia, 199
in malignant measles, 108
in the plague, 315
in pyæmia, 344, 347
in relapsing fever, 280
in remittent fever, 368
in scarlet fever, 85, 89, 92
in typhoid fever, 203
in typhus, 259
in yellow fever, 285, 295
uræmic, in cholera, 402
Coma-vigil in typhus, 259
Compound inflammation corpuscles of Gluge, 867, 876
Compression of the brain by hemorrhage, 920
of the spinal cord, 1017
Concretions in the brain, 896
Concussion, of the brain, a cause of abscess, 934
of the spinal cord, 1016
Condylomata, syphilitic, 424, 430, 441
Condy's fluid in diphtheria, 80
Confluent smallpox, 128, 130
Congenital malformations, of the brain, 603
of the meninges, 844
of the spinal cord, 1018
Congenital syphilis, *see* Syphilis.
Congestion of the brain, article on, 844
of the spinal cord, 965
Congestive apoplexy, 848
Conium, in hooping-cough, 54
in influenza, 46
in rickets, 495
application of, to rheumatic joints, 572
Conjunctiva, pustules on, in smallpox, 134
Conjunctivæ, injection of, in dengue, 99
in diphtheria, 65
in influenza, 41
in measles, 107, 110
in typhus fever, 256, 259

Conjunctivæ—
hemorrhage under, in malignant smallpox, 132, 137
hemorrhage under, in purpura, 461
Conjunctivitis after smallpox, 135
treatment of, 144
Consanguinity a cause of disease, *see* Hereditary predisposition.
Consciousness, double, 659
Consequences, of epilepsy, 776
of sunstroke, 670
of convulsions, 744
Constipation, effect on the temperature of fevers, 121
Constipation, in cerebro-spinal meningitis, 298, 301
in dengue, 100
in diphtheritic paralysis, 70
treatment of, 82
in glanders, 189
in gout, 515, 519
in acute gout, 513
in measles, 107
in meningitis, 809
in parotitis, 118
in the plague, 519
in purpura, 462
in acute rheumatism, 559
in rickets, 476
treatment of, 495
in roseola, 105
in scarlet fever, 87
in scurvy, 451, 453
in typhus, 258
in yellow fever, 258
Constitutional syphilis, *see* Syphilis.
Contagion, in cerebro-spinal meningitis, 309
of cholera, 394
in diphtheria, 62
in erysipelas, 322
of glanders among horses, &c., 185
among men, 188
in hooping-cough, 48
in hydrophobia, 195
in influenza, 37
in parotitis, 120
in the plague, 316, 320
in pyæmia, 339, 343
in relapsing fever, 274
[of relapsing fever questioned, 269]
in scarlet fever, 84
in smallpox, 138
in syphilis, 429
through fœtus, 429
imperfect effects of, on syphilis, 428
in typhoid fever, 233, 238
in typhus fever, 253
in varicella, 125
in yellow fever, 281, 295
Convulsionnaires, 647
Convulsions, article on, 737
infantile, 738
symptoms, 740
sequelæ, 744
causes, 745
prognosis, 749
treatment, 749
occurring in adults, 752
unilateral, 752
causes, 754
general or bilateral, 758
Convulsions—
treatment, 761
Convulsions, from abscess of the brain, 938
from congestion of the brain, 846, 849
from cerebritis, 855
from meningeal hemorrhage, 842
from simple meningitis, 809
from tubercular meningitis, 820, 823
from chronic hydrocephalus, 837, 838
from opium poisoning, 929
from softening of the brain, 863
from sunstroke, 667
from tumors of the brain, 746, 885
Convulsions, in diphtheria, 65
in hooping-cough, 51
treatment of, 56
in hydrophobia, 199
in measles, 106, 111
treatment of, 115
in the plague, 316
in pyæmia, 342, 347
in rickets, 483
in scarlet fever, 85, 89, 95
in smallpox, 130, 132
in typhus, almost always uræmic, 259
in yellow fever, 292
[puerperal, 761]
Convulsions, infantile, diagnosis of, from tubercular meningitis, 745
reflex, diagnosis of, from epilepsy, 778
value of, in diagnosis of cerebral hemorrhage, 927
influence of, on prognosis of sunstroke, 668
Co-ordination, muscular, course of the conductors of, in spinal cord, 945
loss of, in alcoholism, 676, 678
in locomotor ataxy, 984
in general paralysis of the insane, 606
in writer's cramp, 735
from section of the posterior columns of the cord, 942
from section of the lateral columns, 943
Copper, sulphate of, 315
in dysentery, 383
in hooping-cough, 55
in typhoid fever, 248
salts of, local use of, in diphtheria, 80
Cord round the body, sensation of, in myelitis, 959
Cornea, interstitial inflammation of, in hereditary syphilis, 429, 430, 441, 442
opacity of, in syphilitic keratitis, 442
ulceration of, in cerebro-spinal meningitis, 293, 300
in cholera, 403
in scarlet fever, 93
treatment of, 98
in smallpox, 133, 134
treatment of, 144
Corpora amylacea in the brain, 844
Corpus striatum, the, a frequent seat of cerebral hemorrhage, 903
of cerebral softening, 972
arrangement of arteries in, 909
symptoms of hemorrhage into, 911
Corymbose smallpox, 128, 131
Coryza, in diphtheria, 65
an early symptom of hooping-cough, 49
in measles, 107, 110
occasional absence of, 110
in scarlet fever, 85, 87, 89, 95
treatment of, 96
as a sequel of scarlet fever, 90
Cough, in diphtheria, 67
in glanders, 189, 190
in hooping-cough, 49, 51, 52
in influenza, 42
in laryngitis complicating measles, 111
in measles, 107, 111
in pyæmia, 345
in typhoid fever, 204
in typhus, 258
in rheumatoid arthritis, 552
in smallpox, 131
Cough, spasmodic, a symptom of spinal irritation, 994
Counter-irritation in epilepsy, 781
in insanity, 620
in neuralgia, 1046
in spinal irritation, 998
[in tetanus, 980]
Coup de soleil, 661
Course of disease considered generally, 25
Cowpox, *see* Vaccination.
Cowpox, accelerated, 160
retarded, 160
spurious, 161
not prophylactic of chickenpox, 124
Cramp, writer's, 732
Cramps, in cholera, 399
treatment of, 416, 418
in yellow fever, 235
in choleraic diarrhœa, 407
in the legs in gout, 513, 515, 518
Cramps, muscular, in the extremities, causes of, 1059
treatment of, 1060
in epilepsy, 774
in paralysis agitans, 721
Cranial bones, softness of, in rickets, 477
Cranial nerves, paralysis of, from syphilitic neuromata, 435, 440
Creasote, in yellow fever, 294
inhalations for chronic laryngitis after measles, 116
Crescentic form of syphilitic ulceration, 432
Crescents in rash of measles, 108
of smallpox, 129
Cretinism, 604, 605
Crick in the neck, 575
Crimea, scurvy in the, 447
Cross-paralysis, 913, 914
Croton oil, externally, in rheumatoid arthritis, 557

Croup, association of, with typhoid fever, 224
formerly confounded with diphtheria, 60, 61
Crowding in houses, a cause of cerebro-spinal meningitis, 312
of the plague, 318
of typhus fever, 252
Crural neuralgia, 1035
Crus cerebri, symptoms of hemorrhage into, 913
Cruveilhier's atrophy, 786
Cry, the peculiar, of epilepsy, 771
of meningitis, 809, 822
Cryptogamic theories of origin of cholera, 397
Cubic space required in smallpox, 140
Cultivation, effect of, on malaria, 353
[Curara, used in hydrophobia, 200]
Curvature of the spine in rickets, 478, 491
Cutaneous anæsthesia, 1065
Cutis anserina, occasionally present in scarlet fever, 85
in typhoid fever, 218
Cutis, inflammation of, in erysipelas, 326
Cysticerci in the brain, 896
in the spinal cord, 1016
Cystic neuroma, 1032
Cystitis, chronic, in gout, 520
treatment of, 547
Cysts in the brain, 897

DAMP as a cause of disease, *see* Moisture.
Dance of St. Guy, 696
Dancing mania, 647, 701
Dandy fever, *see* Dengue.
Deafness, complicating facial neuralgia, 1037
in cerebro-spinal meningitis, 298, 300
during erysipelas, 324
in hereditary syphilis, 429
in locomotor ataxy, 985
in measles, 107, 111
after scarlet fever, 90
in scurvy, 453
in typhus fever, 255, 259
from syphilitic disease of auditory nerve, 435
in vertigo, 690
Death, mode of, in cerebro-spinal meningitis, 304
in cholera, 401, 402, 403, 404
in dengue, 100
in diphtheria, 67, 68, 77
in dysentery, 376, 377
in erysipelas, 324
in glanders, 190
in gout, 537, 538
in hooping-cough, 50
in hydrophobia, 199
in influenza, 43
in intermittent fever, 359
in measles, 108
in plague, 315
in purpura, 466
in pyæmia, 344
in relapsing fever, 278, 279, 280
in remittent fever, 366
Death—
in rheumatism, 537
in rickets, 482
in scarlet fever, 89, 90
in smallpox, 130, 132
in scurvy, 452, 453
in typhoid, 201, 246
in typhus, 246
in yellow fever, 285
Death, apparent, in catalapsy, 655
rapid, from cerebral hemorrhage, 925
causes of, in convulsions, 750
in delirium tremens, 680
in dementia, 601
in the insane, 618
in acute mania, 597
in melancholia, 595
Debility, general, predisposes to neuralgia, 1027
Debility of parents a cause of rickets, 473
Decomposition, rapidity of, in scurvy, 456
Definition of disease, 17
Deformities, due to essential paralysis of children, 1006
to paralysis agitans, 721
to wasting palsy, 788
from gout, 516
in chronic rheumatoid arthritis, 559
Degeneration, albumenoid, *see* Amyloid degeneration.
Deglutition, difficulty of, in chorea, 693
in spinal meningitis, 954
in tetanus, 971
Delirium, in cerebro-spinal meningitis, 297, 298, 299, 301
in cholera, uræmic, 402
typhoid, 403
in diphtheria, 66, 67
in dysentery, acute, 376
in erysipelas, 323, 324, 326
in glanders, 189
in gout, 518
in hydrophobia, 199
in influenza, 41, 42
in measles, 107, 108
in parotitis, 119
in the plague, 315
in pyæmia, 344, 347
in relapsing fever, 277
in remittent fever, 367, 368
in rheumatic pericarditis, 530
in scarlet fever, 85, 88, 89, 95
in smallpox, 130, 137
treatment of, 141
in typhoid fever, 203, 204, 205, 221
in typhus, 259
treatment of, 267
in yellow fever, 285, 292, 294
Delirium, character of the, in congestion of the brain, 847
in alcoholism, 679
in softening of the brain, 864
in meningitis, 809
Delirium tremens, 670
symptoms, 678
diagnosis, 681
prognosis, 682
treatment, 686
diagnosis of, from acute mania, 697
Delirium tremens, diagnosis—
from meningitis, 815
Delirium tremens, at invasion of smallpox, 130
treatment of, 141
diagnosis of, from typhus, 263
resemblance of, to delirium in cerebro-spinal meningitis, 297
[Delirium tremens in surgical cases, 679
inflammatory cases of, 680
warm bath in treatment of, 639]
Delusion, definition of, 593
character of, in acute mania, 596
in melancholia, 592, 594
in monomania, 599
hints for the detection of, 607
Dementia, 600
senile, 601
diagnosis of, 594, 609
prognosis, 617
Dengue, article on, 93
definition, 98
diagnosis, 103
etiology, 103
history, 98
propagation, 103
symptoms, 79
synonyms, 98
treatment, 103
Dentition, predisposes to infantile convulsions, 746, 749
a cause of true epilepsy, 765, 766
a cause of roseola, 105
delay of, in rickets, 480
Desiccation, stage of, in smallpox, 129
Desquamation of cuticle, in dengue, 101
in gout, 513, 514, 536
after erythema, in stage of reaction in cholera, 695
in measles, 109
in parotitis, 119
over rheumatic joints, rare, 531
in scarlet fever, 88
after sudamina, 122
in typhoid fever, 218
in yellow fever, 285
Development, arrest of, in hereditary syphilis, 442
Diabetes mellitus, causing rheumatoid arthritis, 554
occurring in gouty subjects, 520
Diagnosis of disease, considered generally, 26
Diaphoretics, in chronic gout, 545
in gout, 540
in gonorrhœal rheumatism, 579
in muscular rheumatism, 575
in pyæmia, 350
in typhoid fever, 247
Diaphragm, affection of, in scurvy, 453
muscular rheumatism in, 574
spasm of, in tetanus, 977
Diarrhœa, in cerebro-spinal meningitis, 298, 301
in reactionary stage of cholera, treatment of, 418

Diarrhœa—
in stage of reaction of cholera, 402, 403
in diphtheria, 65, 68
in erysipelas, 322
in glanders, 189, 190
in gout, 515, 519
in influenza, 41, 43
tendency to, after intermittent fever, 358
in measles, 107, 111
in parotitis, 119
in the plague, 315
in purpura, 462
in relapsing fever, 277, 279
in remittent fever, 367, 368
in rickets, 476, 482, 483
treatment of, 496
in scarlet fever, 87, 89, 90
in scurvy, 453
treatment of, 458
in smallpox, 133
treatment of, 142
in pyæmia, 344, 346
treatment of, 351
in typhoid fever, 202
treatment of, 248
in typhus, 257
treatment of, 268
Diarrhœa, choleraic, 407
treatment of, 420
chronic, after cholera, 415
diagnosis of, from cholera, 409
as a predisposing cause of cholera, 390
a premonitory symptom of cholera, 399
prevalence of, before and during cholera epidemics, 391
Diarrhœa, hill, 396
Diarrhœa, a cause of infantile convulsions, 747, 753
see also Bowels, state of.
Dicrotism of the pulse, in delirium tremens, 679
in epilepsy, 772
in typhus fever, 256
Diet, the necessary, in chronic alcoholism, 685
errors of, a cause of erysipelas, 322
of roseola, 105
in cerebro-spinal meningitis, 313
in cholera, 418, 419
in choleraic diarrhœa, 420
in diphtheria, 78, 81
in dysentery, 382, 383
in epilepsy, 781
in gonorrhœal rheumatism, 579
in gout, 548
in hooping-cough, 54
in influenza, 45
in intermittent fever, 363
in measles, 114
in meningitis, 816
in parotitis, 121
in the plague, 317
in purpura, 468
in pyæmia, 352
in relapsing fever, 280
in remittent fever, 370
in acute rheumatism, 573
in muscular rheumatism, 575
in rheumatoid arthritis, 553
in rickets, 495, 496
Diet—
in scarlet fever, 95
in scurvy, 458
in smallpox, 141, 142
in syphilis, 438
in typhoid, 249
in typhus, 266, 268
Diffusion of cholera, 393
Digitalis, in acute mania, 621
in delirium tremens, 687
in hemorrhage in typhoid fever, 249
in purpura, 468
in rheumatic pericarditis, 572
[in scarlet fever, 97]
in uræmia after cholera, 419
"Digitorum nodii" of Heberden, 552
Dilatation of the heart as a sequela of scarlet fever, 93
Diphtheria, article on, 57
definition, 57
diagnosis, 70
etiology, 61
history, 58
morbid anatomy, 74
name, 58
pathology, 72
prognosis, 77
symptoms, 65
synonyms, 57
therapeutics, 78
formerly confounded with croup, 60
with erysipelas, 58
with scarlet fever, 86, 58, 61
mention of, by the ancients, 58
relation of, to typhoid fever, 223
a sequela of scarlet fever, 93
Diphtheria, nasal, an occasional complication of scarlet fever, 93
Diphtherite, 58
Diphtheritis of labia in measles, 113
Dirt, a predisposing cause of pyæmia, 344
of typhus fever, 252
Discrete smallpox, 128, 129
Diseases, classification of, 32
causes of, 21
course of, 25
definition of, 17
diagnosis of, 26
duration of, 25
functional, 19
history of, 21
names of, 17
signs of, 23
structural, 19
symptoms of, 23
terminations of, 26
Dislocation of joints in rheumatoid arthritis, 551, 553
[Disseminated sclerosis, 1011]
Diuresis, after scarlet fever, 91
Diuretics, in relapsing fever, 280
in kidney complications in typhus, 268
in muscular rheumatism, 575
in scarlatinal dropsy, 97
[in scarlet fever, 96]
Division of nerves for cure of neuralgia, 1047
of neuroma, 1025
Dog, hydrophobia in, 196, 197
Dorsodynia, 574
Dorso-intercostal neuralgia, 1034
Dorso-lumbar neuralgia, 1035
Double consciousness, 659
Double facial palsy, 1053
Douche, cold, in catalepsy, 658
in hysteria, 645
in insanity, 620
in sunstroke, 669
Drains, emanations from, a cause of typhoid fever, 241
Dress, relation of, to sunstroke, 667
Drinking, tendency to, in hysteria, 639
Dropsy, scarlatinal, 91, 92, 97
Drought, a cause of typhoid fever. 238
Drunkards, morning sickness of, 677
Drunkenness, physiology of, 671
diagnosis of, from apoplexy, 928
Duality of syphilitic poison, question of, 423
Duodenum, morbid anatomy of, in yellow fever, 292
Dura mater, the hæmatoma of, 843
inflammation of, 813
syphilic nodes in, 812
tumors of, 843
Duration of disease, considered generally, 25
Duration, of cerebro-spinal meningitis, 298, 299, 303
of cholera, 408
of chorea, 711
of dengue, 102
of diphtheria, 66, 70
of dysentery, mild acute, 373, 375
sthenic, 373
typhoid or malignant, 373
scorbutic, 373
of erysipelas, 324
of acute farcy, 190
of chronic farcy, 190
of glanders, acute, 189
chronic, 190
of acute gout, 513
of hooping-cough, 50
of hydrophobia in dogs, 196
of hydrophobia in man, 199
of intermittent fever, 356
of paroxysm of, 355
of stages of paroxysm of, 357
of influenza, 41
of locomotor ataxy, 989
of measles, 107, 109
of simple meningitis, 813
of spinal meningitis, 955
of tubercular meningitis, 825
of parotitis, 119
of the plague, 315
of pyæmia, 330, 345, 347
of relapsing fever, primary fever, 277
of the relapse, 277
of remittent fever, 365
of acute rheumatism, 559, 567
of articular rheumatism, subacute, 560
of muscular rheumatism, 574
of rickets, 494

Duration—
of scarlet fever, 88
of smallpox, 129
of spinal congestion, 968
of syphilis, 426
of syphilis, secondary symptoms, 425
of tetanus, 979
of typhoid, 201
of typhus, 261
of vaccinia, 159, 160
of varicella, 125, 126
of wasting palsy, 789
of yellow fever, 285
Dysæsthesia, in hysteria, 635
Dysentery, article on, 372
causes, 373
course, malarial, 373
mild acute, 373
sthenic acute, 373
typhoid or malignant, 373
definition, 372
diagnosis, 378
history, 372
modes of commencement, 373
morbid anatomy of acute, 378
of chronic, 379
pathology, 378
prognosis, 380
terminology, 372
symptoms of acute, 376
chronic, 376
malarial, 377
malignant, 377
mild, 375
scorbutic, 377
treatment of acute, 380
chronic, 382
malarial, 382
mild, 380
scorbutic, 382
[Dysentery, astringent enemata for, 382, 383]
Dysentery, complicating scurvy, 453
treatment of, 458
connection of, with typhus, 258
relation of, to typhoid fever, 230
resemblance of syphilitic ulceration of rectum to, 432
Dyspepsia, acute, resemblance to invasion of typhus, 254
as a cause of gout, 520, 534
a cause of vertigo, 691
diagnosis of, from tubercular meningitis, 830
in chronic gout, 518
treatment of, 545
as a premonitory symptom of gout, 515
in rheumatoid arthritis, 551
Dyspepsia, secondary to alcoholism, 677
to hypochondriasis, 625
to hysteria, 639
Dysphagia, in diphtheria, 66, 67
from diphtheritic paralysis, 69
in chronic gout, 519
in parotitis, 119
in scarlet fever, 86, 90
Dyspnœa, in cholera, 400
causes of, 414
in diphtheria, 67
treatment of, 82
Dyspnœa—
in erysipelas, 327
in chronic glanders, 190
in retrocedent gout, 483
in hooping-cough, 50
in hysteria, 636
in influenza, 41, 42
in mercurial poisoning, 803
in myelitis, 961
in parotitis, 119
in scurvy, 451, 452, 453
in spinal congestion, 967
in spinal meningitis, 954
in tetanus, 972
in typhoid fever, treatment of, 249

EAR, disease of the, a cause of convulsions in children, 746
of abscess of the brain, 935, 937
of meningitis, 813
of vertigo, 694
Ears, commencement of erysipelatous inflammation in, 323
hemorrhage from, in malignant smallpox, 132
hemorrhage from, in purpura, 461
hemorrhage from, in yellow fever, 289
inflammation of, in measles, 112
helix of, chalk-stones in, 516, 517, 520, 533, 535
internal inflammation of, in rheumatoid arthritis, 552
lobes of, pain in, in dengue, 99
Eccentric convulsions, 766
Eccentricity, distinction of, from insanity, 609
Ecchymoses, in cerebro-spinal meningitis, 299, 302
in diphtheria, 76
in scurvy, 451, 456
in purpura, 460, 463
in typhus, 257
in yellow fever, 289
of heart in scurvy, 459
subconjunctival in cholera, 410
submucous in cholera, 410, 411
subserous in cholera, 410
subserous in purpura, 463
traumatic, diagnosis of, from purpura, 467
Echinococci in the brain, 896
Eclampsia, 745, 748
Ecstasy, article on, 646
varieties and symptoms, 646
treatment, 648
Eczema, in gouty subjects, 520
of the lip in measles, 110
after vaccination, merely a coincidence, 177
Education, injudicious, a cause of insanity, 589
of hysteria, 643
[Effervescent drinks in yellow fever, 295]
Effusions, nature of, in scurvy, 457
Elbow, deformity of, in rheumatoid arthritis, 551
Elbow—
inflammation of skin over, in smallpox, 133
Electric chorea, 700
Electric irritability and sensibility of muscles, in general paralysis of the insane, 606, 699
in general spinal paralysis, 999
in hysteria, 638
in essential paralysis, 1005, 1006
in locomotor ataxy, 986
in muscular anæsthesia, 783
in myelitis, 961
in softening of the brain, 863
in spinal congestion, 967
in torticollis, 1062
in wasting palsy, 789
Electrical conditions, relation of, to influenza, 36
Electricity, in muscular rheumatism, 575
influence of, on spread of cholera, 387
Electricity, value of, in treatment of chorea, 717
of hysteria, 645
of infantile paralysis, 1007
of locomotor ataxy, 990
of mercurial poisoning, 806
of muscular anæsthesia, 786
of myelitis, 965
of neuralgia, 1046
of paralysis agitans, 730
of torticollis, 1063
of wasting palsy, 798
of writer's cramp, 737
Elevation above sea-level, influence of, on spread of cholera, 388
Elimination of alcohol from the system (with note), 672, 673
Emaciation, in scurvy, 456
in congenital syphilis, 441
in rickets, 476, 482, 494
Embolism, of cerebral arteries, in chorea, 704
a cause of convulsions, 757, 760
of softening of the brain, 868
Embolism, in arteries of brain, in erysipelas, 326
in pyæmia, 337, 340
in scurvy, 455
Emetics, in dengue, 103
in diphtheria, 82
in hooping-cough, 56
in influenza, 45
in intermittent fever, 360
in measles, 115
in parotitis, 120
in purpura, 468
in relapsing fever, 280
in remittent fever, 369
in acute rheumatism, 570
Emotion, sudden, a cause of catalepsy, 654
of epilepsy, 765
of hypochondriasis, 628
of insanity, 589
of paralysis agitans, 725
of somnambulism, 659
Emphysema of lungs, in diphtheria, 76
in hooping-cough, 51
in influenza, 42

Emphysema as a sequel of scarlet fever, 93
Emprosthotonos, in tetanus, 971
Empyema, chronic, causing abscess of the brain, 935
Ems waters in gout, 547
Endemic of syphilis at Rivalta, 180
Endocarditis in acute rheumatism, 560, 563
treatment of, 571
Endocardium, changes of, in pyæmia, 332
Enemata, in hemorrhage of typhoid fever, 249
in hydrophobia, 201
in yellow fever, 293
Enteric fever, *see* Typhoid fever.
Enteritis, liable to become typhoid, 232
Entozoa in the brain, 896
in the spinal cord, 1016
in the intestines, a cause of asthma, 1057
of catalepsy, 655
of spasms, 1056
Ephemeral fever, diagnosis of, from yellow fever, 286
Epidemics, of cholera, 384
departure of, 391
health of communities before and during, 391
mode of invasion of, 391
relation of, to barometric pressure, 387
climate, 388
electricity, 387
moisture, 387
ozone, 387
rain, 387
temperature, 386
of dengue, 98
of diphtheria, 58
mode of propagation of, 62
mortality of, 61
relation of, to climate, 62
season, 62
temperature, 62
of dysentery, 372
of epidemic cerebro-spinal meningitis, 306
of erysipelas, 321
of influenza, 34
course of, 34
distribution of, 34
duration of, 35
influence of atmospheric and other conditions on, 37
occasional intermittent character of, 34
phenomena of, 36
rate of progress of, 34
relation of, to other epidemics, 38
of measles frequently preceding or preceded by epidemics of hooping-cough, 106
of parotitis, 120
of plague, 315, 318
etiology of, 318
phenomena of, 320
of rabies among animals, 193
of relapsing fever, 269
distribution of, 273
mortality of, 279
Epidemics—
propagation of, by contagion, 274
relation of, to destitution and famine, 276
of smallpox, 156
of smallpox in Bengal, 393
of typhus fever, 252
of varicella, 125
of yellow fever, 281
Epididymitis, in parotitis, 119
Epigastrium, counter-irritation to, for vomiting in cholera, 416
heat at, in cholera, 400
oppression at the, in remittent fever, 365, 366
Epiglottis, exudation on, in diphtheria, 67, 76
swelling of, in scarlet fever, 90
ulceration of, in diphtheria, 76
ulceration of, in chronic glanders, 191
Epilepsia mitior, 768
gravior, 769
abortiva, 772
Epilepsy, article on, 762
definition, 762
synonyms, 763
natural history, 763
causes, 763
symptoms, 767
relation between the symptoms, 775
complications, 776
pathology, 777
diagnosis, 778
prognosis, 779
treatment, 780
Epilepsy, caused by gouty diathesis, 519
intermittent fever, 363
cured by intermittent fever, 363
Epilepsy, diagnosis of, in children, 748
connection with catalepsy, 655
diagnosis of, from hysteria, 642
followed by mania, 590
a sequela of sunstroke, 670
Epileptic aphasia, 753
aura, 752, 770
hemiplegia, 753
mania, 776
Epileptiform attacks, from chronic alcoholism, 678
influence of, on prognosis of mania, 617
see also Convulsions.
Epistaxis, in dengue, 100
in diphtheria, 66
in erysipelas, 322
in hooping-cough, 50
in influenza, 41
in measles, 107, 112
treatment of, 115
in purpura, 461
treatment of, 468
in relapsing fever, 277
in scarlatina, 93
in typhoid fever, 202
in variola maligna, 131
in yellow fever, 235, 239
Epistaxis, premonitory of cerebral hemorrhage, 924, 926
Epithelial scales, dissemination of scarlet fever by, 84
Epithelium of bladder in urine after scarlatina, 91
Equinia, *see* Glanders.
Erectile tumors in the brain, 893
Ergot of rye in cerebro-spinal meningitis, 313
in puerperal pyæmia, 350
Erratic erysipelas, 327
Eruptions, *see* Rash.
Erysipelas, article on, 321
causes, 321
definition, 321
diagnosis, 324
morbid anatomy, 325
natural history, 321
pathology, 325
prognosis, 326
symptoms, 322
synonyms, 321
treatment, 327
[treatment of, without alcohol, 328
local applications for, 329]
varieties, 329
Erysipelas, a cause of pyæmia, 330
after vaccination, 161
diagnosis of, from diphtheria, 72
in glanders, 189
in smallpox, 133, 134
treatment of, 143
in smallpox, affecting the pulmonary mucous membrane, 147
in typhoid fever, 225
in typhus fever, 262
treatment of, 268
relation of, to diphtheria, 72
Erysipelas, complicating facial neuralgia, 1038
Erythema, diagnosis of erysipelas from, 325
in cerebro spinal meningitis, 302, 303
in congenital syphilis, 441
in stage of reaction in cholera, 402
in vaccination, 161
Essential convulsions, 745
Essential paralysis of children, 1004
Essential vertigo, 693
treatment of, 695
Etiology, *see* Causes.
Eustachian tube, disease of, a sequel of scarlet fever, 90
in measles, 111
Evacuation stage of cholera, 399
treatment, 415
Evacuations, *see* Stools.
Examination of the insane, hints as to the, 607
Exanthema hæmorrhagicum of Dr. Graves, 462
Excision of neuroma, 1025
of piece of a nerve for cure of neuralgia, 1047
Exciting cause of disease, meaning of the term, 23
Exercise, importance of, in treatment of epilepsy, 782
Exhaustion, a cause of sunstroke, 663, 664
diagnosis of, from tubercular meningitis, 830

Exhaustion in hooping-cough, 52
treatment of, 57
[Expectancy in management of typhoid fever, 250]
Expectant treatment in acute rheumatism, 537
Extravasation of blood, in purpura, 461, 463
in pyæmia, 331
in lungs, 332
subpleural, 332
in heart, 332
in spleen, 333
under mucous membranes, 334
subperitoneal, 334
in brain, 334
in cellular tissue, 334
in scurvy, 451, 456
Exudation, in cholera on mucous membrane of intestines, 411, 412
in croup, how different from diphtheria, 75
in diphtheria, 65, 72, 73
chemical characteristics of, 74
how far contagious, 63
microscopic characters of, 74
on mucous membranes and skin, 68
on wounds, 68
pathology of, 72
treatment of, 78
in dysentery, on mucous membrane of colon, 379
in influenza, 44
inflammatory in pyæmia, 331
in intestines, 334
in cellular tissue and muscles, 334
in digestive tract, 333
in heart, 332
in liver, 333
in lungs, 331
in pericardium, 333
in peritoneum, 334
in pleuræ, 332
in typhoid fever, 212
in typhoid fever on fauces, 224
Eye, gouty deposits in, 529
hemorrhage from, in yellow fever, 289
implication of, in rheumatoid arthritis, 552
intense pain in, in dengue, 99
morbid anatomy of, in pyæmia, 334
sloughing of, in pyæmia, 334
state of, in scurvy, 451
Eye, syphilitic disease of, *see* Iritis, Retinitis, and Keratitis.
Eyeball, suppuration of, cerebro-spinal meningitis, 299, 300
Eyeballs, conjugate deviation of, from hemorrhage into the cerebellum, 915
lateral deviation of, from hemorrhage into the corpus striatum, 912
in acute softening of the brain, 860
Eyelids, a frequent seat of erysipelatous inflammation, 323
swelling of, in smallpox, 129

FACE, anæsthesia of, 1065
histrionic spasm of, 1059
neuralgia of, 1036
paralysis of, from hemorrhage into the corpus striatum, 913
from hemorrhage into the pons, 914, 915
from tubercular meningitis, 823
from wasting palsy, 788, 789
premonitory of cerebral hemorrhage, 924
swelling of, in smallpox, 129, 133
Face, arrest of growth of bones of, in rickets, 480
Face, expression of, in cerebro-spinal meningitis, 298
in cholera, 399, 400
in chronic alcoholism, 677
in chronic hydrocephalus, 838
in chronic mania, 608
in dementia, 600, 601
during incubation of cholera, 399, 409
in dysentery, 376, 377
in epilepsy, 769, 771
in general spinal paralysis, 1000
in hysteria, 1000
in measles, 107
in melancholia, 594
in simple meningitis, 809, 810
in tubercular meningitis, 821
in the plague, 314
in mercurial poisoning, 803, 804
in pyæmia, 344, 345
in remittent fever, 366
in rickets, 477, 484
in scurvy, 451
in hereditary syphilis, 441
in typhoid fever, 203
in typhus, 255
in wasting palsy, 789
in yellow fever, 284, 287, 292, 293
Facial nerve, the, paralysis of, at different parts of its course, 914
Facial palsy, Bell's, symptoms of, 1050
causes, 1051
prognosis, 1052
treatment, 1053
double, 1053
Facies hysterica (of Todd), 1000
Famine, *see* Food, insufficient.
Famine fever, *see* Relapsing fever.
Faradization, in treatment of hysterical paralysis, 1071
of muscular anæsthesia, 736
of myelitis, 965
of wasting palsy, 798
see also Electricity.
Farcy buds, 184
Farcy in horses, &c., acute, 184
chronic, 184
in man, acute, 190
chronic, 190
Fat, deficiency of, in diet, a cause of neuralgia, 1042, 1047
Father, health of, in production of rickets, 473
Fatigue, a predisposing cause of cerebro-spinal meningitis, 309
of cholera, 389, 390
of diphtheria, 64
of neuralgia, 1027
of purpura, 463
Fatty degeneration of muscles, in infantile paralysis, 1006
in wasting palsy, 848
Fauces, condition of, in diphtheria, 65, 66
in erysipelas, 327, 329
in hydrophobia, 109
in scarlet fever, 90
in smallpox, 131
Fear, a cause of death in the plague, 315
a predisposing cause of cholera, 391
Febrile form of congestion of the brain, 847
Feces, *see* Stools.
Feigned catalepsy, 653
epilepsy, 778
insanity, 607
Femur, deformity of, in rickets, 478
Fibrin, amount of, in blood of acute rheumatism, 562
in blood of purpura, 465
in blood of typhus fever, 264
in blood of yellow fever, 289
presence of, in diphtheritic exudation, 74
Fibro-plastic tumors in the brain, 892
Fibrous tumors in the brain, 892
Fifth cranial nerve, anæsthesia of the, 1065
neuralgia of, 1030
paralysis of, 1055
[Filth promotive of diphtheria, 64]
Fingers, deformity of, in rheumatoid arthritis, 551
Fistula lacrymalis after scarlet fever, 90
Fits, apoplectic, 846, 858, 924
cataleptic, 652
in children, 740
epileptic, 770
hysterical, 639
Flatulence, in gout, 518
treatment of, 545
a premonitory symptom of gout, 515
in hypochondriasis, 625, 630
in hysteria, 639
Flea-bites, diagnosis of roseola from, 106
rash in measles from, 113
diagnosis of, from purpura, 467
[Fluorescence of blood, affected by quinine, 362]
Fœtus, communication of syphilis by, 429
rickets in, 475
smallpox in, 488
Folie circulaire, 597
Follicles of skin, how far implicated in variolous eruption, 148
of small intestines, condition of, in typhoid fever, 209
Fontanelle, late closure of, in rickets, 476, 480

Food, animal, want of, a predisposing cause of scurvy, 450
Food, excess of, a cause of gout, 520, 534
insufficient, a predisposing cause of cerebro-spinal meningitis, 309
of the plague, 318
of purpura, 463
of pyæmia, 343
of relapsing fever, 296
of rheumatism, 565
of scurvy, 450
of typhus fever, 252
nature of, influence of, on gout, 529
unwholesome, a cause of cholera, 389
of dysentery, 374
of the plague, 318
of pyæmia, 348
of rickets, 473
of scurvy, 447
of typhoid fever, 243
Food, improper, a cause of infantile convulsions, 743, 747
see also Diet.
Forehead, shape of, in rickets, 480
Foreign bodies, in a wound, a cause of neuralgia, 1038
Fracture of the skull, a cause of abscess of the brain, 934
diagnosis of, from apoplexy, 929
Friction-sound in pericarditis of rheumatism, 560
in pyæmia, pleural and pericardial, 345
Friction, to joints, in rheumatoid arthritis, 558
to skin in gonorrhœal rheumatism, 579
in chronic gout, 545
in muscular rheumatism, 575
Fright, a cause of catalepsy, 654
of chorea, 709
of convulsions, 757, 758
of epilepsy, 765
of paralysis agitans, 725
of tetanus, 978
Fright of a pregnant woman, a cause of idiocy in the child, 604
Frontal sinuses, expansion of, in rickets, 480
Function and structure, general relations of, 19
Functional disease as contrasted with structural, 19
Fungi in atmosphere, as a cause of influenza, 37, 39
theory of, for the propagation of cholera, 396
Furor transitorius, 597

GAIT, the peculiar, of general paralysis of the insane, 605
of common hemiplegia, 1001
of hysterical hemiplegia, 1001
of locomotor ataxy, 984
of paraplegia, 963
in disease of the cerebellum, 990
Galbanum plaster in rheumatoid arthritis, 557
Gall-bladder, exudation in, in diphtheria, 75
Gallic acid, in cholera, 416, 419
in choleraic diarrhœa, 420
in chronic dysentery, 383
in purpura, 468
in scarlet fever, 97
in typhoid fever, 249
glycerine of, in diphtheria, 80
Galvanism, value of, in treatment, *see* Electricity.
Ganglia, sympathetic, supposed inflammation of, in yellow fever, 291
Gangrene, in cerebro-spinal meningitis, 303
in cholera, 404, 414
in glanders, 189
in measles, treatment of, 115
in the plague, 314
in pyæmia, 331
in smallpox, 133
treatment of, 143
in typhus, 257
Gangrene, a sequela of scarlet fever, 93
of genitals in cholera, 404
in erysipelas, 327
in smallpox, 134, 143
of the lung in measles, 113
of the lungs, tendency to, in melancholia, 595
in scurvy, 453
in pyæmia, 331
a sequel of typhus, 262
of mouth, in measles, 112
of mucous membrane of colon in cholera, 411
in dysentery, 379
of skin in erysipelas, 326
of vulva in measles, 112
Gastein waters in gout, 547
Gastro-enteritis, diagnosis of, from tubercular meningitis, 830
Gelatine, absence of, in rickety bones, 491
General diseases, determined by agents operating from without, list of, 33
by conditions existing within the body, list of, 415
General paralysis of the insane, symptoms of, 605, 999
diagnosis, 609
General spinal paralysis, 999
Genitals, gangrene of, in cholera, 404
in erysipelas, 326
in smallpox, 134, 143
[German measles, 117]
Germs, cholera, in stools, theory of, for the propagation of cholera, 395
objections to, 396
Glanders, article on, 182
definition, 182
history, 182
in horse, &c., 183
acute, 183
chronic, 183
in man, 185
diagnosis of, 191
etiology, 188
morbid anatomy, 191
Glanders—
prognosis, 192
symptoms, 189
therapeutics, 192
Glands, *see* under their special names.
Gliomata in the brain, 892
a cause of cerebral hemorrhage, 904
Globus hystericus, 636
Glossitis in smallpox, 133
[Glosso-labio-laryngeal paralysis, 1010]
Glottis, œdema of, in erysipelas, 327
in typhoid fever, 225
after typhus, 262
paralysis of, in diphtheria, 69
Gluten obtained from cranial bones in rickets, 491
Glycerine, topical application of, in diphtheria, 79
Goitre, relation of, to idiocy, 605
Gonorrhœa, a cause of gangrene of genitals in smallpox, 134
Gonorrhœal rheumatism, article on, 573
definition, 576
history, 576
symptoms, 576
treatment, 579
Gonorrhœal rheumatism, diagnosis of rheumatism from, 567
Gout, article on, 512
after effects of, 515
causes of, 526
classification, 512
condition of blood in, 520
definition of, 512
description of an acute attack of, 512
diagnosis of, 536
diseases occurring with, 523
history of, 512
irregular, 518
morbid anatomy, 523
pathology of, 531
phenomena during an acute attack of, 513
phenomena of chronic, 516
prognosis, 537
retrocedent, 518
symptoms, constitutional, of chronic, 517
symptoms premonitory of acute, 515
synonyms, 497
treatment of, 538
Gout, a cause of local paralysis, 1051
of neuralgia, 1044
of neuritis, 1021
Gout, diagnosis of rheumatism from, 566
diagnosis of, from rheumatoid arthritis, 555
Gout, chronic, diagnosis of, from rheumatoid arthritis, 555
Gouty diathesis, a cause of muscular rheumatism, 574
a predisposing cause of erysipelas, 327
[of neuralgia, 1041]
Gouty kidney, 525

Grain, diseased, a supposed cause of cerebro-spinal meningitis, 310
"Grape cure" in scorbutic dysentery, 382
Gravel in gouty diathesis, 520
Gray substance of the spinal cord, functions of the, 943
Grief, a cause of gout, 529
of rheumatoid arthritis, 554
Growth, arrest of, in rickets, 488, 493
Guaiacum in chronic gout, 542
in rheumatism, 570
in muscular rheumatism, 575
in rheumatoid arthritis, 557
Gummata, *see* Nodes.
Gums, bleeding from, in purpura, 461
in yellow fever, 289
redness of, in measles, 110
swelling of, in scurvy, 451
treatment of, 458
Gurgling in right iliac fossa in typhoid fever, 202, 204
Gutta-percha, solution of, in chloroform, for pitting in smallpox, 144
Gymnastics, value of, in treatment of chorea, 716
of infantile paralysis, 1007
of myelitis, 965

HÆMATEMESIS in alcoholism, 677
in cholera, 407
in plague, 315
in purpura, 460, 461
treatment of, 468
in yellow fever, 289
Hæmatine, effusion of, the cause of coloration of the skin in yellow fever, 287
Hæmatoidin in urine after scarlet fever, 91
Hæmatoma of the dura mater, 843
Hæmatorachis, 1007
Hæmaturia, in cerebro-spinal meningitis, 302
in diphtheria, 68
in intermittent fever, 358
occasional in measles, 103, 111
in Pali plague, 318
in plague, 315
in purpura, 691
in remittent fever, 367
in pyæmia, 346
a sequel of scarlet fever, 91
in malignant smallpox, 131
in yellow fever, 290
Hæmoptysis, in hooping-cough, 51
in purpura, 461
in scurvy, 457
in malignant smallpox, 131
in typhoid fever, 203
Hæmorrhoids, from chronic alcoholism, 678
as sequelæ of dengue, 103
in gout, 519
Hair, loss of, in syphilis, 432
thickness of, in rickets, 476, 477
Hallucinations, definition of, 593
Hallucinations—
character of, in delirium tremens, 679
in general paralysis, 606
in melancholia, 593
in mania, 597
in monomania, 599
Hands, deformity of, from gout, 516
Haut mal, le, symptoms of, 769
Head, deformities of the, in idiocy, 603
injury of, a cause of abscess of the brain, 934
of insanity, 591
shape of, in chronic hydrocephalus, 837
Head, deformities of the bones of, in rickets, 480
position of, in rickets, 178
Headache, absence of, in sunstroke, 666
persistence afterwards, 670
due to cerebral diseases, generally, 756
to abscess of the brain, 934, 937
to cerebritis, 855
to simple meningitis, 809
to tubercular meningitis, 822
to cerebral softening, 865
to tumor of the brain, 884
premonitory of cerebral hemorrhage, 924
Headache, in epidemic cerebro-spinal meningitis, 297, 293
in dengue, 99, 100
in diphtheria, 65, 66
in erysipelas, 322
in glanders, 189
in gout, 515, 518
in influenza, 41, 42
in intermittent fever, 357
in roseola, 105
in parotitis, 118
in the plague, 314
in relapsing fever, 277
in remittent fever, 366, 367
in scarlet fever, 85, 88
in smallpox, 132
in typhoid fever, 202, 204
in typhus fever, 254, 258
in varicella, 125
in yellow fever, 284, 286, 292
Health, previous, influence on liability to cholera, 390
Hearing, impaired, *see* Deafness.
Heart, affection of, in gout, 519
treatment of, 546
alteration of relation of, to chest walls in rickets, 493
degeneration of, in yellow fever, 291
disease of, a cause of petechiæ, 461
of vertigo, 691
predisposes to cerebral congestion, 848
relation of, to chorea, 699
to insanity, 590
displacement of, by large spleen, in intermittent fever, 358
fatty degeneration of, in typhoid fever, 218
in typhus, 264
morbid anatomy of, in cholera, 410
Heart, morbid anatomy of—
in pyæmia, 332
in scurvy, 457
hypertrophy of, predisposes to cerebral hemorrhage, 906
inflammation of, never truly gouty, 534
palpitation of, in gout, 515, 518
paralysis of, in diphtheria, 69
relation of diseases of, to acute rheumatism, 563
sounds of, in typhus fever, 256
syphilitic muscular nodes in, 433
weakness of, in scurvy, 455
Heart-burn in gout, 515, 518
treatment of, 545
Heat apoplexy, 661
Heat, diagnostic value of, in myelitis, 960
excessive, a cause of congestion of the brain, 848
Heat, effect of, on scarlet fever poison, 95
sense of, a premonitory symptom of rickets, 476
a cause of disease, *see* Temperature of air.
Hectic in rickets, 482
Hemeralopia in scurvy, 453
[Hemianæsthesia in hysterical cases, 636]
Hemiplegia, from abscess of the brain, 937
epileptic, 753
hysterical, 633
in cerebro-spinal meningitis, 300
in children, 742
diagnosis of, from essential paralysis, 743
due to hemorrhage into the cerebellum, 915
into the corpus striatum, 911
into the crus cerebri, and pons, 913, 914
into the optic thalamus, 913
from meningeal hemorrhage, 842
with insensibility, 926
without loss of consciousness, 925
Hemispheres, cerebral, symptoms of disease in the, 911
Hemorrhage, a predisposing cause of pyæmia, 343
a predisposing cause of rheumatoid arthritis, 554
capillary, in gout, supposed to exist by Gairdner, 522
critical, in relapsing fever, 278
from bladder, in purpura, 461
from the bowels, in cholera, 405
in dysentery, 375, 376, 377
in measles, 111
in plague, 315
in purpura, 460, 461
in remittent fever, 367, 368
in scarlet fever, 90
in scurvy, 453
in typhoid fever, 203, 208
treatment of, 249
in typhus, 258

Hemorrhage—
from cervical bubo in scarlet fever, 97
from gums in scurvy, 451
treatment of, 458
from kidneys in purpura, 461
from lungs in purpura, 461
from the mucous membranes in cerebro-spinal meningitis, 299
in variola maligna, 131, 137
from pericardium in yellow fever, 291
from skin in purpura, 460
from sloughing in neck after scarlet fever, 90
from stomach in yellow fever, 285, 293
into cellular tissue in purpura, 461
into brain in purpura, 461
into muscles in typhus, 265
tendency to, in diphtheria, 66, 67, 68, 73
an unfavorable symptom of diphtheria, 67, 77
in Pali plague, 318
in the plague, 315
in purpura, 461
supposed causes of, 463
in smallpox, 137
in typhoid, an unfavorable symptom, 247
in yellow fever, 289
Hemorrhage, cerebral, 902
relation of, to congestion of the brain, 851
to softening of the brain, 908
Hemorrhage into the spinal cord, 1007
Hemorrhagic diathesis, diagnosis of, from purpura, 467
Hepatitis, gouty, 518
in remittent fever, 368
syphilitic, 434
Hepatization of lung after measles, 111, 112
in pyæmia, 331, 332
Hereditary predisposition in diphtheria, 64
in erysipelas, 322
in gout, 526, 536
in rheumatism, 564
in rheumatoid arthritis, 554
in rickets, 473
Hereditary syphilis, a protection against acquired syphilis, 427
Hereditary taint, a cause of alcoholism, 674, 675
of infantile convulsions, 749
of epilepsy, 763
of cerebral hemorrhage, 905
of hypochondriasis, 623
of hysteria, 633
of insanity, 588
of locomotor ataxy, 989
of neuralgia, 1041
of paralysis agitans, 725
of somnambulism, 659
of spinal irritation, 997
of tubercular meningitis, 818
of wasting palsy, 786, 790
Hereditary taint, influence of, on prognosis of epilepsy, 779
of insanity, 617
of neuralgia, 1041
Hernia, an occasional result of influenza, 42
Herpes, relation of, to neuralgia, 1034, 1039
Herpes of lips, a favorable sign in intermittent fever, 359
Herpetic eruption in cerebro-spinal meningitis, 302
Hiccup, pathology of, 1058
Hiccup, in cholera, 402
treatment of, 419
in remittent fever, 367
in typhoid fever, 203
in yellow fever, 285, 292
Hip-disease a sequela of scarlet fever, 93
Hip-joint, condition of, in rheumatoid arthritis, 552
Hip-joint, disease of the, diagnosis of, from infantile paralysis, 1004
Histrionic paralysis, 1050
Histrionic spasm of the face, 1059
Homburg waters in gout, 547
Homicidal impulse, in melancholia, 594
in dementia, 601
Hooping-cough, article on, 48
causes, 48
complications of, 50, 56
definition, 48
diagnosis, 52
history, 48
morbid anatomy, 52
pathology, 52
prognosis, 53
symptoms, 49
treatment, 53
Hooping-cough, a predisposing cause of measles, 106
of tubercular meningitis, 810
rickets following, 476
Horn-pock, 132, 133
Hot climates, predispose to sunstroke, 661
to tetanus, 978
Human intercourse as a means of diffusion of cholera, 393
of diphtheria, 61
of dengue, 103
of influenza, 37
of typhus fever, 253
Humerus, deformity of, in rickets, 479
Hydatid cysts in the brain, 897
Hydræmia, a predisposing cause of rickets, 473
Hydrocephalus, acute, in rickets, 483
in hooping-cough, 51
chronic, in rickets, 483
Hydrocephalus, chronic, article on, 836
morbid anatomy, 836
symptoms, 837
diagnosis and treatment, 839
Hydrochloric acid, free, in the blood, a supposed cause of rickets, 473
free, in black vomit, 290
internal administration of, in hooping-cough, 56
in rickets, 495
in typhus, 268
topical use of, in diphtheria, 78
in scarlatina, 97
Hydrocyanic acid, in cholera, 418
for vomiting in cholera, 416
in dyspepsia of chronic gout, 545
in hooping-cough, 54
for vomiting in influenza, 40
value of, in insanity, 621
in rickets, 495
in typhoid fever, 247
in yellow fever, 294
Hydrogen in the air of marshes, 353
Hydrophobia, article on, 192
causes in dog, &c., 195
in man, 197
definition, 192
diagnosis, 199
history, 192
morbid anatomy, 199
prognosis, 200
symptoms in dog, &c., 196
in man, 197
synonyms, 192
therapeutics, 200
Hydrophobia, diagnosis of, from tetanus, 971, 978
Hydrorachis, 1018
Hydrothorax, as a sequel of scarlet fever, 93
Hygienics generally considered, 30
Hyoscyamus, in gouty cystitis, 546
in influenza, 46
in rickets, 495
in yellow fever, 294
application of, to rheumatic joints, 572
Hyperæsthesia of skin, in cerebro-spinal meningitis, 298, 299
occasionally occurring in diphtheria, 69
in general paralysis, 606
in hysteria, 635
in meningitis, 809, 823
Hypercinesis, 1055
Hypertrophy of the brain, 888, 899
of the spinal cord, 1015
Hypochondriacal melancholia, 592
Hypochondriasis, article on, 623
definition, 623
nomenclature, 623
history, 624
symptoms, 624
diagnosis, 626
prognosis, 628
etiology, 628
pathology and treatment, 629
Hypochondriasis, diagnosis of, from melancholia, 609, 627
Hypodermic injection, of atropia, in neuralgia, 1045
of arsenic, in chorea, 1043
in neuralgia, 712
of morphia, in delirium tremens, 688
in insanity, 621
in neuralgia, 1044
in torticollis, 1064
in wasting palsy, 799
in writer's cramp, 737
Hypophosphites, the, in treatment of chorea, 717
of chronic alcoholism, 686

Hypopion in smallpox, 134
[Hypostatic pneumonia in typhoid fever, 221, 249
in typhus, 258]
Hysteria, article on, 630
causes, 631
symptoms, 634
interparoxysmal, 634
paroxysmal, 639
pathology, 640
diagnosis, 642
prognosis, 643
treatment, 644
Hysteria, diagnosis of, from epilepsy, 642, 778
from neuralgia, 1040
from tumor of the brain, 886
predisposes to alcoholism, 639
to insanity, 589
relation of, to hypochondriasis, 626
to muscular anæsthesia, 784 *et seq.*
Hysterical mania, 640
paralysis, 638
paralysis agitans, 724
paraplegia, 1000
[Hystero-Epilepsy, 649]

ICE, sucking of, in cholera, 416, 417
in diphtheria, 78
in dysentery, 380
in measles, 114
in parotitis, 121
in hæmetemesis in purpura, 468
in remittent fever, 370
for sore throat in scarlet fever, 96, 97
in typhus, 268
in yellow fever, 294
to abdomen for hemorrhage in typhoid fever, 249
to head in erysipelatous meningitis, 329
to head in cerebro-spinal meningitis, 313
Ice to spine, in epilepsy, 782
Ichorous fluids, entrance of, into circulation, a cause of some symptoms of pyæmia, 340
Ideation, perverted in hysteria, 634
in insanity, 592
Idiocy, description of, 603
varieties, 604
relation of, to epilepsy, 777
to meningitis, 825
following infantile convulsions, 744
Idiopathic neuroma, 1022
tetanus, 976
Idiosyncrasy, effect of, in modifying syphilis, 426
Ileo-cæcal valve, swelling of, in typhoid fever, 210
Ileum, morbid anatomy of, in typhoid fever, 209
Ilia, deformity of, in rickets, 480, 493
Iliac fossa, tenderness and gurgling of, in typhoid fever, 202
treatment of, 248
Illusion, definition of an, 593 (note).
Imbeciles, intellectual, 604
Imbeciles—
moral, 602
Imbecility, a sequel of typhoid fever, 209
Imperial drink for thirst in measles, 114
Incisor teeth, malformation of, in congenital syphilis, 441
Incontinence of urine, nocturnal, 660
in sunstroke, 666
Incubative period, of cholera, 392
of chicken-pox, 125
of diphtheria, 63
of glanders in horses, 183, 184
in man, 189
of hooping-cough, 48
of hydrophobia in the dog, 196
in man, 198
of influenza, 38
of intermittent fever, 355
of measles, 106
of parotitis, 120
of remittent fever, 366
of rickets, 476
of scarlet fever, 84
of smallpox, 129
of syphilis, primary, 443
constitutional, 424
of typhus fever, 254
of yellow fever, 282
India, residence in, as a predisposing cause of cholera, 390
Indian hemp, in treatment of delirium tremens, 686
of neuralgia, 1045
Indigestion, predisposes to cerebral congestion, 848
to convulsions, 757
to epilepsy, 765
Induration of the brain, 888
Induration of the spinal cord, 1008
a result of myelitis, 962
Induration of syphilitic chancre, 424
absorption of, under mercurial treatment, 435
of tissues in tertiary syphilis, 431
Infantile paralysis, article on, 1004
symptoms, 1004
prognosis, 1005
treatment, 1006
Infantile remittent fever, rickets mistaken for, 475
Infants, convulsions in, 738
Infarcts in the brain, 875
Infection, *see* Contagion.
Inflammation, cerebral, in chorea, 707
relation of, to pain and spasm, 947
Inflammation, production of, by occluded circulation, 336
[Inflammation, treatment of, 31]
Influenza, article on, 33
consideration of special symptoms, 41
definition, 33
diagnosis, 43
general course of disease, 41
history, 34
morbid anatomy, 44
Influenza—
mortality, 43
pathology, 44
prognosis, 44
spread of disease, 34
synonyms, 38
treatment, 45
varieties, 43
Influenza, its relation to other epidemic diseases, 38
its relation to diseases of brutes, 39
prevalence of, during cholera epidemics, 391
Infra-mammary pain, neuralgic, 1034
Inherited syphilis, diagnosis of, 440
[Inhibition in pathology of reflex paralysis, 1003]
Injections into veins in cholera, 421
Injury, an exciting cause of rheumatoid arthritis, 554
Injury of the head, a cause of abscess of the brain, 934
of cerebral hemorrhage, 904, 929
of insanity, 591
Injury to nerve, a cause of facial palsy, 1051
of neuralgia, 1028, 1033
Injury to spinal cord, effects of, in different regions, 946
a cause of spinal irritation, 997
of spinal meningitis, 955
of wasting palsy, 787
Inoculability of influenza, 38
Inoculation, in diphtheria, 63
in glanders, 185
in scarlet fever, 95
of sheep-pox, 128
of cows with variolous matter, a means of obtaining vaccine lymph, 176
for smallpox, 156
in Bengal, 398
cases justifying, 157
history of, 156
modification of course of smallpox by, 139
mortality from, 157
phenomena of, 157
of syphilis, 424, 426
impossible in varicella, 124
Insanity, article on, 584
synonyms, 584
definition, 584
classification, 585
causes, 587
varieties and their symptoms, 591
diagnosis, 607
prognosis, 616
therapeutics, 618
[Insanity, physiological classification of, 586
American statistics of, 591
without cerebral disease, 613]
Insanity, relation of, to alcoholism, 675
to hypochondriasis, 628
Insanity following typhus, 259
Insolation, article on, 661
a cause of insanity, 591, 666
diagnosis of, from apoplexy, 667

Insomnia, in chronic alcoholism, 675
in cholera, 402,
treatment of, 419
in delirium tremens, 679
treatment, 685
in insanity, 596
in smallpox, treatment of, 142
in tetanus, 974
in torticollis, 1062
in typhus, 258
treatment of, 268
Inspiration, characters of, in hooping-cough, 50
difficulty of, in rickets, 481
Insufflation of lungs in rickets, 494
Intellect, state of, in cerebritis, 855
in cerebro-spinal meningitis, 297, 301
in cholera, 400
in dengue, 100
in diphtheria, 65
in dysentery, 376
in erysipelas, 322
in chronic hydrocephalus, 839
in influenza, 41
in intermittent fever, 357, 358
in measles, 107
in meningitis, 809
in the plague, 314
in pyæmia, 344, 346
in remittent fever, 367
in rickets, 476, 481
in scurvy, 451, 453
in malignant smallpox, 132
in acute cerebral softening, 858
in tumor of the brain, 834
in typhoid fever, 203
in typhus, 258
in yellow fever, 284, 285
see also, Mind, state of.
Intellectual occupations a cause of insanity, 588
Intemperance, a predisposing cause of cholera, 390
a cause of delirium in smallpox, 130
a cause of general paralysis, 605
of idiocy, 604, 678
of insanity, 589, 607
of purpura, 463
a predisposing cause of pyæmia, 343
a predisposing cause of typhus, 252
predisposes to neuralgia, 1042
to "rheumatic meningitis," 812
to mercurial tremor, 802, 805
Intercostal neuralgia, 1031
Intercostal rheumatism, 574
Intermarriage of relations a cause of idiocy, 604
a supposed cause of rickets, 473
Intermissions, in cerebro-spinal meningitis, 311
in erysipelas, 325
in influenza, occasionally, 41
in intermittent fever, 355, 357
Intermissions—
in the pyrexia in typhoid fever, 228
in relapsing fever, 277
Intermittent fever, article on, 354
causes, 356
definition, 354
diagnosis, 358
history, 354
modes of commencement, 354
morbid anatomy, 358
prognosis, 359
symptoms, 356
synonyms, 354
treatment, 360
varieties of, 355
Intermittent fever, coexistence of, with typhoid fever, 228
complicating cerebro-spinal meningitis, 303
localities of, in England, 228
relation of, to typhoid fever, 228, 233
Intestines, amyloid degeneration of mucous membrane of, in rickets, 483
atrophy of, in chronic dysentery, 379
thickening of, in chronic dysentery, 639
gangrene of, in dysentery, 379
hemorrhage from, in purpura, 460, 461
morbid anatomy of, in cholera, 411
in diphtheria, 76
in dysentery, 378
in the plague, 316
in pyæmia, 334
in scurvy, 457
in typhoid fever, 209
in typhus, 265
in yellow fever, 292
muscular rheumatism in, 574
pathology and lesions of, in typhoid fever, 219
perforation of, in typhoid fever, 203, 208, 210
tubercular ulceration of, diagnosis of, from typhoid ulceration, 245
ulceration of, in pyæmia, 35
in typhoid fever, 210
ulceration of, in typhus fever, complicated with dysentery, 265
Intoxication, alcoholic, physiology of, 671
Invasion, of cerebro-spinal meningitis, 297
of cholera, 398
in dengue, 99
of diphtheria, 65
of dysentery, 373
of erysipelas, 322
of gout, 512
of glanders, 189
of hooping-cough, 49
of hydrophobia, 198
of influenza, 41
of intermittent fever, 354, 356
of measles, 106
of parotitis, 118
of plague, 314
of pyæmia, 344
Invasion—
of relapsing fever, 277
of remittent fever, 365
of rickets, 475
of acute rheumatism, 559
of muscular rheumatism, 573
in scarlet fever, 85
of scurvy, 451
of smallpox, 132
of syphilis, 424
of typhoid, 202
of typhus fever, 254
of yellow fever, 284
Iodide of potassium, in treatment of mercurial tremor, 806
of local poisoning, 807
of spinal meningitis, 956
of syphilitic disease of the brain, 901
see also Potassium, iodide of.
Iodine, in treatment of chronic hydrocephalus, 840
in rheumatoid arthritis, 575
in rickets, 496
inhalations for chronic laryngitis after measles, 116
external use of, in rheumatoid arthritis, 557
Ipecacuanha, in dengue, as an emetic, 104
in diphtheria, 82
in dysentery, 380, 381, 383
in influenza, 46
in measles, 115
in purpura, 468
in hooping-cough, 54
as an emetic in relapsing fever, 280
in gonorrhœal rheumatism, 579
in rickets, 496
in pulmonary complications of typhoid, 249
"Irish purpuric disease," 462
Iritis, in cerebro-spinal meningitis, 298, 300
in congenital syphilis, 430
in secondary stage of syphilis, 425
frequent relapse of, 436
influence of mercury on, 436
treatment of, 436
Iritis, complicating facial neuralgia, 1038, 1046
Iron, excess of, in blood in purpura, 465
presence of, in black vomit of yellow fever, 290
Iron, after cholera, 419
after dengue, 104
in diphtheria, 78, 79
in erysipelas, 328
in chronic gout, 545
in hooping-cough, 55
after influenza, 47
[in chronic intermittent, 364]
after measles, 116
in rheumatoid arthritis, 556
in rheumatic pericarditis, 572
in rickets, 496
after smallpox, 143
Iron, value of, in treatment of chorea, 711
Iron, acetate of, in diphtheria, 79
Iron and ammonia, citrate of, in rickets, 496

Iron and quinine, citrate of, in dysentery, 383
in rickets, 496
Iron, carbonate of, in hooping-cough, 55
Iron, iodide of, in rheumatoid arthritis, 557
in rickets, 496
Iron, magnetic oxide of, in rickets, 496
Iron, perchloride of, in diphtheria, 79
in scarlatinal dropsy, 97
for hemorrhage in typhoid fever, 249
Iron, pernitrate of, in diphtheria, 79
in dysentery, malarious, 382
chronic, 383
Iron, phosphate of, after intermittent fever, 364
in rickets, 496
Iron, potassio-tartrate of, in acute rheumatism, 573
Iron, sesquichloride of, in cerebro-spinal meningitis, 313
in diphtheria, 79
in erysipelas, 828
in purpura, 467
in rickets, 496
Iron, sulphate of, in hooping-cough, 55
Irrigation, cold, in meningitis, 816
Irritability, muscular, in cerebral softening, 863
electric, *see* Electric irritability of muscles.
Irritation, eccentric, a cause of epilepsy, 766
spinal, 991
Itching of skin, in dengue, 101
treatment of, 104
in roseola, 105
in smallpox, 143
treatment of, 143
in varicella, 126

JACTITATION in dengue, 100
James's powder in remittent fever, 371
Jaundice, as a sequela of dengue, 103
in hypochondriasis, 626
occasionally present in influenza, 43
in relapsing fever, 277, 278
in remittent fever, 368
in pyæmia, 333, 344, 345, 346
in scarlet fever, 88
in yellow fever, 287
purpura attending, 463
Jaw, occasionally fixed in rheumatoid arthritis, 551
Jaw, lower, elongation of, in rickets, 480
movements of, impaired in mumps, 119
Joints, ankylosis of, from gout, 514, 516, 525
favored by local blood-letting, 540
effect of rheumatism on, 531
effusion into, in gout, 514
in gonorrhœal rheumatism, 576
in rheumatoid arthritis, 553
Joints—
fatty degeneration of, in rheumatoid arthritis, 554
morbid anatomy of, in acute rheumatism, 563
in rheumatoid arthritis, 553
in gout, 523
number affected in gout, 515
pain in, in dengue, 99
before purpura, 462
in pyæmia, 344
in relapsing fever, 278
neuralgic, after rheumatism, 560
in rickets, 476, 482
in syphilis, 425
state of, in acute rheumatism, 559, 561
in gonorrhœal rheumatism, 576
in rheumatism, sub-acute, 560
in chronic rheumatoid arthritis, 551
in acute rheumatoid arthritis, 552
in acute gout, 513, 514
local treatment of, 540
stiffness of, in scurvy, cause of, 457
suppuration of, in cerebro-spinal meningitis, 298, 303
in pyæmia, 331, 334, 344, 346
after scarlet fever, 91, 95
swelling in flexures of, in scurvy, 452
treatment of, 458
swelling of, in dengue, 99
Jugular veins, dilatation of, in rickets, 476
Jungle fever, *see* Remittent fever, 365

KERATITIS, interstitial in hereditary syphilis, 430, 440, 442
Kidneys, disease of the, from chronic alcoholism, 684
deficient action of, a cause of local paralysis, 1049
effect of lead on, 530
morbid anatomy of, in cerebro-spinal meningitis, 306
in cholera, 411
in diphtheria, 73, 76
in erysipelas, 326
in gout, 519, 525, 534
in purpura, 464
in pyæmia, 333
in scarlet fever, 94
in typhus, 265
in yellow fever, 292
suppuration of, diagnosis of, from pyæmia, 348
see also Bright's disease.
Kissingen waters in gout, 547
Knee, condition of, in rheumatoid arthritis, 552
Knock-knee in rickets, 478

LABIA, diphtheritis of, in measles, occasionally, 113
occasionally affected in parotitis, 119
Lactation, insanity of, 593
Lactation, prolonged, a predisposing cause of acute rheumatism, 565
Lactation, prolonged, a predisposing cause of rheumatoid arthritis, 554
Lactic acid, in the blood, a supposed cause of rheumatism, 566
free in the blood, a supposed cause of rickets, 487
in urine in acute rheumatism, 563
in urine in rickets, 484
Lacunæ of bone, formation of, in rickets, 490
Larch bark, tincture of, in purpura, 468
Laryngeal asthma in rickets, 483
Laryngeal nerve, recurrent, supposed seat of hooping-cough, 52
Laryngismus stridulus, 741
connection of, with rickets. 838
common in hydrocephalic children, 742
Laryngismus stridulus, in rickets, 483
treatment of, 497
Laryngitis, complicating measles, 111
treatment of, 115
in relapsing fever, 278
complicating typhoid fever, 223
chronic, an occasional sequel of influenza, 42
of measles, 111
syphilitic, 432
treatment of, 437
Laryngotomy in diphtheria, 83
in erysipelas, 329
Larynx, affection of, in rheumatoid arthritis, 552
condition of, in diphtheria, 67
in glanders, 191
in smallpox, 146
smallpox eruption in, 131
ulceration of, in typhoid fever, 217
in tertiary syphilis, 432
Latent scarlet fever, 89
Lateral columns of the spinal cord, functions of the, 913
[Lateral spinal sclerosis, 1069]
Lateral ventricles of the brain, hemorrhage into, 903, 911
Lead, influence of, on excretion of uric acid by the kidney, 530
Lead-poisoning, diagnosis of pains in, from muscular rheumatism, 575
a cause of gout, 530
symptoms of, 807
treatment, 807
diagnosis of, from tumor of the brain, 887
from wasting palsy, 790
from writer's cramp, 734
Lead, acetate of, in cholera, 416
in diphtheria, 79
in dysentery, 383
in influenza, 46
in hooping-cough, 56
in purpura, 468
in hemorrhages in purpura, 468
in typhoid fever, 248, 249

Lead, acetate of—
as a lotion in otorrhœa, 116
[Lead cerate in rheumatoid arthritis, 558
in scrofula of bones, &c., 511]
Leaping ague, 702
[Lemonade as a diuretic, 97]
Lemon-juice in scurvy, 458, 459
Lichen, febrile, diagnosis of, from smallpox, 135
vaccine, 159
Liebig's beef tea, 114, footnote.
Ligaments, affected in rare cases of erysipelas, 326
gouty deposits in, 516
state of, after articular rheumatism, 564
weakening of, in rickets, 493
Ligamentum teres, destruction of, in rheumatoid arthritis, 553
Light, intolerance of, in hysteria, 635
in meningitis, 809
want of, a cause of rickets, 473
Limbs, stiffness of, in dengue, 99
paralysis of, in diphtheria, 70
Lime-juice, in acute rheumatism, 570
as a prophylactic in scurvy, 448, 455, 458, 459
Lime, salts of, in gout, 544
Lime, phosphate of, found in rare cases in chalk-stones, 517
Lime-water, in cholera, 418
as a gargle in diphtheria, 70
in gout, 544
in rickets, 495, 496
in yellow fever, 294
Lips, redness of, in measles, 110
Lithia in gout, acute, 539
chronic, 544
Lithia, carbonate of, external application of, to chalk-stones, 546
Liver, abscess of, in dysentery, 379
agency of, in decomposing poisons introduced into the portal system, 218, 224
agency in elimination of poison of yellow fever, 281
antiseptic influence of, on chyme, 219
cirrhosis of, in alcoholism, 677
disease of, in dengue, 103
a cause of purpura, 463, 465
derangement of, in malarious dysentery, 377
in chronic gout, 518
derangement of, the cause of all the symptoms in typhoid fever, 218
enlargement of, in relapsing fever, 279
in rickets, 483
in typhus, 265
morbid anatomy of, in cerebro-spinal meningitis, 306
in cholera, 411
in diphtheria, 76
in malarious dysentery, 379
in erysipelas, 323
Liver, morbid anatomy of—
in intermittent fever, 359
in purpura, 463
in pyæmia, 333
in relapsing fever, 279
in scurvy, 457
in syphilis, 434
in typhoid fever, 215
in typhus, 265
in yellow fever, 292
syphilitic affections of, 434
tenderness of, in relapsing fever, 277
Lividity of the face in epilepsy, 771
Lobelia, in influenza, 46
in hooping-cough, 55
Locality, change of, to avoid cholera, 422
[Localization of brain lesions, recent researches on, 916]
Localized paralysis agitans, 725
Local spasms, article on, 1055
Lochia, changes of, in puerperal pyæmia, 344
Locomotor ataxy, article on, 980
definition, 980
symptoms, 981
post-mortem appearances, 982
causes, 989
prognosis, 989
diagnosis, 989
treatment, 990
Locomotor ataxy, diagnosis of, from muscular anæsthesia, 784
from paraplegia, 789
Logwood, decoction of, in dysentry, 383
Lucid intervals, in mania, 597
in melancholia, 597
Lumbago, 574
Lunatics, increasing number of, 587
Lung, apoplexy of, in cerebro-spinal meningitis, 303
in purpura, 462
atrophy of, in hooping-cough, 51
collapse of, in cholera, 410
in influenza, 44
in rickets, 494
Lungs, chronic disease of the, a cause of abscess of the brain, 935
congestion of, in alcoholism, 683, 684
in apoplexy, 904
in cholera, 421
in erysipelas, 326
in hooping-cough, 50
in influenza, 41, 42, 44
in malignant measles, 108
in sunstroke, 667
a cause of death in rickets, 482
in typhoid fever, 203, 204
local, in pyæmia, 332
consolidation of, after typhus, 258
gangrene of, in measles, 113
in melancholia, 595
in scurvy, 452, 456
after typhus, 262
emphysema of, in rickets, 494
Lungs—
inflammation of, from gout, 519
treatment of, 546
morbid anatomy of, in cholera, 410
in diphtheria, 76
in glanders, 191, 192
in hydrophobia, 199
in influenza, 44
in pyæmia, 331
in scarlatinal dropsy, 92
in scurvy, 456
in smallpox, 146
in typhoid fever, 217
in yellow fever, 291
œdema of, in cerebro-spinal meningitis, 303
physical examination of, during life, in complications of hooping-cough, 51
in influenza, 42
in measles, 107, 112
in pyæmia, 345
in scurvy, 452
in typhus fever, 258
Lupus, in hereditary syphilis, 429
phagedænic, in tertiary syphilis, 432
Lymph, absorption of, under mercurial treatment of syphilis, 436, 437
effusion of, in pyæmia, 331
on pericardium, 333
on pleuræ, 332
effusion of, in syphilitic keratitis, 442
in syphilitic iritis, 425
in syphilitic orchitis, 434
Lymphatic glands, albuminoid degeneration of, in rickets, 483
enlarged, diagnosis of, from parotitis, 120
inflammation of, after vaccination, 159
state of, in dengue, 101
in diphtheria, 65, 66, 76
in erysipelas, 324
in farcy, 190
in glanders, 189, 192
in hydrophobia, 198
in measles, 109
in the plague, 314, 315, 316
in scarlet fever, 86, 88, 90
in syphilis, 433
in typhoid fever, 215, 218
suppuration of, after typhus, 262
Lymphatic vessels, inflammation of, in erysipelas, 324
in farcy, 190
Lymphatics, distribution of the, in the brain, 850

MACULÆ of typhus, 256
Magnesia in chronic gout, 544
in rickets, 495
sulphate and carbonate of, in gout, 540
sulphate of, in chronic gout, 545
in purpura, 467
Malaria, 352
causes of, 352
distribution of, 353

Malaria—
effects of, 353
supposed nature of, 353
Malaria, a supposed cause of cerebro-spinal meningitis, 311
a cause of dysentery, 374
of neuralgia, 1029
necessary for the production of ague, 356
relation of, to cholera, 388
[Malaria, protection from, by trees, 353
disappearance of, with building of towns, 353]
Malarial cachexia, treatment of, 363
Malarial fevers, article on, 352
intermittent fever, 354
nature of malaria, 353
remittent fever, 365
[Malarial stupor, simulating congestion of the brain, 854]
Malarious dysentery, 373, 377
treatment of, 382
Malformations, congenital, of the brain, 603
of the meninges, 844
of the spinal cord, 1018
Malic acid in scurvy, 455
Malignant dysentery, 373, 377
Malignant measles, 107
Malignant scarlet fever, 88
Malignant smallpox, 128, 131
Malleoli, enlargement of, in rickets, 481
Malt liquors, causes of gout, 527, 528
Mammæ, inflammation of, in parotitis, 119
Mania, 595
acute, 596
from alcoholism, 680
dancing, 647
epileptic, 776
hysterical, 640
partial, 599
puerperal, 598
recurrent, 598
sine delirio, 601
Mania in retrocedent gout, 519
in scurvy, 453
in tertiary syphilis, 440
Mania, acute, diagnosis of, from hydrophobia, 199
in tertiary syphilis, 433
Marasmus, a sequel of typhoid fever, 208
Marienbad waters, in gout, 547
presence of lithia in, 544
Marshes, a cause of malaria, 353
composition of air of, 353
Mastication, difficulty of, in spinal meningitis, 954
in mumps, 119
Masticatory spasm, 1059
Masturbation, a cause of insanity, 589, 599
of epilepsy, 766
of wasting palsy, 788
Maturation stage of smallpox, 129
Measles, article on, 106
complications, 111
definition, 106
diagnosis, 113
prognosis, 114
symptoms, 106
Measles—
synonyms, 106
treatment, 114
varieties, 107
Measles, a cause of purpura, 463
complicating vaccination, 160
diagnosis of, from smallpox, 135
of dengue from, 103
of roseola from, 106
from scarlet fever, 94
from typhus, 262
case of, mistaken for dysentery, 378
hemorrhagic, diagnosis of, from purpura, 466
malignant, 107
rash in, 107
malignant, diagnosis of, from cerebro-spinal meningitis, 304
predisposes to tubercular meningitis, 819
[Measles, typhus, in U. S. Army, 108
German, 117]
Meat, diseased, how far a cause of typhoid fever, 243
Meatus auditorius occasionally affected in diphtheria, 68
Medulla oblongata, the centre of the epileptic zone, 777
symptoms of hemorrhage into, 915
congestion of, in hydrophobia, 199
how far concerned in hooping-cough, 49, 52
Melæna in purpura, 461
Melancholia, 592
acute, 595
attonita, 594
from alcoholism, 680, 683
diagnosis of, from dementia, 609
from hypochondriasis, 627
prognosis, 616
Melancholia, occasional occurrence of, in tertiary syphilis, 433
Melanoid growths in the brain, 891
Membrane, false, *see* Exudation.
Memory, loss of, in dementia, 600
in cerebral congestion, 845
in mercurial poisoning, 804
in softening of the brain, 884
state of the, in epileptics, 774
in mania, 597
Meningeal hemorrhage, 840
treatment of, 842
Meninges of brain, affected in tertiary syphilis, 433
congestion of, in diphtheria, 76
supposed gouty deposits in, 518
state of, in cerebro-spinal meningitis, 305
suppurative inflammation of, in diphtheria, 76
Meninges, the, adventitious products in, 843
congenital malformations of, 844
Meningitis, acute, in the young, 809
in adults, 810, 817
partial or local, 811, 813
rheumatic, 811
syphilitic, 812
tubercular, 817
a complication of epilepsy, 776
diagnosis of, from acute mania, 607
from delirium tremens, 815
from typhus and typhoid fevers, 815, 830
Meningitis, chronic, 816
a cause of general paralysis, 816
diagnosis of, from epilepsy, 779
from cerebral softening, 882
from tumor of the brain, 887
Meningitis, cerebral, in erysipelas, 327
in influenza, 42
in typhus, 265
Meningitis, epidemic cerebro-spinal, *see* Cerebro-spinal meningitis.
Meningitis, simple, article on, 808
definition, 808
symptoms of acute form, 808
varieties, 811
course, 813
pathological anatomy, 814
etiology, 814
diagnosis and treatment, 815
symptoms of the chronic form, 816
treatment, 817
Meningitis, spinal, article on, 951
symptoms, 951
post-mortem appearances, 955
causes, 955
diagnosis and prognosis, 955
treatment, 956
diagnosis of, from myelitis, 963
Meningitis, spinal, in gout, 519
in acute rheumatism, 560
treatment of, 572
Meningitis, tubercular, article on, 817
causes, 817
symptoms in the child, 818
meningitis of the base, 820
of the vertex, 827
symptoms in the adult, 829
diagnosis, 829
morbid anatomy, 832
prognosis and treatment, 835
Menorrhagia in purpura, 461
in yellow fever, 289
a predisposing cause of rheumatoid arthritis, 554
Menstruation, a cause of roseola, 105
a predisposing cause of erysipelas, 322
effect of influenza on, 43
effect of typhus on, 261
supposed influence of, on occurrence of rheumatism, 564
supposed preventive of gout, 527, 536

Menstruation—
disorders of, relation of, to hysteria, 633
to insanity, 590
irregular, a predisposing cause of rheumatoid arthritis, 554
suppression of, a cause of spinal congestion, 968
of rheumatism, 565
of somnambulism, 660
Mercurial inunction in rheumatic pericarditis, 571
Mercurial tremor, 801
Mercury, diagnosis of pains from, from muscular rheumatism, 575
in cerebro-spinal meningitis, 313
in cholera, 418
in diphtheria, 82
in dysentery, 383
in gout, 540
in influenza, 46
in remittent fever, 371
in acute rheumatism, 568
in rheumatic pericarditis, 571
in rickets, 495
in scurvy, danger of, 452
in typhoid fever, 247
in syphilis, 429, 435
how far preventive of constitutional syphilis, 429
early use of, not preventive of constitutional syphilis, 436
modes of administration of, in syphilis, 438
local use of, in syphilis, 438
value of, in treatment of chronic hydrocephalus, 839
of insanity, 622
of meningitis, 815
of syphilitic disease of the brain, 901
Mercury, bichloride of, in syphilis, 438
Mercury, biniodide of, ointment of, for enlarged spleen, 364
Mesenteric glands, atrophy of, after typhoid fever, 208
enlargement of, in rickets, 482
inflammation of, in scarlatina, 233
morbid anatomy of, in plague, 319
morbid anatomy of, in typhoid, 205, 215
tubercular disease of, in pulmonary phthisis, 221
tubercle of, diagnosis of, from typhoid, 245
Mesmerism, the theory of, 656
Mesocolic glands, morbid anatomy of, in typhoid, 215
Metacarpal bones, morbid anatomy of, in rheumatoid arthritis, 551
Metallic tremor, article on, 801
synonyms, 801
definition, 801
mercurial tremor, causes, 801
symptoms, 802
course and prognosis, 804
diagnosis, 805
Metallic tremor—
pathology and morbid anatomy, 805
treatment, 805
lead tremors, 806
symptoms, 807
prognosis, 807
diagnosis and treatment, 807
[Metalloscopy and metallotherapy, 637]
Metastastis in gout, 518
treatment of, 546
in parotitis, 199
Metatarso-phalangeal articulation of great toe, commonly affected in gout, 513, 535
morbid anatomy of, in gout, 524
[Methomania, 681
treatment of, 689]
Microscopic appearances, of the brain, in abscess of the brain, 936
in congestion, 851, 852
in softening, 875
of nodes in the dura mater, 812
of the muscles in wasting palsy, 790
of the spinal cord, in locomotor ataxy, 983
in tetanus, 977
in wasting palsy, 792
Middle cerebral artery, embolism of, 869
Migraine, or sick headache, 1031
Miliaria, general pathology of, 122
in rheumatic fever, 123
in scarlet fever, 85, 88
in scarlet fever rash, 123
Miliary aneurisms in the brain, *see* Aneurisms.
Milk, anti-scorbutic properties of, 459
[contamination of, a cause of typhoid fever, 243]
want of, a cause of rickets, 474
Milk, ass's, in rickets, 495
Millbank prison, dysentery at, 374
Mind, activity of, a predisposing cause of diphtheria, 64
anxiety of, a cause of rheumatoid arthritis, 554
depression of, before purpura, 462
depression of, as a predisposing cause of scurvy, 450
exertion of, a cause of gout, 529
Mind, state of, in chronic alcoholism, 676
in chorea, 699
in congestion of the brain, 845
in chronic cerebral softening, 864
in delirium tremens, 679
in epileptics, 773
in general paralysis, 606
in hypochondriasis, 625
in hysteria, 635
in locomotor ataxy, 986
in torticollis, 1062
Mineral waters in gout, 547
Mineral waters—
in rheumatoid arthritis, 557
Mist, cholera, of Mr. Glaisher, 387
Moisture, a cause of dysentery, 374
of purpura, 463
a cause of articular rheumatism, 559, 565
of gonorrhœal rheumatism, 576
of muscular rheumatism, 574
of rickets, 473
a predisposing cause of scurvy, 450
influence of, on cholera, 387
Moisture of the atmosphere, relation of, to the occurrence of sunstroke, 666
Mollities ossium, diagnosis of, from rickets, 485
Monomania, course and symptoms, 599
prognosis, 617
treatment, 622
relation of, to mania, 598
to dementia, 598
Monomanie, instinctive et raisonnante, 602
Moral imbeciles, 602
Moral insanity, 596, 601
diagnosis, 609
treatment of, 622
Morbid anatomy, generally considered, 28
Morbid sleep, causes of, 659
Morbilli, *see* Measles.
Morning sickness, from alcoholism, 677
Morphia, in cerebro-spinal meningitis, 313
in cholera, 416, 418
in diphtheria, 78
in hooping-cough, 55
in influenza, 46
in acute rheumatism, 568
in smallpox, 142
in yellow fever, 294
endermic use of, in muscular rheumatism, 956
enemata of, in hemorrhage in typhoid fever, 249
external application of, in gout, 540
to rheumatic joints, 572
Morphia, value of, in treatment, *see* Opium.
injection of, *see* Hypodermic injection.
Mortality, in cerebro-spinal meningitis, 303
in cholera, 408
from convulsions, 749
from dengue, 98
in diphtheria, 17
in dysentery, 381
in erysipelas, 327
in glanders, 192
from gout, 537
in hooping-cough, 53
in hydrophobia, 194
in influenza, 43
from insanity, 618
in intermittent fever, 356
in measles, 114
from paralysis agitans, 729
in plague, 317
in pyæmia, 347
in relapsing fever, 279

Mortality—
in remittent fever, 368, 370
in rheumatism, 567
from rickets, 474
in scarlet fever, 94
in scurvy, 447, 457
in smallpox, 145
in inoculated smallpox, 156
from sunstroke, 668
in typhoid fever, 247
in typhus, 265
in varicella, 127
amongst workers in mercury, 804
Mortality, to populations, from cholera, 386, 388, 392
from dysentery, 373, 381
from smallpox, 167
Mother, health of, influence of, in production of rickets, 473
Motorial phenomena, abnormal, in epileptics, 774, 776
in congestion of the brain, 845
in hysteria, 637
in meningitis, 823
in softening of the brain, 860
Mouth, bleeding from, in yellow fever, 289
distortion of the, in cerebral hemorrhage, 913
in facial paralysis, 1050
in tetanus, 971
gangrene of, in measles, 112
scars around, in hereditary syphilis, 441
Movement cure, the, efficacy of, in chorea, 716
in myelitis, 965
Movement, relation of, to pain, in cerebral meningitis, 809
in spinal meningitis, 953
in neuralgia, 1033
Mucous membranes, affection of, in secondary syphilis, 424, 426
in tertiary syphilis, 431
Mucus in stools of dysentery, 375, 376, 377
vomiting of, in dengue, 100
[Multiple sclerosis, 1011]
Mumps, *see* Parotitis.
Murexide test for uric acid, 521
Murmur, cardiac, in rheumatic endocarditis, 560
occasional, in scarlet fever, 93
Muscæ volitantes, in chronic alcoholism, 676
in congestion of the brain, 845
Muscles, affected rarely in erysipelas, 325
extravasations of blood into, in purpura, 461
in scurvy, 456
morbid anatomy of, in pyæmia, 334
pain in, in epidemic cerebro-spinal meningitis, 297, 298
rigidity of, in typhus, 255, 259
syphilitic affections of, 433
twitching of, in dengue, 100
weakness of, in rickets, 477, 482
Muscles, electrical state of, *see* Electric condition.
Muscles, chiefly or primarily affected, in alcoholism, 676
in chorea, 698
in general paralysis of the insane, 605, 999
in general spinal paralysis, 999
in hysterical paralysis, 638, 1000
in lead palsy, 807
in mercurial tremor, 802
in paralysis agitans, 720
in infantile paralysis, 1006
in wasting palsy, 788, 789
in writer's cramp, 733
Muscles, prolonged contraction of, in spinal irritation, 995
spasmodic contractions of, in tetanus, 971
Muscular anæsthesia, article on, 783
diagnosis of, from locomotor ataxy, 784
relation of, to paraplegia, 784
Muscular atrophy, progressive, 786
diagnosis of, from infantile paralysis, 1006
from injury to nerve-trunks, 1050
from lead-poisoning, 790
Muscular fibre, degeneration of, in diphtheria, 74
during purpura, 464
in scarlet fever, 94
in typhus, 264
in typhoid fever, 217
softening of, in yellow fever, 291
of heart, state of, in pyæmia, 333
of intestine, changes in, in typhoid ulceration, 210
Muscular sense, the, 785
loss of, in general paralysis, 606
in locomotor ataxy, 987
hallucinations of, in mania, 597
Muscular tremor, from alcoholism, 676
from mercurial poisoning, 802
from paralysis agitans, 720
Mushrooms, poisoning by, resembling typhoid fever, 243
Musk, in hooping-cough, 56
in parotitis, 123
in typhus, 268
Myalgia, diagnosis of, from neuralgia, 1040
[Myalgia, of Inman, 573]
Myelitis, article on, 956
symptoms, 956
post-mortem appearances, 962
causes, 963
diagnosis, 963
prognosis and treatment, 964
Myelitis, complicating typhoid fever, 225
Myelitis convulsiva, 700

NACHPOCKEN, 173
Nails, affection of, in syphilis, 432
Names of disease, 17
Naphtha, in treatment of chorea, 715
Narcotics, in treatment of acute mania, 621
of infantile convulsions, 750
of delirium tremens, 686
Nares, discharge from, in glanders, 189, 190
inflammation of, in congenital syphilis, 429
occlusion of, in diphtheria, an unfavorable symptom, 77
treatment of, 79
Nasal diphtheria, a complication of scarlet fever, 90
Nasal fossæ, affection of bones of, in syphilis, 433
mucous membrane of, state of, in diphtheria, 65
state of, in glanders, 191
state of, in influenza, 41
Natural history of disease, 21
Nausea, with vomiting, *see* Vomiting.
Nausea, in mild dysentery, 375
in invasion of intermittent fever, 355, 356
an early symptom of malarial poisoning, 355, 356, 365
in purpura, 462
Necrosis of bones, a sequel of scarlet fever, 90
in scurvy, 453, 456
of jaw, a sequela of scarlet fever, 93
of long bones, from syphilis, 433
of nasal bones, in chronic glanders, 191
of palate and nasal bones, in syphilis, 433
Negroes predisposed to tetanus, 978
Nephritis, in gout, 519, 520
diagnosis from renal calculus, 520
from inflammation of bladder, diagnosis of, from pyæmia, 348
Nerves, division of, for cure of neuroma, 1025
of neuralgia, 1047
Nerves, syphilitic deposit in, 435
Nervous constitution or diathesis, the, predisposes to alcoholism, 673
to insanity, 588
to spinal irritation, 996
relation of, to hysteria, 632
Nervous depression as a cause of gout, 529
Nervous system, changes in, the real cause of roseola, 105
Nervous system, peculiarities of the, in children, 738
Neuralgia, article on, 1026
definition and synonyms, 1026
symptoms, 1027
varieties, 1027
complications, 1037
diagnosis, 1040
prognosis, 1040
pathology and etiology, 1041
treatment, 1042
Neuralgia, in epidemic cerebro-spinal meningitis, 298, 299

Neuralgia—
in gout, 519
diagnosis of pleurisy from, 575
diagnosis of, from hysteria, 643
relation of, to alcoholism, 674, 1042
Neuralgic iritis, 1038
treatment of, 1045
Neuralgic pains in influenza, 42
in locomotor ataxy, 954
Neuritis, acute, 1020
chronic, 1021
Neuroma, idiopathic, 1022
traumatic, 1025
a cause of epilepsy, 1024
Neuromata, syphilitic, 435
Nitric acid, in hooping-cough, 56
in rickets, 495
as a caustic in bites of rabid animals, 200
[Nitrite of amyl in epilepsy, 782]
Nitro-hydrochloric acid in rickets, 495
Nodes, syphilitic, on bones, 433
of cellular tissue, 432
in the muscles, 433
periosteal, in congenital syphilis, 429, 440
treatment of, 457
periosteal swellings, in scurvy, mistaken for, 452
Nomenclature of disease, 17
Nose, discharge from, in glanders, 189, 190
in congenital syphilis, 429, 430
sunken bridge of, a sign of inherited syphilis, 441
Nux vomica, in glanders, 192
in hooping-cough, 56
in rheumatoid arthritis, 557
in rickets, 496
Nyctalopia in scurvy, 453

OBJECTIVE symptoms considered generally, 25
Occipital nerve, the great, neuralgia of, 1032
Occiput, tenderness of, in rickets, 477
Occupation, a predisposing cause of alcoholism, 673
of insanity, 588
of meningitis, 814
of wasting palsy, 786
influence of, on liability to cholera, 390
want of, a cause of hypochondriasis, 626, 629
of hysteria, 632
value of, in treatment of epilepsy, 782
of insanity, 619
of hysteria, 644
of hypochondriasis, 629
Ochlesis, a predisposing cause of typhus fever, 252
Odor, the peculiar, of the breath in alcoholism, 677
Œdema, rare in diphtheria, 67
in dengue, 100
in erysipelas, 323
in purpura, 462
in rickets, 482, 483
Œdema—
in scarlet fever, 92
in smallpox, 129
a sequel of typhoid fever, 209
of joints in gout, 513, 514, 536
over rheumatic joints, a rare occurrence, 561
of lung in influenza, 42
of lung in scurvy, 453, 456
Œdema glottidis, in erysipelas, 327
in scarlet fever, 86, 92
in typhoid fever, 225
after typhus, 262
Œsophagus, affection of, in gout, 519
exudation on, in diphtheria, 75, 76
inflammation of, in hydrophobia, 199
muscular rheumatism in, 574
spasm of, 1058
ulceration of, in tertiary syphilis, 432
ulceration of, in typhoid fever, 216
Oinomania, description of, 681
prognosis, 683
Olecranon, abscess of the bursa over the, in gout, 517
enlargement of, in rickets, 488
Olivary bodies, functions of the, 943
Operations, surgical, a cause of pyæmia, 338
Ophthalmia, complicating dengue, 103
in gout, 520
in relapsing fever, 279
treatment of, 280
in smallpox, 134
treatment of, 144
gonorrhœal, complicating gonorrhœal rheumatism, 576, 577
[Ophthalmia, a sequela of measles, 113]
Ophthalmoscopic appearances in locomotor ataxy, 985
in meningitis, 824
in tumor of the brain, 884
Opisthotonos in cerebro-spinal meningitis, 297, 300
in meningitis, 823
in tetanus, 971
Opium, in cerebro-spinal meningitis, 313
in cholera, 415, 416, 418
in choleraic diarrhœa, 420
in dengue, 104
in diphtheria, 78, 81
in dysentery, 380
in erysipelas, 328
in hooping-cough, 55
in influenza, 46
in intermittent fever, 360
in measles, 115
in pyæmia, 351
in acute rheumatism, 568
in rheumatic pericarditis, 571
in gonorrhœal rheumatism, 579
in rickets, 495
in scurvy, 458
in smallpox, 142
in typhoid fever, 247
Opium—
in typhus, 268
enemata, in chronic dysentery, 383
in typhoid fever, 248
for diarrhœa in diphtheria, 82
external application of, in gout, 540
in muscular rheumatism, 575
Opium, value of, in treatment of chorea, 715
of infantile convulsions, 751
of delirium tremens, 686, 688
of mania, 621
of spinal meningitis, 956
of neuralgia, 1044
of tetanus, 979
Opium-eating, a cause of insanity, 589
Opium-poisoning, diagnosis of, from cerebral hemorrhage, 929
rapid death from, 930
Optic neuritis, relation of, to cerebral hemorrhage, 923
a symptom of meningitis, 824
of cerebral tumor, 846
Optic thalamus, the, arterial circulation through, 909
a frequent seat of cerebral hemorrhage, 903
of cerebral softening, 872
symptoms of hemorrhage into, 913
Orchitis, in mumps, 119
syphilitic, 434
Organic cerebral disease, diagnosis of, from epilepsy, 779
Os calcis, nodes on, in tertiary syphilis, 433
Osseous tumors in the brain, 893
Ossification, abnormalities of, in rickets, 488
Osteo-malakia, diagnosis of, from rickets, 485
Otitis, in influenza, 42
in smallpox, 133
Otorrhœa, in cerebro-spinal meningitis, 300
in measles, 112
after scarlet fever, 90
treatment of, 97
Otorrhœa, or otitis, chronic, a cause of abscess in the brain, 935, 937
of convulsions, 746
of facial palsy, 1051
of meningitis, 813, 831
Over-crowding, a cause of heat apoplexy, 662, 663
Over-eating, a cause of cerebral hemorrhage, 934
of somnambulism, 659
see also Food, excess of.
Over-exertion, a cause of congestion of the brain, 848
of neuralgia, 1027
of paralysis agitans, 725
of wasting palsy, 787, 790
of writer's cramp, 734
Over-work, a cause of convulsions, 757
of epilepsy, 765
of hysteria, 633, 641
of hypochondriasis, 627
of sunstroke, 663, 664

Over-work, a cause—
of vertigo, 694
Oxalates, in urine of diphtheria, 68
of rickets, 484
Oxalic acid, in the blood in gout, 521
free, in the blood, a supposed cause of rickets, 487
in urine of gouty subjects, 520
Oxygen, inhalation of, in cholera, 421
Ozone, deficiency of, in cholera epidemics, 387
its influence in exciting influenza, 37
influence of, on occurrence of typhoid, 238

PACKING, wet, in acute mania, 620
Pain in the head, *see* Headache.
Pain, articular and muscular, in glanders, 189, 190
character of, in cerebro-spinal meningitis, 297
in dengue, 100
in erysipelas, 323
in gout, 512, 514, 536
in influenza, 41
in parotitis, 119
in acute rheumatism, 561
in muscular rheumatism, 574
in back, in epidemic cerebro-spinal meningitis, 297, 299
in intermittent fever, 355, 356
in relapsing fever, 277
in smallpox, 132, 135
in yellow fever, 284
in limbs, in erysipelas, 323
in relapsing fever, 277
in typhoid fever, 202, 204
its effect on appetite and digestion, 121
Pains, in the back, from spinal congestion, 967
from spinal meningitis, 953
character of the, in hypochondriasis, 625
in hysteria, 635
in spinal irritation, 993
the sudden, paroxysmal, of neuroma, 1022, 1023
of locomotor ataxy, 984
of spinal hemorrhage, 1007, 1016
neuralgic, relation of, to inflammation, 947
Palate, ulceration of, in tertiary syphilis, 433
bones of, frequently affected in tertiary syphilis, 433
Palate, soft, condition of, in diphtheria, 65, 75
redness of, in influenza, 41
state of, in measles, 110
state of, in scarlet fever, 86
unilateral paralysis of, in facial palsy, 1052
Pali plague, 316
history and symptoms of, 318
Palpitation of the heart in retrocedent gout, 519
in spinal irritation, 994
Palsy, facial, 1050
metallic, 801
Palsy—
shaking, 718
Pancreas, injection of, a post-mortem appearance in typhus, 265
morbid anatomy of, in cholera, 411
in typhoid fever, 217
in yellow fever, 292
Papillæ of skin in rash of measles, 108
Papules in smallpox, 129, 202
in typhoid, 202, 204, 217
in vaccinia, 159
in varicella, 126
Paralysis, absence of, in some cases of apoplexy, 927
in locomotor ataxy, 984
in spinal irritation, 996
in sunstroke, 667
in spinal meningitis, 953
Paralysis, in cerebro-spinal meningitis, 298, 300
in gout, 519
in pyæmia, 346
facial, a sequel of scarlet fever, 90
diphtheritic, 68, 74
affecting the bladder, 70
heart, 69
muscles of abdomen, 70
of deglutition, 69
of limbs, 69
of respiration, 69
of tongue, 69
nerves of special sense, 69
pathology of, 73
treatment of, 83
local, in relapsing fever, 278
of lower jaw, in hydrophobia, 199
of special nerves from syphilitic nodes, 433, 435, 440
Paralysis agitans, article on, 718
definition, 718
history, 719
varieties and description, 720
causes, 725
diagnosis, 726
complications, 727
pathology and morbid ana- 727
prognosis, 729
treatment, 729
references, 730
Paralysis, a complication of sciatica, 1036
following infantile convulsions, 744
[following scarlet fever, 93]
partial, in chorea, 698
Paralysis, general, of the insane, 605, 999
due to chronic meningitis, 817
or to cerebritis, 856
Paralysis, general spinal, 999
essential, of children, 1004
histrionic, 1050
hysterical, 638, 1000
from cerebral softening, 862, 865
see also Hemiplegia and Paraplegia.
Paralysis, local, from nerve disease, article on, 1048
facial palsy, 1050
of the third cranial nerve, 1054
Paralysis, facial—
of the fifth, sixth, &c., 1055
[Paralysis, spastic spinal, 1009]
Paraplegia, diagnosis of, from muscular anæsthesia, 330
from locomotor ataxy, 963, 989
due to hysteria, 640, 1000
to myelitis, 958
to reflex causes, 1001
incomplete, from spinal congestion, 966
Paroxysm, the epileptic, 770
hysterical, 639
tetanic, 971
Partial mania, 599
Par vagum, paralysis of, in diphtheria, 69
Parotid glands, inflammation of, in cerebro-spinal meningitis, 303
in cholera, 404
in dengue, 102
in influenza, 43
in parotitis, 119
in relapsing fever, 278
inflammation of, in typhus, 261
treatment of, 268
a sequel of typhus, 262
morbid anatomy of, in typhus, 265
suppuration of, in cholera, 404
after scarlet fever, 90
Parotitis, article on, 118
definition, 118
diagnosis, 120
pathology, 119
symptoms, 118
synonyms, 118
treatment, 120
Patella, nodes of, in tertiary syphilis, 433
Pathological anatomy considered generally, 28
Pathology, considered generally, 27
in relation to diagnosis, 27
Pelvis, deformity of, in rickets, 480, 536
Pemphigus, complicating purpura, 460
Penis, gangrene of, an occasional sequel of cholera, 404
in erysipelas, 326
Pepsine, in yellow fever, 294
Perforation of the bowel in typhoid fever, 175, 203, 208
Pericarditis, in cerebro-spinal meningitis, 303
in cholera, 412
in influenza, 43
in pyæmia, 333, 346
in acute rheumatism, 560, 563
treatment of, 570
as a sequela of scarlet fever, 93
absence of tendency to, in acute rheumatoid arthritis, 552
supposed occurrence of, after injection of lactic acid into the peritoneum, 566
Pericardium, effusion into, in acute rheumatism, 560
in scarlatinal dropsy, 92

Pericardium, effusion into—
in scurvy, 457
in yellow fever, 291
extravasation of blood into, in purpura, 463
morbid anatomy of, in pyæmia, 333
white patches on, in rickets, 493
Periodicity, in mania, 597
in epilepsy, 775
in neuralgia, 1029
in somnambulism, 659
of gout, cause of, 535
use of, in diagnosis of gout, 536
impress of, on various diseases, by malaria, 354
Periosteum, inflammation of, in secondary syphilis, 425
in tertiary syphilis, 432
effusion under, in scurvy, 452
gouty deposits under, 516, 525
nodes of, in tertiary syphilis, 426, 432
thickening of, over heads of bones in rickets, 490
Peripneumonia notha, *see* Influenza.
Peritoneum, congestion of, in cholera, 411
effusion into, in the plague, 316
morbid anatomy of, in cholera, 412
morbid anatomy of, in pyæmia, 334
Peritonitis, in cholera, 412
in pyæmia, 334
in acute rheumatism, 560
as a sequel of scarlet fever, 93
in smallpox, 133
in typhoid fever, from perforation of intestines, 203, 208
from suppuration of mesenteric glands, 215
in typhus, 258
tubercular, diagnosis of, from typhoid fever, 245
Perivascular canals in the brain, 850, 909
dilatation of, from chronic congestion, 852
state of, in tubercular meningitis, 834
[Pernicious fever, 359]
Perspiration, *see* Sweat and Sweating.
Petechiæ, in cerebro-spinal meningitis, 299, 302
in diphtheria, 91
in glanders, on serous surfaces, in horses, 184
in malignant measles, 108
in the plague, 314
in pyæmia, 331, 332, 334, 335
in purpura, 460, 463
in relapsing fever, 278
in remittent fever, 368
in scarlet fever, 86
in scurvy, 451
in smallpox, 130, 132
in semi-confluent smallpox, 130
in typhus, 257
in yellow fever, 293
Petit Mal, le, symptoms of, 768
Peyer's patches, enlargement of, in cholera, 411
in scarlet fever, 94
in typhus, 264
fragments of, in stools of typhoid, 244
inflammation of, in pneumonia, 221
in scarlatina, 223
inflammatory products in, in typhoid fever, microscopical appearances of, 212
morbid anatomy of, in diphtheria, 76
in typhoid fever, 209
in yellow fever, 292
tubercular disease of, in pulmonary phthisis, 226, 245
ulceration of, in severe forms of intermittent fever, 229
in pulmonary phthisis, 221
in scurvy, 231
in typhoid fever due to hepatic congestion, 219
Phantom tumor, in hypochondriasis, 627
in hysteria, 643
Pharynx, affection of, in gout, 519
occasionally affected in parotitis, 119
condition of, in diphtheria, 65
in hydrophobia, 199
in measles, 69, 110
in scarlet fever, 86, 88
in syphilis, 424
in typhus, 257
exudation from, in diphtheria, 65, 67, 75
muscular rheumatism in, 574
paralysis of, in diphtheria, 69
smallpox eruption in, 131
spasm of, 1058
spasm of, in hydrophobia, 198
ulceration of, in glanders, 190
in tertiary syphilis, 432
in typhoid fever, 216
Phlebitis, a cause of pyæmia, 338
a sequel of typhus, 262
Phlegmasia dolens after abortion, in typhoid fever, 206
Phlegmon, diagnosis of, from erysipelas, 325
Phlegmonous inflammation in smallpox, 133
Phosphates, deposit of, from the urine, in chorea, 700
excess of, in the urine of acute mania, 597
in urine, in rickets, 477, 484
in scarlet fever, 87, 88
in typhus, 260
Phosphates of iron, quinine, and strychnine, formula for, 364, foot-note
Phosphoretted hydrogen in the air of marshes, 353
Phosphoric acid, in roseola, 106
free in the blood, a supposed cause of rickets, 487
Phosphorus, value of, in treatment of chronic alcoholism, 686
of chorea, 717
Phosphorus, value of, in treatment of locomotor ataxy, 990
of myelitis, 964
of spinal irritation, 998
Phrenic nerve, supposed seat of hooping-cough, 52
Phthisical insanity, 590
Phthisis, connection of, with alcoholism, 683
tendency to, in the insane, 590
a sequel of chronic mercurial poisoning, 804
in measles, 112
relation of gouty diathesis to, 520
tubercular disease of intestines in, 221, 226, 245
ulceration of intestines in, resembling typhoid fever, 226
Physical sign, meaning of the term, 24
Pigeon-breast, in rickets, 479
Pigment, deposits of, in the brain, after chronic congestion, 852
Pigment in urine, in intermittent fever, 358
in remittent fever, 367
in scarlet fever, 87
Pigmentation of viscera in intermittent fever, 359
Piles in gout, 519
as a sequel of dengue, 103
Pins and needles, causes of infantile convulsions, 750
Pitting after smallpox, prevention of, 144
after syphilitic eruptions, 432, 439, 441
after varicella, 126, [136]
Pituitary and pineal bodies, tumors of the, 893
Plague, the, article on, 314
causes of, 317
definition, 314
diagnosis, 315
morbid anatomy, 316
natural history, 317
prophylaxis, 317
symptoms, 314
synonyms, 314
treatment, 317
[Plague, since 1873, 320]
Plaques jaunes, of Cruveilhier, in the brain, 878
Plethora, supposed relation of, to gout, 531
Pleura, effusion into, in the plague, 316
in pyæmia, 332
in rickets, 482
in scarlatinal dropsy, 92, 95
in typhoid fever, 221
effusion of bloody fluid into, in scurvy, 452, 456
morbid anatomy of, in pyæmia, 332
Pleurisy, diagnosis of pleurodynia from, 575
Pleurisy, in cerebro-spinal meningitis, 519
in cholera, 303
in gout, 404
in influenza, 42
in pyæmia, 332
in relapsing fever, 278

Pleurisy—
in rickets, 482
in acute rheumatism, 560
treatment of, 572
a sequela of scarlet fever, 93
in smallpox, 133, 146
treatment of, 143
in hereditary syphilis, 430
in typhoid fever, 222
Pleurodynia, 574
Pneumogastric nerve, paralysis of, in diphtheria, 69
supposed paralysis of, in influenza, 42
supposed seat of hooping-cough, 52
Pneumogastric nerves, affection of, in diphtheria, 69
Pneumonia, complicating delirium tremens, 683
influence of, on prognosis, 682
treatment of, 689
gouty, 518
in cerebro-spinal meningitis, 303
in cholera, 404
in diphtheria, 76
in chronic glanders, 190
in influenza, 42
a sequel of measles, 112
treatment of, 116
in relapsing fever, 278
in scarlet fever, 90, 93
in smallpox, 133
treatment of, 143
in typhoid fever, 221
in typhus, 258, 265
treatment of, 268
idiopathic, diagnosis of, from typhus, 263
diagnosis of pyæmia from, 348
lobular, concealing the commencement of rickets, 476
lobular, in pyæmia, 332
Podophylline, in gout, 540
in intermittent fever, 363
[Polio-myelitis, 1004]
Pons Varolii, symptoms of hemorrhage into, 914
Portal venous system, congestion of, in cholera, 410
Portio dura, paralysis of the, 1050
Port-wine, influence of, in production of gout, 528
Posterior columns of the spinal cord, functions of the, 942
degeneration of, in locomotor ataxy, 982
[Posterior spinal sclerosis in locomotor ataxy, 983]
Potash in gout, acute, 539
chronic, 543
want of, in food, a supposed cause of scurvy, 455
Potash, acetate of, in intermittent fever, 362
in measles, 115
Potash, arsenite of, in rheumatoid arthritis, 557
Potash, bicarbonate of, in intermittent fever, 360, 362
in acute rheumatism, 569, 572
in muscular rheumatism, 575
in rickets, 495
topical use of, in rheumatism, 572
Potash, chlorate of, in diphtheria, 80, 82
in acute rheumatism, 569
in yellow fever, 294
for suppression of urine in influenza, 47
Potash, citrate of, in intermittent fever, 363
in acute rheumatism, 569, 573
in rickets, 497
in smallpox, 141
Potash, nitrate of, in dengue, 104
in influenza, 46
in acute rheumatism, 569
in scurvy, 456
in yellow fever, 294
as an external application in rheumatism, 572
Potash, permanganate of, in diphtheria, 80
Potash, tartrate of, in acute rheumatism, 573
Potassa fusa, as a caustic in bites of rabid animals, 200
Potassæ, liquor, in rickets, 495
Potassium, bromide of, in cerebro-spinal meningitis, 313
in rheumatoid arthritis, 557
see, also, Bromide.
Potassium, iodide of, in cerebro-spinal meningitis, 313
in chronic gout, 542
causing purpura, 463
in rheumatism, 570
in gonorrhœal rheumatism, 579
in muscular rheumatism, 575
in rheumatoid arthritis, 557
in rickets, 496
in scurvy, 458
in syphilis, 437, 438
see, also, Iodide.
Potato, failure of crop followed by scurvy, 446
Poverty, relation of, to alcoholism, 673
Predisposing causes of disease, meaning of the term, 21
Pregnancy, danger of, in smallpox, 137
exemption from scarlet fever, caused by, 94
influence of, in dengue, 102
in relapsing fever, 279
in typhoid fever, 208
in typhus, 261
Pregnancy, insanity of, 593
Pregnancy predisposes to chorea, 710
to torticollis, 1061
Pressure, on nerve trunks, a cause of local paralysis, 1049, 1050
Priapism, in myelitis, 961
in spinal meningitis, 954
Primary fever, stage of, in smallpox, 129, 132
Prognosis, generally considered, 29
Progressive locomotor ataxy, 980
Progressive muscular atrophy, 786
Prophylaxis, of cerebro-spinal meningitis, 312
of cholera, 421
of intermittent fever, 361
Prophylaxis—
of pyæmia, 348
of relapsing fever, 280
in scurvy, 448, 459
of typhoid fever, 250
of typhus, 269
[of yellow fever, 284]
Prostate, abscess of, in pyæmia, 334
Protective influence of vaccination, 154, 155, 166
Prurigo in gouty diathesis, 520
Prussic acid, *see* Hydrocyanic.
[Pseudo-hypertrophic muscular paralysis, 799]
"Psorenterie," 211
Psoriasis in gouty subjects, 520
Psoriasis palmaris in constitutional syphilis, 431
Ptosis, diagnostic value of, 1054
a complication of facial neuralgia, 1038
of locomotor ataxy, 985
Ptyalism, in dengue, 102
in smallpox, 129
Puberty as a cause of renewed susceptibility to smallpox after vaccination, 174
Pubes, deformity of, in rickets, 480
[Puerperal convulsions, 761?]
Puerperal convulsions, relation of, to epilepsy, 766
Puerperal fever, relation of, to pyæmia, 330
Puerperal mania, 590, 598
prognosis of, 617
Puerperal state, a predisposing cause of scarlet fever, 94
Pulmonary apoplexy, complicating cerebral hemorrhage, 904
Pulmonary apoplexy, in diphtheria, 76
in purpura, 463
in pyæmia, 332
Pulmonary arteries, engorgement of, in cholera, 410
theory of contraction of, in cholera, 412
Pulse, in cerebro-spinal meningitis, 298, 302
in cholera, 399, 400
in uræmia in cholera, 403
in delirium tremens, 679
in dengue, 99
in diphtheria, 65, 67, 69, 77
in dysentery, acute, 376
malignant, 377
scorbutic, 377
in epilepsy, 771
in erysipelas, 322, 324, 326
in glanders, 189
in acute gout, 513
in hooping-cough, 50
in hydrophobia, 198
in influenza, 42
in intermittent fever, 357
in mania, 597
in measles, 107
in melancholia, 594
in acute meningitis, 809, 810
in tubercular meningitis, 821
in mercurial poisoning, 803
in myelitis, 961
in malarious neuralgia, 1029
in parotitis, 118, 119
in the plague, 315

Pulse—
in purpura, 462
in pyæmia, 344, 345
in relapsing fever, 277
in remittent fever, 366
in acute rheumatism, 559, 562
in muscular rheumatism, 574
in acute rheumatoid arthritis, 552
in rickets, 476, 482
in roseola, 105
in scarlet fever, 85, 87, 88, 89
in scurvy, 452
in smallpox, 133
in sunstroke, 667
in tetanus, 972
in typhoid fever, 202
in typhus fever, 256, 261, 262
value of, in prognosis in typhus, 266
in varicella, 126
in yellow fever, 284, 285, 288, 292
Pulse, value of the, in diagnosis of cerebral hemorrhage, 922, 930
occasional irregularity of, in children, 830
Pupils, condition of the, in catalepsy, 653
in cerebro-spinal meningitis, 300
in infantile convulsions, 755
in delirium tremens, 679
in epilepsy, 768, 771
in facial neuralgia, 1037
in general paralysis, 605, 606
after cerebral hemorrhage, 921
in hydrophobia, 196
in locomotor ataxy, 986
in acute meningitis, 809, 810
in tubercular meningitis, 822
in opium-poisoning, 929
in scurvy, 453
in softening of the brain, 860
in sunstroke, 667
in tetanus, 975
in typhoid, 246
in typhus, 246, 259
in yellow fever, 293
Purgatives, in cerebro-spinal meningitis, 312
in cholera, 418
in cholera fever, 407
danger of, during cholera epidemics, 389, 422
in delirium tremens, 687
in dengue, 103, 104
in diphtheria, danger of, 81
in erysipelatous meningitis, 329
in gout, acute, 539
chronic, 545
in hooping-cough, 54
in intermittent fever, 360
in acute mania, 621
in measles, 115
ready action of, in measles, 111
in simple meningitis, 815
in tubercular meningitis, 835
in parotitis, 120
in the plague, 317
in purpura, 467
in pyæmia, 350
in remittent fever, 369
in acute rheumatism, 570
Purgatives—
in gonorrhœal rheumatism, 579
in rickets, 495
in scarlet fever, 95
in scarlatinal dropsy, 97
in smallpox, 141
in typhoid, danger of vegetable, 247
in typhus, 268
in yellow fever, 293
Purging in cholera, 399, 402
occasional absence of, in cholera, 406
a means of elimination of cholera-poison, theory of, 413
Purpura, article on, 460
causes, 463
definition, 460
diagnosis, 466
nature of, 460
pathological anatomy of, 463
prognosis, 466
symptoms of, 460
treatment of, 467
varieties of, 461
Purpura, diagnosis of, from scurvy, 454
Purpura hæmorrhagica, 461
papulosa of Hebra, 463
rheumatica, 461
senilis, 461
causes of, 466
simplex, 461
urticans, 462
Purpura, in cerebro-spinal meningitis, 298, 299, 302, 303
in cholera, 415
in diphtheria, 67, 76
in smallpox, 131, 137
Pus in blood, a supposed cause of pyæmia, 339
in joints in rheumatism, 564
in urine in pyæmia, 346
in veins in pyæmia, 335, 339
in vessels in erysipelas, 326
theory of its presence in blood of pyæmia, 336
Pustular eruption about mouth in influenza, 42
Pustules, in congenital syphilis, 441
in glanders, 189
on skin in pyæmia, 335, 344, 345
in smallpox, 129, 130, 146
corymbose, arrangement of, 131
doubtful occurrence of, in alimentary canal, 146
on eye, 134
morbid appearance of, 146
relation to secondary fever, 133
in benign smallpox, 132
Putrid remittent fever, 367
Pyæmia, article on, 330
commencement of, 347
definition, 330
diagnosis, 347
duration, 347
etymology, 330
general pathology, 338
morbid anatomy, 331
mortality, 347
prognosis, 347
symptoms collectively, 344
Pyæmia—
symptoms in relation to various organs, 345
treatment, 348
Pyæmia, a cause of abscess of the brain, 936, 937
Pyæmia, diagnosis of gout from, 536
of rheumatism from, 567
in chronic farcy, 191
after revaccination, 161
in smallpox, 133
a sequel of typhus, 262
relation of, to erysipelas, 330
to puerperal fever, 317
Pyrmont waters, in rheumatoid arthritis, 557
Pyrosis in gout, 518

QUARTAN fever, 355
Quicksilver workers, sanitary condition of, 804
Quinine, in chronic alcoholism, 685
in cerebro-spinal meningitis, 311, 313
after cholera, 419
after dengue, 104
in diphtheria, 78, 81
in malarious dysentery, 382, 384
in erysipelas, 328
in chronic gout, 542
influence of, on excretion of uric acid, 542
in influenza, 46
in intermittent fever, 361
after measles, 116
in neuralgia, 1043
in pyæmia, 350
in purpura following rheumatism, 468
in relapsing fever, 280
in remittent fever, 370
in acute rheumatism, 569
in rheumatoid arthritis, 557
in rickets, 496
in scarlet fever, 96
in smallpox, 142, 143
in typhoid fever, 247
after varicella, 127
in yellow fever, 293
[Quinine in influenza, 46
in typhoid fever, 250
substitutes for, 363]
Quino-alkaline treatment of acute rheumatism, 572
Quotidian fever, 355

RABIES canina, *see* Hydrophobia.
Race, as a predisposing cause of typhus fever, 253, 266
influence of, on susceptibility to smallpox, 138
Rachalgia, *see* Pain in the back.
Rachitis, *see* Rickets.
Radius, deformity of, in rickets, 479
[Rage, in the dog, endangers hydrophobia, 197]
Rain, influence of, on cholera, 387
influence of, on occurrence of typhoid fever, 235, 242
Raptus melancholicus, 594

Rash, in cerebro-spinal meningitis, 298
in dengue, 101
its relation to the course of the disease, 101
occasional in diphtheria, 68, 71
in influenza, 42
in measles, 107, 108
in measles, diagnosis of, 113
in measles, recession of, 109
in malignant measles, 107
in purpura, 460
in relapsing fever, absence of, 277
in roseola, 105
in scarlet fever, 85, 89
in smallpox, 132
in corymbose smallpox, 131
in malignant smallpox, 132
on mucous membrane in smallpox, 131, 137
value of, in prognosis, 136
occasional absence of, in smallpox, 139
in constitutional syphilis, 424
duration of, 424
treatment of, 436, 437
in inherited syphilis, 441
in interval between secondary and tertiary stages of syphilis, 425, 426
treatment of, 437
in typhoid fever, 202, 204, 217
in typhoid fever, absence of, occasionally, 217
in typhus fever, 256
value of, in prognosis, 266
in varicella, 125
Raving madness, 596
Raw meat for diarrhœa, after measles, 117
Reaction, stage of, in cholera, 401
complications of, 401
imperfect, 402
in cholera, physiology of, 414
Reactionary fever in cholera, 402
Receptaculum chyli, emptiness of, in typhoid fever, 215
Recession of ribs in inspiration in rickets, 479
Rectum, affection of, in gout, 519
stricture of, from syphilitic ulceration, 432
ulceration of, in tertiary syphilis, 432
Recurrence, of cholera, in the same individual, 392
of diphtheria, 64
of gout, 513, 536, 538
of parotitis, 120
of relapsing fever, 275
of rheumatism, 561
of muscular rheumatism, 574
of gonorrhœal rheumatism, 576, 577
of roseola, 106
in scarlet fever, 84
of smallpox, 139
of syphilis, 426, 427
of typhus fever, 426
of varicella, unknown, 125
of yellow fever, 286
Recurrent mania, 598
Red softening of the brain, 874
Reflex action, diminished in diphtheria, 70
Reflex nervous irritability, increase of, from injury of the spinal cord, 945
in hysteria, 638
in tetanus, 972
absence of, in myelitis, 960
no excess of, in spinal congestion, 967
in spinal meningitis, 954
Reflex nervous irritation, a cause of chorea, 710
of insanity, 612
of paralysis agitans, 724
of paraplegia, 1001
Regimen, *see* Diet.
Regiments, marching, liability of, to cholera, 389
Relapses, in cerebro-spinal meningitis, 304
in cholera, 402
in dengue, 101, 102
in diphtheria, 64
in erysipelas, 324
in measles, 106
in relapsing fever, 277
in scarlet fever, 84
in tertiary syphilis, 431
in typhoid fever, 203
rare in typhus, 261
Relapsing fever, article on, 269
bibliography of, 269
definition, 269
etiology, 274
geographical distribution, 273
history, 269
nomenclature, 269
symptoms, 277
synonyms, 273
therapeutics, 280
Relapsing fever, non-identity of, with typhus, 276
occurring in epidemics, with typhus, 276
[Relapsing fever in Philadelphia, 274, 275, 277, 279]
Remissions, in cerebro-spinal meningitis, 311
in erysipelas, 325
in simple meningitis, 813
in tubercular meningitis, 825
in remittent fever, 365, 366
in pyrexia of typhoid, 228
of pyrexia in typhus fever, 255
in yellow fever, 285
Remittent fever, article on, 365
causes, 366
definition, 365
diagnosis, 368
history, 365
modes of commencement, 365
pathology, 368
prognosis, 369
symptoms, 366
synonyms, 365
treatment, 369
Remittent fever, diagnosis of dengue from, 103
diagnosis of, from yellow fever, 286
relation of, to typhoid fever, 228
Remittent fever, infantile, complicating hooping-cough, 51
Remittent fever, infantile—treatment of, 57
rickets mistaken for, 475
Renal calculi, diagnosis of pain from, from lumbago, 575
Residence, change of, a predisposing cause of typhoid fever, 238
Respiration, character of the, in catalepsy, 653
after cerebral hemorrhage, 922
in cerebro-spinal meningitis, 298, 302
in cholera, 400
in diphtheria, 65, 67
in diphtheritic paralysis, 69
in hooping-cough, 50
in intermittent fever, 357
in measles, 107
in tubercular meningitis, 821
in parotitis, 118
in pyæmia, 344, 345
in scarlet fever, 87
in scurvy, 452
in sunstroke, 666
in typhoid fever, 203
in typhus, 258
Respiration, difficulty of, *see* Dyspnœa.
Rest, importance of, in treatment of chorea, 716
of hysteria, 644
of spinal meningitis, 956
of writer's cramp, 735, 737
Restraint, in treatment of delirium tremens, 689
of acute mania, 619
Retarded cow-pox, 160
Rete mucosum, changes of, in smallpox, 147
Retention of urine, in dysentery, 376
in meningitis, 823
in myelitis, 960
a rare symptom in smallpox, 141
in typhoid fever, 204, 208
in typhus, 261
Retina, congestion of the, in meningitis, 824
degeneration of, in Bright's disease, 923
Retinitis, in congenital syphilis, 441
in secondary stage of syphilis, 425
treatment of, 436
influence of mercury on, 437
Retraction of the abdominal walls in meningitis, 810, 820
Retrocedent gout, 518
Revaccination, 155
indications for, 174
necessity for, 173
phenomena of, 161
protective power of, 175
time for, 174
Rheumatic gout, *see* Rheumatoid arthritis.
Rheumatic iritis, 1038
treatment of, 1045
Rheumatic meningitis, 811
neuralgia, 1044
Rheumatism, article on, 558
articular rheumatism, causes of, 564
definition, 559
description of acute, 559

Rheumatism—
subacute, 560
diagnosis of, 566
history, 559
morbid anatomy, 563
pathology, 565
prognosis, 567
symptoms, considered specially, 561
synonyms, 559
treatment, 567
muscular rheumatism, causes of, 573
definition, 573
description, 573
diagnosis, 575
history, 573
pathology, 574
prognosis, 575
synonyms, 573
treatment, 575
Rheumatism, a cause of embolism of the brain, 859
of insanity, 591
of local paralysis, 1049, 1051
of sciatica, 1037
of spinal meningitis, 955
connection of, with chorea, 699, 710
predisposes to neuritis, 1021
Rheumatism, acute, an exciting cause of rheumatoid arthritis, 554
complicating scarlet fever, 91, 93
treatment of, 97
diagnosis of, from pyæmia, 348
diagnosis of, from rheumatoid arthritis, 555
purpura during convalescence from, 461
Rheumatism, chronic, diagnosis of, from rheumatoid arthritis, 555
diagnosis of, from gonorrhœal rheumatism, 577
resemblance of pains in early stage of scurvy to, 451, 454
Rheumatism, gonorrhœal, diagnosis of, from rheumatoid arthritis, 556
Rheumatism, muscular, a distinct disease from articular, 574
Rheumatismus metallicus, 801
Rheumatoid arthritis, article on, 550
causes, 554
definition, 550
description of acute, 552
of chronic, 551
diagnosis, 555
diet and regimen, 858
history, 550
morbid anatomy, 553
pathology, 554
prognosis, 556
synonyms, 550
treatment, 556
Rheumatoid arthritis, confounded with chronic rheumatism, 561
diagnosis of rheumatism from, 566
Rhonchi, character of, in broncho-pneumonia of measles, 112
in influenza, 42
Rhonchi, character of—
in measles, 107
in typhus, 258
in lung affection of scurvy, 453
Rhythmical movements, use of, in treatment of chorea, &c., 649
Ribs, deformity of, in rickets, 479, 481
Rice-water stools in cholera, chemical and microscopical characters of, 404
Rickets, article on, 472
causes, 473
definition, 472
diagnosis, 472
morbid anatomy, 487
prognosis, 494
symptoms, 475
synonyms, 473
therapeutics, 495
Rickets, a cause of convulsions in children, 747
of hypertrophy of the brain, 899
of laryngismus stridulus, 742
connection of, with chronic hydrocephalus, 839
[Rickets, infrequency of, in the United States, 474]
Rigidity, muscular, distinction between the "early" and "late" varieties of Todd, 960
occurs in cerebral softening, 927, 929
in myelitis, 960
in spinal meningitis, 953
in tetanus, 971
in tubercular meningitis, 820, 823
Rigor mortis in scurvy, 456
Rigor mortis, rapid occurrence of, after tetanus, 970, 976
Rigors, in cerebro-spinal meningitis, 297, 298
in dengue, 99
in diphtheria, 65
in dysentery, acute, 376
in erysipelas, 322
in chronic farcy, 190
in glanders, 189
in acute gout, 513
in influenza, 41
in intermittent fever, 357
in measles, 106
in parotitis, 118
in the plague, 314
in pyæmia, 344, 345
a characteristic symptom of pyæmia, 348
in relapsing fever, 277
in remittent fever, 366
in acute rheumatism, 559
in scarlet fever, absent, 85
in scurvy, 452
in smallpox, 132
in typhoid fever, 204
in typhus fever, 254, 258
in yellow fever, 284
Risus sardonicus vel caninus, 1059
in tetanus, 971
Roseola, article on, 104
cause, 105
course, 105
definition, 104
diagnosis, 106
Roseola—
prognosis, 106
treatment, 106
diagnosis of, from flea bites, 106
from measles, 106
from scarlet fever, 94, 106
Roseola, in cerebro-spinal meningitis, 298, 303
in smallpox, 132
after vaccination, 159
Roseola, syphilitic, diagnosis of, from measles, 113
resemblance to rash of measles, 113
Rossalia, an old name for scarlet fever, 83
[Rötheln, article on, 117
diagnosis of, 117
symptoms, 117
treatment, 117]
Rubeola, *see* Measles.
Rupia syphilitica, 425, footnote
Russian epidemic, identity of, with relapsing fever, 273
mortality of, 280

SACRUM, deformity of, in rickets, 480
displacement of, in rickets, 493
St. Guy, the dance of, 696
St. John, the dance of, 647, 701
St. Vitus, the dance of, 696
origin of, 647
relation of, to paralysis agitans, 719
[Salicylic acid in rheumatism, 570
in typhoid fever, 250]
Salines in chronic gout, 543
Saline, injections into veins in cholera, 421
treatment of acute rheumatism, 569
treatment of cholera, 421
Saliva, diminished flow of, occasionally in parotitis, 119
Salivary glands, enlargement of, in hydrophobia, 199
treatment of, 268
inflammation of, a sequel of typhus, 262, 265
swelling of, in dengue, 102
unaffected in plague buboes, 317
Salivation, in dengue, 102
occasionally in parotitis, 119
in smallpox, 129
in scurvy, 453
Salt meat, a cause of purpura, 463
influence of, in the production of scurvy, 448, 449
Salts, injection of, into veins in cholera, 421
Salts of blood, transudation of, in cholera, 414
Sarcocele, syphilitic, 434
Sarsaparilla in syphilis, 437
[Scab, for vaccination, 163]
Scabies often coincident with syphilitic eruptions, 439
Scalp, suppuration under, in smallpox, 142
Scammony, in dengue, 104
in remittent fever, 369

Scapulæ, deformity of, in rickets, 480
Scapulodynia, 574
[Scarlatina, popular error concerning, 89]
see Scarlet fever.
Scarlatinal dropsy, treatment of, 97
Scarlet fever, article on, 83
causes, 84
definition, 83
diagnosis, 93
history, 83
morbid anatomy, 94
prognosis, 94
prophylaxis, 95
sequelæ, 89
symptoms of latent, 89
symptoms of malignant, 88
symptoms of ordinary, 84
treatment, 95
Scarlet fever, hemorrhagic, 93
surgical, 94
Sydenham's, 89
Scarlet fever, occasional association of, with diphtheria, 71
a cause of purpura, 463
confounded with diphtheria, 86
complicated with rheumatism, 565
complicating vaccination, 160
diagnosis of dengue from, 103
diagnosis of, from diphtheria, 70
diagnosis of, from measles, 113
diagnosis of roseola from, 106
diagnosis of, from roseola in smallpox, 132
mistaken for rheumatic fever, 94
rash mistaken for typhus, 94
relation of, to diphtheria, 58, 61, 64, 70
relation of, to typhoid fever, 223
Schwalbach waters in rheumatoid arthritis, 557
Sciatica, 1035
Sciatica in gout, 519
Sciatica neuritis, 1021
[Sclerosis, lateral, 1009
multiple, 1011
posterior, 983
recent study of, 1009
treatment of, 1014]
Sclerosis of the brain, in idiocy, 603
in insanity, 615
of the spinal cord, 1008
in locomotor ataxy, 982
see also Induration.
Sclerotic coat of eye, deposits of urate of soda in, 517, 520
Sclerotitis in gout, 520
in rheumatoid arthritis, 552
Scorbutus, or scurvy, article on, 445
definition, 445
diagnosis, 454
etiology, 445
morbid anatomy, 456
pathology, 455
prognosis, 457
symptoms, 451
Scorbutus, or scurvy—
synonyms, 445
therapeutics, 458
Scorbutus, as a cause of cholera, 390
complicating dysentery, 372, 373, 377
complicating remittent fever, 367
diagnosis of, from purpura, 467
outbreak of, among the troops in the Crimea, 447
relation of, to typhoid fever, 231
Scrivener's palsy, 732
[Scrofula, article on, 497
anatomy, 503
causation, 507
history, 497
pathology, 503
symptomatology, 498
treatment, 509]
Scrofula, a cause of caries of the vertebræ, 1017
communicated by vaccination, 177
effect of, on course of syphilis, 426
Scrofulosis, diagnosis of, from rickets, 485
supposed relation to rickets, 484, 485
Scrotum, gangrene of, a sequel of cholera, 404
after smallpox, 134
[Scurvy, best preventives of, 459]
see Scorbutus.
Season, influence of, on cerebral-spinal meningitis, 308
on cholera, 386
on diphtheria, 62
on gout, 530
on hooping-cough, 48
on influenza, 36
on pulmonary complications in measles, 111
on parotitis, 120
on the plague, 319
on pyæmia, 344
on relapsing fever, 274
on rheumatism, 565
on scarlet fever, 84
on epidemics of smallpox, 156
on typhoid fever, 235
on typhus fever, 253
on vaccination, 160
Secondary fever in smallpox, 129
Secondary syphilis, *see* Syphilis.
Secretion suppressed in cholera, 400
Secunderabad barracks, dysentery at, 374
Sedatives, in treatment of epilepsy, 780
see also Opium.
Sedentary occupations, a cause of sciatica, 1035, 1036
Semiconfluent smallpox, 128, 130
Semilunar ganglia, supposed inflammation of, in yellow fever, 291
Senega, in diphtheria, 82
in influenza, 46
in long complications of typhus, 268
Senile vertigo, 693
treatment of, 695
Sensation, condition of, in chronic alcoholism, 678
in chorea, 698
in congestion of the brain, 845
in epileptics, 774
in general paralysis, 606
in hysteria, 635
in infantile paralysis, 1004
in locomotor ataxy, 984
melancholia attonita, 594
in meningitis, 823
in cerebral softening, 859
in tumors of the brain, 884
in spinal congestion, 966
in spinal meningitis, 954
in wasting palsy, 789
Sensation, loss of, *see* Anæsthesia.
Senses, special, paralysis of, in diphtheria, 69
state of, in congestion of the brain, 845, 846
in idiocy, 604
in locomotor ataxy, 985
in cerebral softening, 859
in vertigo, 690
Sensibility, loss of, in diphtheria, 69
Septicæmia, relation of, to pyæmia, 330
Septum narium, perforation of, in chronic glanders, 190
Sequelæ, of convulsions, 744
of epilepsy, 772
of sunstroke, 669
Serous apoplexy, 930
Serpentaria in influenza, 46
Serum of blood, changes of, in gout, 521
changes of, in acute rheumatism, 562
character of, in yellow fever, 288
effusion of, in rickets, 482
Sewage in drinking water as a cause of cholera, 389
Sewage gases, as a cause of cholera, 388
a cause of typhoid fever, 241
[Sewer gas promotive of typhoid fever, 241, 251]
Sex, considered generally as a cause of disease, 22
influence of, on mortality in diphtheria, 61
influence of, on the movements of respiration, 946
influence of, on occurrence of cerebro spinal meningitis, 308
of cholera, 390
of chicken-pox, 125
of erysipelas, 322
of gout, 527, 536
of hooping-cough, 49
of influenza, 40
of parotitis, 120
of pyæmia, 344
of rabies in dogs, 196
of relapsing fever, 274
of rheumatism, 564
of gonorrhœal rheumatism, 577
of chronic rheumatoid arthritis, 551, 554, 555
of rickets, 475

Sex, influence of, on occurrence of scarlet fever, 84, 95
of cellular nodes in tertiary syphilis, 432
of typhus fever, 252
influence of, on prognosis of chronic alcoholism, 682
of epilepsy, 774, 779
of insanity, 618
Sex, as a predisposing cause of congestion of the brain, 848
of chorea, 697
of epilepsy, 764
of general paralysis, 605
of hypochondriasis, 626
of hysteria, 631
of insanity, 587
of laryngismus stridulus, 741
of locomotor ataxy, 988
of simple meningitis, 814
of tubercular meningitis, 818
of paralysis agitans, 725
of wasting palsy, 786
of writer's cramp, 734
Sexual development, arrest of, in hereditary syphilis, 436
Sexual diseases, a cause of alcoholism, 674
Sexual disorders, of women, a cause of epilepsy, 767
of hysteria, 632, 639
of insanity, 590
Sexual excess, a cause of dementia, 600
of epilepsy, 766
of hysteria, 632
of insanity, 589, 602
of locomotor ataxy, 989
of myelitis, 963
of paralysis agitans, 725
of sciatica, 1036
of spinal irritation, 997
of wasting palsy, 788
Sexual organs of women, involution of the, a cause of insanity, 587
of neuralgia, 1031
Sexual power, loss of, in idiocy, 604
in general paralysis, 606
state of, in locomotor ataxy, 986
Shaking palsy, 718
Sheep-pox, 128
inoculation of man from, results of, 128
Sherry, influence of, in production of gout, 528
Shock, a predisposing cause of pyæmia, 343
Shock, nervous, a factor in the apoplectic condition, 921
a cause of insanity, 589
of neuralgia, 1028
see also Fright and Emotion.
Sick-headache, description of, 1031
Sight affected in diphtheria, 69, 74
in typhus, 259
Sight, defects of, premonitory of cerebral hemorrhage, 923
from cerebritis, 855
in chronic alcoholism, 676
in meningitis, 810
from neuralgia, 1037
Sight—
loss of, in locomotor ataxy, 985
after infantile convulsions, 744
in chronic hydrocephalus, 838
Signs of disease considered generally, 23
Silver, deposits of, in the brain, 899
Silver, nitrate of, in typhoid fever, 248
topical use of, in bites of rabid animals, 200
in diphtheria, 78
in erysipelas, 329
in hooping-cough, 55
in scarlet fever, 96
in smallpox, to prevent pitting, 144
in scurvy, to gums, 458
in variolous ophthalmia, 144
Singultus, 1058
Sinus of the brain, thrombosis of, 870
Skin, condition of the, in mania, 597
in mercurial poisoning, 803
in sunstroke, 666
Skin, diseases of the, due to chronic alcoholism, 677
Skin, hemorrhage into, in purpura, 464
question of elimination of uric acid by, in gout, 523
state of, in cerebro-spinal meningitis, 298, 302
in dengue, 99
in diphtheria, exudation on, 68
in dysentery, acute, 376
malignant, 377
mild, 375
in erysipelas, 323, 324, 326
in farcy, 190
in gout, 514, 520
in hooping-cough, 50
in hydrophobia, 198
in influenza, 42
in intermittent fever, 356, 358
in measles, 107
in the plague, 314
in purpura, 460
in pyæmia, 344
in acute rheumatism, 559
in gonorrhœal rheumatism, 576
in muscular rheumatism, 574
in rickets, 476
in relapsing fever, 277, 278
in remittent fever, 366
in scarlet fever, 85, 87, 89
in scurvy, 451
in smallpox, 146
in secondary syphilis, 424
in tertiary syphilis, 431
in typhoid fever, 202, 217
in typhus fever, 256
in yellow fever, 285
yellow tint of, in yellow fever, 285, 286, 290, 292
Skull, bones of, thickening of, in syphilis, 440
Skull, deformities of the, in idiocy, 603
in chronic hydrocephalus, 837, 838
Skull, fracture of the, a cause of abscess of the brain, 934
diagnosis of, from apoplexy, 929
[Skunk, subject to rabies, 197]
Sleep, effect of, on prognosis of delirium tremens, 682
value of, in acute mania, 621
Sleeplessness, *see* Insomnia.
Sleep-walking, 658
Sloughing in erysipelas, 324
Smallpox, article on, 127
after vaccination, 149
anatomical characters of a variolous pock, 146
definition, 127
description, 124
diagnosis, 135
epidemic diffusion of, 156
history of, 127
infectious nature of, 138
inoculation for, 156
morbid appearances of, 146
mortality, 145
prognosis, 136
susceptibility to, 137
symptoms, 132
synonyms, 127
treatment of, 140
varieties of, 129
Smallpox, a cause of purpura, 463
complicating scarlet fever, 93
diagnosis of, from continued fever, 136
diagnosis of, from febrile lichen, 15
diagnosis of, from measles, 135
diagnosis of, from scarlatina, 132
diagnosis of, from varicella, 136
non-identity of, with chickenpox, 124
theory of hereditary immunity from, 427, 428
Smell, sense of, affected rarely in cerebro-spinal meningitis, 301
Snuffles in hereditary syphilis, 429, 441
Soda in gout, acute, 539
chronic, 543, 557
Soda, arseniate of, in rheumatoid arthritis, 557
Soda, bicarbonate of, in reactionary stage of cholera, 418
Soda, phosphate of, in gout, 544
Soda, sulphate of, in purpura, 467
Soda, tartrate of, in smallpox, 141
Softening, of bones, in rickets, 478, 487
of brain substance in pyæmia, 334
an occasional sequel of typhus, 259
Softening of the brain, acute, 857
chronic, 874
post-mortem, 879
red, 864
white, 873
yellow, 875
predisposes to cerebral hemorrhage, 907

Softening of the brain—
secondary to abscess, 939
Softening of the spinal cord, 1008
Soil, nature of, influence of, on occurrence of cerebro-spinal meningitis, 309
of cholera, 388
of influenza, 36
of the plague, 318
Solitary glands, enlargement of, in cholera, 411
enlargement of, in dysentery, 378
ulceration of, in dysentery, 379
inflammation of, in pneumonia, 221
inflammation of, in scarlet fever, 223
morbid anatomy of, in typhoid fever, 209, 211
in typhus, 265
swelling of, in scarlet fever, 94
tubercular disease of, in pulmonary phthisis, 221
ulceration of, in severe forms of intermittent fever, 228, 229
ulceration of, in scurvy, 231, 457
Somnambulism, article on, 658
varieties and symptoms, 659
treatment, 660
Sordes on lips, distinction of, from diphtheritic exudation, 72
on teeth and gums in diphtheria, 66, 72
in erysipelas, 324
in measles, 107, 108
in malignant measles, 108
in pyæmia, 344
in relapsing fever, 278
in remittent fever, 368
in typhoid fever, 203
in typhus fever, 255, 257
in yellow fever, 287
Sore throat, in dengue, 102
in diphtheria, 65
herpetic, diagnosis of, from diphtheria, 107
in erysipelas, 322
in chronic glanders, 190
in measles, 107
in scarlet fever, 85, 86, 89, 90
treatment of, 96
as a sequel of scarlet fever, 90
treatment of, 96
in smallpox, 131
treatment of, 141
in secondary syphilis, 424
in tertiary syphilis, 430
in typhoid, 203, 223
Spa waters in rheumatoid arthritis, 557
Spasm, definition of, 1055
relation of, to spinal congestion, 950
Spasm, of dorsal muscles, in cerebro-spinal meningitis, 297, 299
Spasm, muscular, complicating epilepsy, 774
facial neuralgia, 1038
paralysis agitans, 721
sciatica, 1037
Spasm—
in cerebral softening, 865
in spinal irritation, 995
in tetanus, 971
Spasmodic asthma, causes of, 1056
Spasmodic cholera, 407
Spasms, local, article on, 1055
asthma or bronchial spasm, 1056
cardiac spasm, 1057
hiccup, 1058
muscular cramp, 1059 *et seq.*
treatment, 1060
[Spastic spinal paralysis, 1009]
Specific gravity of the brain, in insanity, 615
Speech affected in parotitis, 119
changes in, from abscess of the brain, 938
from cerebral congestion, 845
from general paralysis, 605
from mercurial poisoning, 802
from softening of the brain, 858, 860
in wasting palsy, 789
defective, in epilepsy, 753
a premonitory sign of cerebral hemorrhage, 924
Speech, loss of, *see* Aphasia.
Spermatorrhœa, treatment of, 660
Sphincter ani, paralysis of, in chronic dysentery, 376
Sphincters, paralysis of the, in myelitis, 960
in spinal meningitis, 954
partial, in locomotor ataxy, 985
absence of, in spinal congestion, 966
in spinal irritation, 996
Sphygmograph, use of the, in alcoholism, 672, 679
an aid in prognosis, 682
in epilepsy, 772
Spina bifida, 1018
Spinal congestion, article on, 965
symptoms, 965
post-mortem appearances, 968
diagnosis, 968
prognosis, 968
treatment, 968
Spinal congestion, diagnosis of, from reflex paraplegia, 1002
relation of, to infantile paralysis, 1004
Spinal cord, affection of, in gout, 519
atrophy of, 1015
concussion of, 1016
congestion of, 965
congestion of, in yellow fever, 291
hemorrhage into, 1007
hypertrophy of, 1015
induration of, 1008
irritation of, 991
local softening of, in cerebro-spinal meningitis, 305
œdema of, in cerebro-spinal meningitis, 305
physiology of, 944
Spinal cord—
softening of, 1008
tumors, 1016
Spinal induration, 1008
a result of myelitis, 962
Spinal irritation, article on, 991
symptoms, 991
post-mortem appearances, 997
causes, 997
diagnosis, 997
prognosis and treatment, 998
Spinal nerves, physiology of the, 942
Spinal paralysis, general, symptoms of, 999
treatment, 1000
Spinal tenderness, localized, a symptom of hysteria, 635
of spinal irritation, 993, 994
absence of, in myelitis, 960
in spinal congestion, 967
in spinal meningitis, 954
Spine, deformity of, in rickets, 477, 478, 491
Spirits, distilled, a very slight cause of gout, 527, 528
Spleen, albuminoid degeneration of, in rickets, 483
disease of the, 623
enlargement of, in intermittent fever, 358
treatment of, 364
in remittent fever, 368
in typhoid fever, 204
morbid anatomy of, in cholera, 411
in diphtheria, 72, 76
in erysipelas, 326
in influenza, 43
in intermittent fever, 359
in relapsing fever, 279
in the plague, 316
in purpura, 463
in pyæmia, 333
in rickets, 493
in scarlet fever, 94
in scurvy, 457
in typhoid fever, 215
in typhus, 265
in yellow fever, 292
tenderness of, in relapsing fever, 277
in typhoid fever, 204
Splenization of the lung in typhoid fever, 221
Splints in gonorrhœal rheumatism, 579
Sponging, cold, in parotitis, 121
tepid, in measles, 114
for malaria, 124
in parotitis, 121
in remittent fever, 369
in typhus, 269
Spurious cow-pox, 161
Sputa, in hooping-cough, 50
in influenza, 42
in measles, 111
in pyæmia, 345
in scurvy, 452
in typhoid fever, 202
in typhus, 258
Squills, in influenza, 46
in yellow fever, 294
Stammering in mercurial poisoning, 801, 802
relation of, to chorea, 704

Staphyloma, a rare sequel of cholera, 404
Starvation, *see* Food, deficiency of.
Status epilepticus, 754
Steam, use of, in diphtheria, 83
in influenza, 45
in scarlet fever, 96
Steel wine in rickets, 496
Stenosis, *see* Stricture.
Sterno-mastoid a frequent seat of muscular nodes in syphilis, 433
Stertorous breathing, in sunstroke, 667
value of, in prognosis of cerebral hemorrhage, 922
Stiffness of joints, after dengue, 103
in gout, 516, 525
in scurvy, 452
in rheumatoid arthritis, 551
Stiffness, muscular, *see* Rigidity.
Stigmata in purpura, 460
Stimulants, abuse of, in hysteria, 639, 674
in neuralgia, 674, 1046
excess of, a cause of congestion of the brain, 848
predispose to sunstroke, 662
value of, in treatment of chorea, 715
of delirium tremens, 687
of infantile convulsions, 751
see also Alcohol.
Stomach, affection of, by gouty metastasis, 519
disorders of the, causing vertigo, 691
treatment of, 695
disturbance of, by malaria, 354
hemorrhage from, *see* Hæmatemesis.
irritability of, in intermittent fever, 355
in remittent fever, 365
morbid anatomy of, in cholera, 411
in diphtheria, 76
in hydrophobia, 199
in intermittent fever, 358
in the plague, 316
in scurvy, 457
in typhoid fever, 216
in yellow fever, 291
muscular rheumatism in, 574
pain at the pit of, in tetanus, 977
Stomatitis, in hereditary syphilis, 429
gangrenous, after measles, 112
treatment of, 115
Stone-pock, 132
Stools, in dengue, 100
in cholera, 399, 400, 401
chemical examination of, 404
microscopical examination of, 404
in cholera as the medium of contagion, 395
in cholera, removal of, 422
in cholera, retention of, 413
in diarrhœa, choleraic, 407
in dysentery, acute, 375, 376
chronic, 376
malarious, 377
Stools, in dysentery—
malignant, 377
mild, 375
scorbutic, 377
in glanders, 189
in gout, 515
under influence of colchicum, 539
in measles, 111
in the plague, 315
in pyæmia, 344, 346
in remittent fever, 367
in rickets, 476, 482, 484
in scurvy, 453
in typhoid fever, 203, 204, 207
microscopical examination of, in typhoid, 244
as a means of communicating typhoid fever, 243
in typhus, 258, 263
in yellow fever, 285, 293
rice-water, 399
Strabismus, causes of, 1059
in cerebro-spinal meningitis, 300
in diphtheria, 69
in chronic hydrocephalus, 837
in locomotor ataxy, 987
in meningitis, 810, 822
occurs after fits in children, 744
in softening of the brain, 860
Strain of muscles, a cause of muscular rheumatism, 574
Structural disease as contrasted with functional, 19
Strumous disease of the vertebræ, diagnosis of, from spinal irritation, 997
Strychnia, after cholera, 419
[in diphtheritic paralysis, 83]
in glanders in the horse, 192
in hooping-cough, 56
after intermittent fever, 364
in rheumatoid arthritis, 557
poisoning by, diagnosis of, from tetanus, 978
value of, in treatment of alcoholism, 686
of chorea, 713
of myelitis, 964
Subjective symptoms considered generally, 24
Sublingual gland, inflammation of, after typhus, 262
Submaxillary gland, suppuration after typhus, 262
swelling of, in parotitis, 119
Submaxillary lymphatic glands, enlargement of, in diphtheria, 66
Subsultus tendinum, in typhus, 259
treatment of, 268
Suckling, prolonged, a cause of rickets, 474
a predisposing cause of rheumatism, 565
want of, a cause of rickets, 474
Sudamina and miliaria, article on, 122
Sudamina, causes of, 122
general pathology of, 122
in influenza, 42
in pyæmia, 335, 344, 345
in relapsing fever, 278
Sudamina—
in typhoid fever, 203, 218
Sugar, to be avoided in neuralgia, 1046
Sugar in the urine in typhus, 260
increased production of, in the liver of drunkards, 684
Suicide, tendency to, in abscess of the brain, 938
in chronic alcoholism, 678
in melancholia, 592, 594
in tertiary syphilis, 433
Sulphates in urine in acute rheumatism, 563
in typhus, 260
Sulphites, use of, in influenza, 47
in pyæmia, 351
Sulphur, in muscular rheumatism, 575
Sulphur baths, in treatment of mercurial tremor, 806
of lead-poisoning, 807
Sulphuretted hydrogen in the air of marshes, 353
Sulphuric acid, in choleraic diarrhœa, 420
in hooping-cough, 56
in typhoid fever, 248
Sulphurous acid, in diphtheria, 80, 82
suggested use of, in influenza, 47
supposed efficacy of, in pyæmia, 351
[Sunshine, curative of neuralgia, 1048]
Sunstroke, article on, 661
definition, 661
synonyms and history, 661
etiology, 664
[frequency of, in cities, 664
promoted by intemperance, 665]
symptoms, 666
diagnosis, 667
pathology, 667
morbid anatomy, 668
mortality, 668
prognosis and prophylaxis, 668
treatment, 669
[by hypodermic injection of morphia, 670]
of sequelæ, 670
Suppression of urine, in influenza, 43
in malignant dysentery, 377
in smallpox, 141
in yellow fever, 290, 293
Suppuration, from chalk-stones in gout, 517
in erysipelas, 323, 326
in pyæmia, 331
of bones and joints in pyæmia, 334
of lymphatic glands in tertiary syphilis, 433
of parotid gland in parotitis, 119
of parotid glands after typhus, 262
of periosteal nodes in tertiary syphilis, 433
see, also, Abscess.
Supra-renal capsules, congestion of, in diphtheria, 77
Surgical operations a cause of pyæmia, 330, 343

Surgical scarlet fever, 94
Sutures, deepening of, in rickety skull, 491
Sweat, characters of, in acute rheumatism, 562
nature of, in rheumatoid arthritis, 553
Sweating, in cholera, 400, 406, 415
in intermittent fever, 357
in pyæmia, 344, 345
in relapsing fever, 277
in remittent fever, 366
in acute rheumatism, 559
absence of, in acute rheumatoid arthritis, 552
at night, in chronic dysentery, 377
critical, in yellow fever, 285
excessive, of no use in gout, 523
of head, in rickets, 476, 482
relation of, to sudamina, 123
Symmetry, in symptoms of acute rheumatism, 561
of congenital syphilis, 430
of secondary syphilis, 424, 425
of tertiary congenital syphilis, 440
Sympathetic epilepsy, 1024
Sympathetic nervous system, influence of, on occurrence of purpura, 465, 466
supposed affection of, in typhoid fever, 220
Symptoms of disease considered generally, 23
general and local, 24
objective and subjective, 24
Syncope, a cause of death in diphtheria, 66, 69
in gouty affection of the heart, 519
in the plague, 315
in purpura, 461
in relapsing fever, 279
in scurvy, 452, 455
Syncope, diagnosis of, from epilepsy, 778
from congestive apoplexy, 849
in hysteria, 635
Synovial fluid, effusion of, in gout, 525
in acute rheumatism, 561, 563
in rheumatoid arthritis, 553
Synovial membrane, inflammation of, in gout, 514
in acute rheumatism, 561, 563
in rheumatoid arthritis, 553
Syphilis, article on, 423
diagnosis of, 438
diagnosis of inherited syphilis, 440
stages of, 424
modes of communication, 429
tertiary symptoms, or sequeiæ, 431
treatment, 435
Syphilis, a cause of abscess of the brain, 936
of infantile convulsions, 746
of insanity, 590
of local paralysis, 1050
of spinal meningitis, 955
Syphilis, a cause—
of neuralgia, 1043
of paralysis agitans, 725
of purpura, 463
of sciatica, 1037
of wasting palsy, 788
Syphilis, a specific fever, 423
communicated by vaccination, 178
communication of, by fœtus to mother, 429
diagnosis of pains in, from muscular rheumatism, 575
in parents, a supposed cause of rickets, 473
nature of the poison of, 423
tabular view of stages of, 426
tabular statement contrasting acquired and inherited, 443
theory of imperfect contagion in, 428
transmission of, from parent to offspring, 429, 430
to the third generation, 431
value of treatment in, 413
Syphilis, hereditary, diagnosis of, 440
diagnosis of, from rickets, 485
a protection against acquired syphilis, 427
symptoms of, 429
Syphilis, secondary, diagnosis of, 439
symptoms of, 425, 426
question of prevention of, by treatment, 428
symmetry of symptoms of, 425, 431
treatment of, by mercury, 435
Syphilitic disease of the brain, diagnosis of, 756
a cause of convulsions, 760
Syphilitic mania, pathology of, 614
Syphilitic meningitis, 812
tumors in the brain, 890

TABES DORSALIS, 981, 989
Tache cérébrale, of Trousseau, 821
Talipes following infantile paralysis, 1006
Tannic acid in cholera, 416
in choleraic diarrhœa,
in hooping-cough, 56
in yellow fever, 294
local use of, in hemorrhage in purpura, 468
Tapping the head in chronic hydrocephalus, 839
Tarantism, history of, 701
Taraxacum, in dyspepsia of chronic gout, 545
in intermittent fever, 362
Tarsus, deposits in joints of, in gout, 525
Tartar emetic in treatment of chorea, 714
Tartaric acid, uselessness of, in scurvy, 456
Tartarized antimony, *see* Antimony.
Taste, loss of, in diphtheria, 69
Tea, in scarlet fever, 96
Teeth, early decay of, in rickets, 480
grinding of, a fatal sign in smallpox in children, 137
incisors, deformity of, from hereditary syphilis, 441
late appearance of, in rickets, 480
loss of, in scurvy, 451
Teeth, extraction of, causing abscess of the brain, 936
Teething, a cause of roseola, 105
irritation of rickets mistaken for, 475
see, also, Dentition.
Temper, irritability of the, in congestion of the brain, 847
in gout, 515
in hysteria, 635
in chronic meningitis, 816
in rickets, 476
from softening of the brain, 857
from tumor of the brain, 884
Temperament, influence of, on gout, 527
Temperament, the insane, 588
the nervous, 632, 996
Temperature of body, in cerebro-spinal meningitis, 298, 303
variations of the, after cerebral hemorrhage, 922
in cholera, 399, 410
occasional rise of, after death, in cholera, 410
in dementia, 601
in dengue, not recorded, 101, 102
in diphtheria, 65, 66, 77
in diphtheria, a sudden rise of, an unfavorable sign, 77
in erysipelas, 324
in general paralysis, 607
in acute gout, 511
in influenza, 41
in intermittent fever, 357, 358
in mania, 597
influence of, on prognosis of mania, 617
in measles, 109
in parotitis, 118
in the plague, 314
in pyæmia, 345
in relapsing fever, 277
in remittent fever, 366
in acute rheumatism, 561
in rheumatoid arthritis, chronic, 551
acute, 552
in scarlet fever, 85, 89
local diminution of, after section of nerve, 1025
increase of, after injury to the spinal cord, 945
in sunstroke, 666
in tetanus, 972
in tubercular meningitis, 822
after death from tetanus, &c., 972
in typhoid fever, 202, 218
in typhus fever, 255, 261
in typhus, value of, in prognosis, 266
in yellow fever, 287

Temperature of body—
use of, in diagnosis of rickets, 485
Temperature of air, influence of, on cerebro-spinal meningitis, 309
on cholera, 386
on erysipelas, 322
on influenza, 37
a cause of malaria, 353
influence of, on malarial fevers, 355
on the plague, 318, 319
on remittent fever, 365, 366
on rheumatism, 565
on typhoid fever, 235
on yellow fever, 284
sudden changes of, a cause of roseola, 105
predispose to tetanus, 978
see, also, Cold.
Temporal bone, caries of the, a cause of abscess of the brain, 935, 937
of facial paralysis, 1051
of meningitis, 813, 831
Temporo-maxillary articulation, implication of, in rheumatoid arthritis, 551
Tender spots, or foci, in neuralgia, 1030
Tenderness of whole body in rickets, 477
[Tendon reflex, 987]
Tendons, infiltration of, by urate of soda, in gout, 517
Tenesmus in dysentery, mild, 375
acute, 376
in gouty inflammation of the rectum, 519
Tents, disadvantages of, for cholera hospitals, 420
Teplitz waters in gout, 547
Terminations of disease considered generally, 26
Tertian fever, 355
Tertiary symptoms of syphilis, 425
occurrence of, in other diseases besides syphilis, 423
Tertiary syphilis, 431
diagnosis of, 439
how far modified by mercury, 435
Testicle, abscess of, in pyæmia, 334
inflammation of, in parotitis, 119
swelling of, in dengue, 101
syphilitic diseases of, 434
Tetanus, article on, 968
symptoms, 969
post-mortem appearances, 976
causes, 977
diagnosis, 978
prognosis and treatment, 979
Tetanus, diagnosis of, from hydrophobia, 199
Tetany, of Trousseau, probably a form of spinal irritation, 995
Therapeutics, considered generally, 30
Third cranial nerve, paralysis of the, 1054
Thoracic duct found empty after death from typhoid fever, 215
Thorax, deformity of, in rickets, 479, 491
mechanism of deformity of, in rickets, 489
Thrombi in pyæmia, 337, 340, 342
Thrombosis of the cerebral sinus, 870
a cause of abscess of the brain, 938
of cerebral softening, 861
Thrush, diagnosis of, from diphtheria, 72
Thumb, deformity of, in rheumatoid arthritis, 551
spasmodic contraction of, in hooping-cough, 51
Thymic asthma, in rickets, 483
Tibia, deformity of, in rickets, 478
nodes on, in syphilis, 433
Tic épileptiforme, of Trousseau, 1032
Tinnitus aurium, in chronic alcoholism, 676
in vertigo, 691
Toe, great, affection of, in gout, 513, 514
explanation of, 535
Tongue, in cerebro-spinal meningitis, 298, 301
in dengue, 100
in diphtheria, 65, 66
in dysentery, acute, 376
chronic, 377
mild, 375
in erysipelas, 324, 326
in acute gout, 513
in influenza, 41
in measles, 107, 108
in parotitis, 118
in the plague, 315
in purpura hæmorrhagica, 461
in pyæmia, 344, 346
in relapsing fever, 277, 278
in remittent fever, 366
in acute rheumatism, 559
in gonorrhœal rheumatism, 576
muscular rheumatism in, 574
in scarlet fever, 85, 87, 88
in scurvy, 453
smallpox eruption on, 131, 133
in syphilis, 424
syphilitic muscular nodes in, 433
in typhoid fever, 202, 203, 204, 216
in typhus, 288
in varicella, 126
in yellow fever, 285, 286, 287, 292
Tongue, early affection of the, in general paralysis, 605
in delirium tremens, 680
paralysis of, from hemorrhage into the corpus striatum, 913
into the medulla oblongata, 915
Tongue-biting, value of, in diagnosis of convulsions, 755
in epilepsy, 771
in tetanus, 975
Tonsils, abscess of, in typhoid fever, 216
Tonsils—
enlarged, in parotitis, 119
state of, in diphtheria, 65, 75
state of, in measles, 110
enlargement of, after measles, 111
state of, in scarlet fever, 86, 88
ulceration of, in syphilis, 424
Tonsillitis, diagnosis of, from diphtheria, 70
Tophi in chronic gout, 516
Tormina in dysentery, mild, 375
acute, 376
Torticollis, 574
article on, 1060
definition and synonyms, 1060
causes, 1061
symptoms, 1061
diagnosis, 1062
pathology, 1063
prognosis, 1063
treatment, 1063
Torulæ, in black vomit, 290
in urine in yellow fever, 290
Toxic paralysis agitans, 725
tetanus, 978
Trachea, in diphtheria, 67
smallpox eruption in, 131
in smallpox, 146
ulceration of, in chronic glanders, 191
ulceration of, in typhoid fever, 217
Tracheotomy, in diphtheria, 82
statistics of, 83
in epilepsy, 782
in erysipelas, 329
Trance, cataleptic, 635, 653
Traumatic, cerebral hemorrhage, 904
neuroma, 1022
tetanus, 976
Trees, effect of, on malaria, 353
Tremblement métallique, 725, 801
Trembles, the, 718, 801
Tremor, artuum, 718
ab hydrargyro, 801
saturninus, 806
Tremor, a premonitory symptom of cholera, 399
in typhus, 255, 259
Tremors, classification of, 727
in alcoholism, 676
in epileptics, 774
in paralysis agitans, 720
in wasting palsy, 789
Trigeminal neuralgia, 1030
Trismus in cerebro-spinal meningitis, 300
Trismus, nascentium, 976
in children from worms, 1059
in tetanus, 971
Tubercle, in the brain, 889
in rickety children, 484
of the lung, a sequel of typhoid fever, 209
of mesenteric glands, in pulmonary phthisis, 221, 225
diagnosis of, from typhoid, 245
of Peyer's patches in pulmonary phthisis, 221
in the spinal cord, 1016
Tubercle, the painful subcutaneous, 1022

Tubercular diathesis, a predisposing cause of rheumatoid arthritis, 554
meningitis, article on, 817
[cases of, with recovery, 825, 835]
peritonitis, chronic, diagnosis of, from typhoid fever, 245
ulceration of intestines, diagnosis of, from typhoid, 245
Tuberculosis, diagnosis of, from rickets, 484
a sequel of typhoid fever, 209
a sequel of influenza, 42
acute, after measles, 112
non-identity of, with rickets, 486
Tuberculosis, acute, symptoms of, in children, typhoid form, 828
pulmonary form, 829
Tubuli uriniferi, deposits of urate of soda in, in gout, 526
Tumors of the brain, 883
a cause of convulsions, 746, 760
of cerebral hemorrhage, 904
of meningitis, 832
of chronic hydrocephalus, 836
diagnosis of, from abscess, 940
from epilepsy, 779
from cerebral softening, 881
Tumors of the spinal cord, 1016
Tunbridge waters, in rickets, 496
Tunica vaginalis, inflammation of, in parotitis, 119
Turkish baths, in chronic gout, 545
in gonorrhœal rheumatism, 579
in insanity, 620
Turpentine in treatment of chorea, 715
in purpura, 467
for hemorrhage in typhoid fever, 248
in lung complications of typhus, 268
in yellow fever, 293
in muscular rheumatism, 575
[Turpentine, oil of, in typhoid fever, 248]
Tympanites, in scarlet fever, 87
in typhoid fever, 203, 208
treatment of, 248
in typhus, 257
Tympanum, disease of, a sequel of scarlet fever, 90
Typhoid, cholera, 402
Typhoid fever, article on, 201
associated pathology, 221
causes, 235
clinical history of, 202
definition, 201
diagnosis, 244
distribution, 234
morbid anatomy, 209
pathology, 221
preliminary observations on, 202
prognosis, 246
Typhoid fever—
prophylaxis, 250
synonyms, 201
treatment of, 247
varieties, 232
Typhoid fever, contagious, 233
simple inflammatory, 232
paludal, 233
Typhoid fever, causes of spontaneous origin of, 240
complicating cerebro-spinal meningitis, 303
diagnosis of, from acute meningitis, 815
from tubercular meningitis, 830
diagnosis of, from pyæmia, 348
diagnosis of, from remittent, 368
diagnosis of, from typhus, 246, 263
microscopical appearances of deposits in Peyer's patches in, 212
relation of, to scarlatina and diphtheria, 223
resemblance of some cases to narcotico-irritant poisoning, 205
[Typho-malarial fever, 233]
Typhus fever, article on, 251
definition, 251
diagnosis, 262
duration, 261
etiology, 252
morbid anatomy, 264
mortality, 265
pathology, 263
prognosis, 265
sequelæ, 261
symptomatology, 254
terminations, 261
therapeutics, 266
varieties, 269
Typhus, connection of, with dysentery, 258
diagnosis of, from delirium tremens, 263
diagnosis of, from measles, 262
diagnosis of, from meningitis, 263
diagnosis of, from plague, 316
diagnosis of, from pneumonia, 263
diagnosis of, from purpura, 466
diagnosis of, from pyæmia, 348
diagnosis of, from typhoid, 246, 263
frequently mistaken for typhoid, 245
not identical with epidemic cerebro-spinal meningitis, 311
occurring in epidemics with relapsing fever, 272
origination of, *de novo*, 254
prevalence of, during cholera epidemics, 391

ULCERATION, of cornea, in cerebro-spinal meningitis, 298, 300
in cholera, 403
Ulceration, of cornea—
in smallpox, 133, 134
treatment of, 144
of gums in measles, 110
of intestine, in diphtheria, 76
in pyæmia, 334
in typhoid fever, 210, 211
in typhoid fever, characters of, repair of, 213
distinctions between typhoid and tubercular, 245
of large intestine in dysentery, 379
repair of, 379
of membrana tympani in scarlet fever, 90
in mouth, in glanders, 189
of mucous membrane in tertiary syphilis, 432
in nose, in glanders, 189, 190
of palate in tertiary syphilis, 430
of Peyer's patches in pulmonary phthisis, 221, 226
of pharynx in tertiary syphilis, 430
in diphtheria, 73, 76
of skin, following rash in dengue, 102
in scorbutic dysentery, 377
in scurvy, 453
in secondary syphilis, 424
in tertiary syphilis, 431
local treatment of, 442
of solitary glands, in typhoid fever, 209
of stomach, in intermittent fever, 358
of throat, in scarlet fever, 86, 90
a sequel of scarlet fever, 90
treatment of, 96, 97
Ulceration of vaccine vesicle, 161
Ulna, deformity of, in rickets, 479
Ulnar neuralgia, 1033
Unilateral chorea, 698
convulsions, in children, 742
in adults, 752
paralysis agitans, 723
Uræmia, a cause of convulsions, 759
diagnosis of, from cerebral congestion, 849
from epilepsy, 778
from cerebral hemorrhage, 930
from softening of the brain, 881
relation of, to serous apoplexy, 930
Uræmia, in cholera, 402
treatment of, 419
in scarlatinal dropsy, 92
in yellow fever, 290, 294
Urate of soda, absence of deposits of, in rheumatic joints, 564
in blood destroyed by gouty paroxysm, 533
the cause of premonitory and irregular symptoms in gout, 534
deposits peculiar to gout, 535
deposits of, in gout, 516

Urate of soda, deposits of—
as chalk-stones, 516
in arytænoid cartilages, 525
in articular cartilages, 524
in bones, 525
in bursæ, 516
in kidneys, 526
in ligaments, 516, 525
in sclerotic coat of eye, 517
subperiosteal, 516, 525
in synovial fluid in gouty joints, 525
in tarsal cartilages, 517
selection of situation for, 533
Urates, deposit of, in urine in diphtheria, 65
in gouty urine, 522
in measles, 107, 111
in acute rheumatism, 562
in typhus, 260
Urea in the blood, in cholera, 406, 412
in gout, 521
in relapsing fever, 278
in acute rheumatism, 562
in typhus, 264
in yellow fever, 288
excretion of, normal in scarlet fever, 87, 91
increased excretion of, in diphtheria, 68, 73
in erysipelas, 324
in remittent fever, 367
in acute rheumatism, 562
in typhoid fever, 208
in typhus, 260
in intermittent fever, 358
diminished excretion of, in cholera, 402
during gout, acute, 522
chronic, 523
in relapsing fever, 278
in rickets, 484
in yellow fever, 290
Urethra, diminished discharge from, during gonorrhœal rheumatism, 576
inflammation of, in gout, 520
spasm of, 1058
Urethral discharge, relation of, to rheumatism, 576
Uric acid, absence of, from blood in rheumatoid arthritis, 554
amount excreted daily in acute gout, 522
in chronic gout, 823
deposits of, in cerebro-spinal meningitis, 301
in diphtheria, 68
in scarlet fever, 87
detection of, 521
diminished excretion of, in gout, 534
from quinine, 542
during remittent fever, 367
in urine of rickets, 484
in scarlet fever, 87
excess of, in blood, the essential phenomenon of gout, 533
in blood in gout, 521
detection of, 521
in blood, in lead poisoning, 530
excretion of normal, in typhus, 260
Uric acid—
increased excretion of, in intermittent fever, 358
in remittent fever, during convalescence, 367
in acute rheumatism, 562
in typhoid fever, 208
increased formation of, in gout, 534
in effused fluids in pericarditis and peritonitis in gout, 523
in fluid of blisters in gout, 523
influence of lead on excretion of, 530
normal amount passed daily in urine, 522
not eliminated by the skin, 523
Urinary calculi, in gout, 520
in rickets, 484
Urinary irritation, in intermittent fever, 355, 356
treatment of, 360
Urinary organs, chronic disease of the, a cause of paraplegia, 703, 1003
Urine, in cerebro-spinal meningitis, 301
in chorea, 699
in dengue, 100
in diphtheria, 65
in dysentery, 375, 377
in erysipelas, 324
in gout, 513, 522
in chronic gout, 518, 523
in gout, value of, in prognosis, 538
in influenza, 43
in intermittent fever, 355, 356, 358
in mania, 597
in measles, 107, 111
in malignant measles, 108
in tubercular meningitis, 824
in myelitis, 961
in parotitis, 118
in the plague, 315
in purpura, 462
in relapsing fever, 278
in remittent fever, 367
in acute rheumatism, 559, 562
in muscular rheumatism, 574
in rheumatoid arthritis, 553
in rickets, 477, 484
in scarlet fever, 87, 88, 91
in sunstroke, 666
in typhoid fever, 203, 207
in typhus, 259
in yellow fever, 285, 290
excretion of alcohol in, 672, 928
incontinence of, in the epileptic paroxysm, 771
in locomotor ataxy, 985
during sleep, 660
in sunstroke, 666
involuntary escape of, in typhus, 261
retention of, in dysentery, 376
in meningitis, 810, 824
in myelitis, 960
in smallpox, a rare occurrence, 141
in typhoid, 203, 205, 208
in typhus, 261
Urine—
suppression of, in cholera, 401
in malignant dysentery, 377
in influenza, 43
in the plague, 315
in relapsing fever, 278
after scarlet fever, 91
a rare occurrence in smallpox, 141
in yellow-fever, 285
Urticaria, in cerebro-spinal meningitis, 302, 303
in reaction after cholera, 402
in dengue, 101
in influenza, 42
in scarlet fever, 86
Uterus, diseases of the, a cause of insanity, 590
hemorrhage from, in malignant smallpox, 132
in yellow fever, danger of, 289
muscular rheumatism in, 574
occasionally involved in parotitis, 119
Uvula, condition of, in diphtheria, 65, 75
in scarlet fever, 90
lateral deviation of, in paralysis of the portia dura, 1052
paralysis of, in diphtheria, 69
ulceration of, in diphtheria, 75

VACCINATION, article on, 158
alleged dangers of, 177
methods of, 179
phenomena of, in the human subject, 159
protective power of, 166
relation of vaccina to variola, 176
revaccination, 299
[Vaccination, with scab, common in the United States, 163]
Vaccination, causes of failure of, 165, 171
complicated by measles, 160
by scarlatina, 160
conditions necessary for, 161
during smallpox, 154
history of, 149, 158
importance of careful performance of, 150
inefficiency of, in prevention of smallpox, 150
influence of, on course of smallpox, 137, 140
influence of, on duration of smallpox, 129
influence of, on mortality from smallpox, 152
influence of, on prognosis in smallpox, 137
no protective power against chicken-pox, 124
proportion of failures in, 165, 171
protective power of, statistics of, 167, 169, 170
value of cicatrix of, in prognosis of smallpox, 151
Vaccine lymph, blood in, 163
blood in, supposed to be the cause of vaccino-syphilitic inoculation, 179

Vaccine lymph—
degeneration of, 172
deterioration of, 153, 154
dried, vaccination from, 164
impure, a cause of spurious cow-pox, 161
mode of collecting, 162
time for collecting, 162
Vaccinia, identity of, with smallpox, 176
in the cow, 158
insusceptibility to, 166
Vaccino-syphilitic inoculation, 178
alleged case of, under M. Trousseau, 180
alleged case at Rivalta, 180
Vagina, diphtheritic exudation in, 68
discharge from, after measles, treatment of, 116
Valerian, in hooping-cough, 56
in rheumatoid arthritis, 557
Valves of heart, affected in pyæmia, 333
changes in, after injection of lactic acid into the peritoneal cavity of a rabbit, 566
disease of, in acute rheumatism, 560, 563
Vari on mucous membranes in smallpox, 131, 137
Varicella, article on, 124
causes, 125
definition, 124
description of disease, 125
diagnosis, 126
prognosis, 127
synonyms, 127
treatment, 127
varieties, 127
Varicella, confounded with smallpox, 124
complicating scarlet fever, 93
diagnosis of, from smallpox, 136
not identical with smallpox, 124
sequelæ of, 126
Varicelloid, 149
Variola, benigna, 132
confluens, 130
corymbosa, 131
discreta, 129
maligna, 131
sine eruptione, 139
varicelloides, 151
see Smallpox.
Variolæ anomalæ, 132
Varioloid, 151
Variolous fever, 139
ophthalmia, 134
Vaso-motor nerves, connection of the, with the spinal cord, 944
Vegetables, in gout, 548
fresh, want of, the cause of scurvy, 445, 455
a cause of purpura, 463
preserved, useless as an anti-scorbutic, 458
Vegetations on valves of heart in pyæmia, 333
Veins, enlargement of, leading from inflamed joints in gout, 513, 514
inflammation of, in erysipelas, 326
Veins—
specially implicated in pyæmia, 339
state of, in pyæmia, 335
pus in, in erysipelas, 326
superficial, enlargement of, in rickets, 476
Venereal excesses in parents a cause of rickets, 473
[Venesection, use of in practice, 31]
see Bloodletting.
Venous congestion a supposed cause of gout, 532
Venous hum in purpura, 462
Ventilation, want of, a cause of pyæmia, 344
predisposes to heat apoplexy, 663
Ventricles of brain, condition of, in insanity, 614
in tubercular meningitis, 833
effusion into, in the plague, 316
hemorrhage into, 903, 911
Ventricular hydrocephalus, 836
Vertebræ, caries of the, symptoms of, 1017
treatment, 1018
Vertigo, article on, 690
definition, 690
varieties and their causes, 691
treatment, 695
Vertigo, in cerebro-spinal meningitis, 297, 301
[from disorder of the liver, 693]
gastric, 691
from overwork, 694
in the plague, 314
premonitory of cerebral hemorrhage, 924
in scurvy, 453
senile, 693
a symptom of chronic alcoholism, 676
of epilepsy, 768
of chronic meningitis, 816
of tumor of the brain, 885
Vesication of skin, in cerebro-spinal meningitis, 298, 302
in erysipelas, 323
occasionally present in rash of dengue, 102
occasionally found in the rash of measles, 109
in pyæmia, 335
in smallpox, cause of umbilication in, 148
in rash of congenital syphilis, 441
in vaccinia, 159
Vibices, in cerebro-spinal meningitis, 299, 302
in the plague, 314
in purpura, 460
in pyæmia, 331, 332
of pleuræ, in pyæmia, 332
Vibratile muscular tremors, in wasting palsy, 789
Vibriones in urine in scarlet fever, 88
Vichy waters, in gout, 547
danger of, in rheumatoid arthritis, 557
presence of lithia in, 544
Villi of small intestine in typhoid fever, 213
Vision frequently affected in diphtheria, 69
see Sight.
Voice, character of, in cholera, 400
in diphtheria, 66
in diphtheritic paralysis, 68
in malignant dysentery, 377
in glanders, 189, 190
in influenza, 42
in measles, 107
in scarlet fever, 86
Vomit, black, in yellow fever, 285, 289
in cholera, chemical examination of, 405
Vomiting, in epidemic cerebro-spinal meningitis, 297, 298, 301
in cholera, 399, 400, 402
in cholera, how far eliminative, 413
treatment of, 417, 418
occasional absence of, 406
uræmic, in cholera, 402
in choleraic diarrhœa, 407
in dengue, 100
in diphtheria, 65, 66, 68
treatment of, 78
in erysipelas, 322
in glanders, 189
in gout affecting the stomach, 519
in hooping-cough, 50
in influenza, 43
in intermittent fever, 357
treatment of, 361
in measles, 107, 111
treatment of, 114, 115
in Pali plague, 318
in parotitis, 118, 119
in the plague, 315
in pyæmia, 344, 346
in relapsing fever, 277, 278
in remittent fever, 366, 367
treatment of, 370
in rickets, 482
in scarlet fever, 85
in smallpox, 732
in typhoid fever, 203, 204
[in typhoid fever, most frequent in children, 204]
in typhoid fever, a symptom of perforation, 208
in typhoid fever, treatment of, 247
in typhus fever, 255, 257
treatment of, 268
in yellow fever, 285, 289, 293
Vomiting, a symptom of chronic alcoholism, 677
of simple meningitis, 809
of tubercular meningitis, 819, 821
of migraine, or sick headache, 1031
of tumor of the brain, 884
Vomiting, from brain disease, 756
premonitory of cerebral hemorrhage, 924
Vulva, gangrenous inflammation of, in measles, 112

WALCHEREN fever, 358, 365
Warburg's tincture in intermittent fever, 362

Warburg's tincture—
in remittent fever, 371
in treatment of sunstroke, 669
Warm affusion in scarlet fever, 96
Wasting palsy, article on, 786
definition, history, and synonyms, 786
etiology, 786
symptoms, 788
course and duration, 789
diagnosis and morbid anatomy, 790
pathology, 793
prognosis, 796
treatment, 798
Wasting palsy, diagnosis of, from writer's cramp, 735
Water, impure, as a cause of cholera, 389
a cause of dysentery, 375
of intermittent fever, 242
of typhoid fever, 241
as a means of propagating malarial fevers, 353
its power of absorbing malaria, 353
want of, a predisposing cause of typhus fever, 253
Watery-pock, 131
[Weekly recurrence of chills, common, 361]
Wet sheet, packing in, for remittent fever, 369
for scarlet fever, 96
White patches, on heart, in rickets, 493
in spleen in rickets, 493
distinction of, from embolism, 493
White softening of the brain, 873
"White streak" in scarlet fever, 85
Wiesbaden waters in gout, 547
danger of, in rheumatoid arthritis, 557
Wildbad waters in gout, 547
Winds, influence of, on spread of cholera, 387
Wine, a cause of gout, 527, 528
choice of, in gout, 549
Women, predisposition of, to disease, *see* Sex and Sexual disorders.
Worms, intestinal, a cause of asthma, 1057
of catalepsy, 655
of chorea, 710
of infantile convulsions, 739
of abdominal spasms, 1056
Wort-pock, 132
Wounds, a cause of tetanus, 977
of the brain, a cause of abscess, 934
Wounds, condition of, in hydrophobia, 198
diphtheritic exudation on, 68
exciting causes of erysipelas, 322, 323
appearance of, in pyæmia, 344
as a cause of pyæmia, 330, 338
treatment of, during pyæmia, 350
Wrists, enlargement of, in rickets, 481, 488
rigidity of, in rheumatoid arthritis, 551
Writer's cramp, article on, 732
definition, 732
synonyms, 733
symptoms, 733
etiology, 784
diagnosis, 734
prognosis and pathology, 735
treatment, 737
Wry-neck, 1060

YELLOW FEVER, article on, 281
altitudinal and horizontal ranges, 284
definition, 281
diagnosis, 285
history, 281
morbid anatomy, 290
pathology, 286
prognosis, 292
symptoms, 284
synonyms, 281
therapeutics, 293
varieties, 294
Yellow fever, diagnosis of, from ephemeral fever, 286
diagnosis of, from remittent fevers, 286, 368
resemblance of severe remittent to, 367
tint of skin in, 287
[Yellow fever not contagious, 283]
Yellow softening of the brain, 875

ZINC, value of, in the treatment of alcoholic insomnia, 685
of chorea, 711
of epilepsy, 780
of neuralgia, 1043
Zinc, oxide of, in hooping-cough, 54, 55
use of, in preventing pitting in smallpox, 144
locally, in erysipelas, 328
Zinc, sulphate of, in diphtheria, 82
lotion for otorrhœa, 116
Zymotic diseases, general nature of, 138

LIST OF CHIEF AUTHORS REFERRED TO IN EACH ARTICLE.

ABSCESS OF THE BRAIN, Article on, by Sir William Gull, Bart., M.D., F.R.S., &c., and Henry G. Sutton, M.B., p. 934.

AUTHORS REFERRED TO.

Abercrombie, Diseases of the Brain, 938
Lebert, Anatomie Pathologique, 936, 937
Prescott Hewett, on Injuries of the Head (Holmes' Surgery, vol. ii.), 934
Rokitansky, Path. Anat. (Syd. Society), 936
Toynbee, Diseases of the Ear, 935
Papers by Sir W. Gull in Guy's Hospital Reports, § 3, vol. viii., and by Dr. Ogle in Med.-Chir. Review, No. LXX., &c.

ADVENTITIOUS PRODUCTS IN THE BRAIN, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., and H. Charlton Bastian, M.D., F.R.S., &c., p. 883.

AUTHORS REFERRED TO.

Abercrombie, on Diseases of the Brain, &c., 884, 898
Andral, Clinique Médicale and Anat. Pathol., 890, 899
Bouchard, de la Path. des Hémorrhagies Cérebrales, 894
Cobbold, S., on Entozoa in the Brain, 897
Cruveilhier, Anatomie Pathologique, 889, 894
Lebert, Anat. Pathologique, 890, 892
Reynolds, J. R., Diagnosis of Diseases of the Brain, 885
Rokitansky, Pathol. Anatomy (Syd. Society), 889, 893
Romberg, Manual of Nervous Diseases (Syd. Society), 887
Virchow, Cellular Pathology, &c., 890, 892
Watson, Sir Thomas, Practice of Physic, 900
Wilks, S., Pathol. Anatomy, 890

ALCOHOLISM, Article on, by Francis Edmund Anstie, M.D., F.R.C.P., &c., p. 670.

AUTHORS REFERRED TO.

Anstie, Dr. E. F., on Stimulants and Narcotics, &c. See list at end of article, 690
Huss, Magnus, Chronische Alkohols-Krankheit, 690
Lallemand, Perrin et Duroy, MM., du Rôle de l'Alcool et des Anæsthétiques dans l'Organisme, 672
Lancereaux, on Morbid Anatomy of Alcoholism, in Archives Générales, Oct. 1865, 684
Marcet, Dr., on Chronic Alcoholic Intoxication, 685
Moreau, Psychologie Morbide, 675
Roesch, on Oinomania, &c. (Annales d'Hygiène, tome xx.), 681
Schulinus, Dr., on the Elimination of Alcohol (in Arch. d. Heilkunde, 1866), 673
Ware, D. J., on the History and Treatment of Delirium Tremens (1831), 671

[ATHETOSIS, Article on, by Henry Hartshorne, A.M., M.D., p. 731.]

CATALEPSY, Article on, by Thomas King Chambers, M.D., F.R.C.P., &c., p. 652.

AUTHORS REFERRED TO.

Goebel, Dr., de Catalepsi, 656
De Haen, Ratio Medendi, 655
Marx, de Spasmis, 656
Tissot, M., Works of, vol. xiii., 654, 657
Van Swieten's Commentary on Boerhaave, 655
Mémoires de l'Académie Royale des Sciences, Paris, &c.

CEREBRAL HEMORRHAGE AND APOPLEXY, Article on, by J. Hughlings Jackson, M.D., F.R.C.P., p. 902.

AUTHORS REFERRED TO.

Brown-Séquard, on the Phys. and Path. of the Nervous System, and Lectures in the *Lancet* (1866), 914
Flint, Dr. Austin, Practice of Medicine, and on Diseases of the Heart, 905, 906
Hutchinson, J., Lectures on Compression of the Brain (London Hospital Reports), 920
Jaccoud, Pathologie Interne, 921
Lockhart Clarke, on the Intimate Structure of the Brain, &c. (Philos. Trans. 1868), 910, 915
Niemeyer, F. von, Handbook of Practical Medicine, 906, 907
Paget, Surgical Pathology, 905
Prescott Hewett, article on Injuries of the Head (Holmes' Surgery, vol. ii.), 929
Todd, Diseases of the Nervous System, 909, 913
Trousseau, Clinique Médicale, vol. i., 924
Virchow's Cellular Pathology, 907
Wilks, S., on Pathology of Nervous Diseases (Guy's Hosp. Rep. 1866), 913, 921
Papers and cases in Pathol. Soc. Trans. by Drs. Bristowe, Moxon, Ogle, &c.
For numerous other references to papers by Drs. Bastian, Broadbent, Gull, Kirkes, George Johnson, &c., see footnotes.

[CHLOROSIS, Article on, by Henry Hartshorne, A.M., M.D., p. 468.]

CHOLERA, Article on, by Edward Goodeve, M.B., p. 384.

AUTHORS REFERRED TO.

Ayre, Dr., on a method of treating cholera, 421
Baly, Dr., on contagion in cholera, 394; on mortality from cholera, 392
Bœhm, on the post-mortem appearances in cholera, 411
Budd, Dr. W., on the intestinal discharges as the medium of communication of cholera, 395
Chevers, Dr., on the effect of elevation on cholera, 388
Colledge, Mr., on the prodromata of cholera, 399
Ewart, Dr., on mortality from cholera, 386, 408
Farr, Dr., on the effect of soil on cholera, 388
Gairdner, Dr. W. T., on cholera stools, 404
Garrod, Dr., on the blood in cholera, 406; on urea in the blood of cholera, 412
Glaisher, Mr., on the atmospheric conditions during cholera, 387
Grainger, Mr., on bad food as a cause of cholera, 389; on the effect of age on the mortality from cholera, 390
Gull, Dr., on cholera stools, 404; on post-mortem rise of temperature in cholera, 410; on the effects of age on mortality in cholera, 390
Johnson, Dr. G., on injection into the veins in cholera, 413
Latta, Dr., his formula for saline injections in cholera, 421
Mackinnon, Dr., on a variety of cholera, 407; on the portability of cholera-poison, 395
Martin, Sir R., on cholera, 407
Morehead, Dr., on contagion in cholera, 394; on consecutive diseases in cholera, 403
O'Shaughnessy, Sir W., on the blood in cholera, 406
Parkes, Dr. E. A., on cholera stools, 404; on the blood in cholera, 406; on the post-mortem appearances of cholera, 410
Rees, Dr. Owen, on a formula for saline injections in cholera, 421
Schmidt, on the blood in cholera, 406; formula for saline injections in cholera, 421
Snow, Dr., on impure water as a cause of cholera, 389
Stevens, Dr., on saline treatment of cholera, 421
Strachey, Mr. J., on the diffusion of cholera, 393
Thom, Mr., on atmospheric conditions during cholera, 387; on cholera fever, 407; on the duration of cholera, 406
Twining, Mr., on spasmodic cholera, 406

CHOREA, Article on, by C. B. Radcliffe, M.D., F.R.C.P., p. 669.

AUTHORS REFERRED TO.

Barlow, Dr., on the value of iron and zinc in the treatment of Chorea, 712
Begbie, Dr., on arsenic in Chorea, 712
Dubini, Dr., on Electric Chorea, 700
Hecker, Dr., on the Epidemics of the Middle Ages, 701
Hillier, Dr., Clinical Lectures on the Diseases of Children, 698, 709
Romberg's Manual of Nervous Diseases (Syd. Soc.), 699
Sydenham, Dr., description of Chorea, 696, 697

Todd, Dr., on the urine in Chorea (Clinical Lectures), 700
Trousseau, Dr., on treatment of Chorea by strychnia (Clinique Médicale), 698, 713
Watson, Sir Thomas, Practice of Physic, 697, 711
West, Dr., on Diseases of Children, 699
And papers by Drs. Kirkes, Hughlings Jackson, Bastian, &c., on the connection of chorea with capillary embolisms in the brain, &c.

CHRONIC HYDROCEPHALUS, Article on, by J. Spence Ramskill, M.D., &c., p. 836.

AUTHORS REFERRED TO.

Barnard, T. H., on Chronic Hydrocephalus, 839
Gölis, on cure of Hydrocephalus by mercurial inunction, 859
Legendre, Recherches anatomo-pathologiques, &c., sur quelques Maladies de l'Enfance, 836
Rilliet et Barthez, Maladies des Enfants, 836
Rokitansky, Pathologische Anatomie, 836
Trousseau, Clinique Médicale, 836
Vrolik, Traité sur l'Hydrocephalie Interne, 837
Watson, Sir Thomas, Practice of Physic, 836
West, on Diseases of Children, 836

CONGESTION OF THE BRAIN, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., and H. Charlton Bastian, M.D., F.R.S., &c., p. 844.

AUTHORS REFERRED TO.

Abercrombie, on Diseases of the Brain, 850
Andral, Clinique Médicale, 850
Bastian, Charlton, paper in *British Medical Journal* (Jan. 1869) on Capillary Embolisms in the Brain as a cause of Delirium, &c., 850
Burrows, on Disorders of the Cerebral Circulation, 850
Durand-Fardel, Maladies des Viellards, 847, 852
Laborde, Ramollissement et Congestion du Cerveau, 852
Reid, Dr. J., Physiol. Anat. and Pathol. Researches, 850
Robin, on perivascular lymphatic sheaths in the Brain (Brown-Séquard's *Journal de Physiologie*, 1859), 850

CONVULSIONS, Article on, by J. Hughlings Jackson, M.D., F.R.C.P., p. 737.

AUTHORS REFERRED TO.

Gee, Dr. S., on Convulsions in Children (St. Bartholomew's Hospital Reports), 741
Gull, Sir William, on Cerebral Aneurisms (Guy's Hospital Reports), 759
Hillier, Dr., Clinical Lectures on Diseases of Children, 739
Jenner, Sir William, on Rickets, 742
Meigs and Pepper, Drs., on Diseases of Children, 733, 748
Niemeyer, Dr. F. von, Handbook of Practical Medicine, 741
Reynolds, Dr. J. R., on Epilepsy, 754
Todd, Dr., on Nervous Diseases, 753
Trousseau, Clinique Médicale (Syd. Society), 738, 750
Vogel, Dr. A., Practical Treatise on Diseases of Children (Raphael's translation), 748
West, Dr., on Diseases of Children, 738
Wilks, Dr., on the Pathology of Nervous Diseases (Guy's Hospital Reports, 1866), 754

DENGUE, Article on, by W. Aitken, M.D., p. 98.

AUTHORS REFERRED TO.

Cavell, Dr., on dengue in general, 98 *et seq.*
Cock, Dr., on dengue in general, 99 *et seq.*
Mouat, Dr., on dengue in general, 98 *et seq.*
Stedman, Dr., on dengue in general, 99 *et seq.*
Twining, Dr., on dengue in general, 98 *et seq.*
Various other authors of less note, *see* p. 98 *et seq.*

DIPHTHERIA, Article on, by William Squire, L.R.C.P. Lond., p. 57.

AUTHORS REFERRED TO.

Abercrombie, Dr. J., on diphtheria in general, 60
Aëtius of Amida, description of diphtheria by, 58
Aëtius Cletus, on the symptoms of diphtheria, 59
Aretæus, description of diphtheria by, 58
Asclepiades, description of diphtheria by, 58
Ballard, Dr., on the mortality in diphtheria, 77; on the relation between scarlatina and diphtheria, 64, 71
Bell, Sir Charles, on the use of sesquichloride of iron in diphtheria, 79
Bretonneau, on the state of the pharynx in diphtheria, 75; on contagion in diphtheria, 63
Brown, Mr. J. D., on the appearance of diphtheria in South Wales, 60
Buchanan, Dr., on scarlet fever complicating diphtheria, 71
Celsus, mention of diphtheria by, 58
Dewar, Dr., on the use of sulphurous acid in diphtheria, 80
Dixon, Mr., on impairment of vision in diphtheritic paralysis, 74
Farr, Dr. W., the introducer of the word "diphtheria," 58
Fothergill, on diphtheria in general, 59, 62, 72
Fox, Dr. Wilson, on diphtheritic exudation, 75
Greenhow, Dr., on recurrence of diphtheria, 64
Gull, Dr., on diphtheritic paralysis, 69; on recurrence of diphtheria, 64
Hache, M., on diphtheritic exudation, 75
Hippocrates, description of diphtheria by, 58
Hoffman, J. F., on paralysis of nerves of special sense in diphtheria, 69
Jenner, Sir William, on the symptoms of diphtheria, 60, 64, 69
Mercatus, on a rash in diphtheria, 72
Mackenzie, Dr., on diphtheria, 60
More, Mr. James, on diphtheria in Tasmania, 61
Roseh, Prof., on tracheotomy in diphtheria, 83
Ryland, Mr., on epidemic diphtheria in 1837, 60
Sanderson, Dr., on excretion of urea in diphtheria, 73
Starr, Dr., on the symptoms of diphtheria, 59
Trousseau, M., on diphtheritic paralysis, 69, 74; on inoculation of diphtheria, 63; on tracheotomy in diphtheria, 83
Wade, Dr., on albuminuria in diphtheria, 73
Webster, Dr., on diphtheria, 60
Various authors of less note who have written on diphtheria, *see* p. 58

DYSENTERY, Article on, by W. E. Maclean, M.D., p. 372.

AUTHORS REFERRED TO.

Aitken, Dr., on the post-mortem appearances of dysentery, 378, 379
Baly, Dr., on the outbreak of dysentery at Milbank Prison, 374; on the post-mortem appearances in dysentery, 379
Chevers, on the causes of dysentery, 375
Crawford, Staff Surgeon, on the causes of dysentery, 374
Docker, Mr., on ipecacuanha in dysentery, 381
Grant, Dr. A., on Bael fruit in dysentery, 382
Mackay, on cold as a cause of dysentery, 375
Martin, Sir R., on Bael fruit in dysentery, 382; on the mortality from dysentery, 373
Morehead, Dr., on mercury in dysentery, 384; on the post-mortem appearances of dysentery, 379
Parkes, Dr. E. A., on the post-mortem appearances of dysentery, 378

ECSTASY, Article on, by Thomas King Chambers, M.D., F.R.C.P., &c., p. 646.

AUTHORS REFERRED TO.

Haygarth, Dr., on the Imagination as a Cause and Cure of the Disorders of the Body, 648
Hecker's Epidemics of the Middle Ages, 647
Hoffman, Medicina Rationalis, 646
Tissot, M., on Religious Ecstasy, 646.

EPIDEMIC CEREBRO-SPINAL MENINGITIS, Article on, by J. Netten Radcliffe, Esq., p. 296.

AUTHORS REFERRED TO.

Day, Dr. H., on the effect of mouldy grain on rabbits, 310; on the eruption in epidemic cerebro-spinal meningitis, 311
Githens, Dr. W. H. H., on the pulse in epidemic cerebro-spinal meningitis, 302; on the mortality in, 305

Gordon, Dr. S., on a case of epidemic cerebro-spinal meningitis, 304; on purpura in epidemic cerebro-spinal meningitis, 303
Klebs, Dr., on the state of the kidneys in epidemic cerebro-spinal meningitis, 306; on the state of the spinal cord, 305
Lyons, Dr., on the varieties of epidemic cerebro-spinal meningitis, 312
Richardson, Dr. B. W., on a supposed cause of epidemic cerebro-spinal meningitis, 310
Sanderson, Dr. Burdon, on the spasms in epidemic cerebro-spinal meningitis, 299; on morbid anatomy of, 305
Stillé, Dr. A., on the treatment of epidemic cerebro-spinal meningitis, 313
Stokes, Prof., on contagion in epidemic cerebro-spinal meningitis, 309

EPILEPSY, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., p. 762.

AUTHORS REFERRED TO.

Bucknill, Dr., on Epilepsy in the Insane (*Asylum Journal*, October, 1855), 772
Doussin Dubreuil, de l'Epilepsie en général, 773
Duckworth Williams, Dr., on the Efficacy of the Bromide of Potassium in Epilepsy, &c., 766
Esquirol, Traité des Maladies Mentales, 763
Herpin, du Pronostic, &c., de l'Epilepsie, 773
Leech and Fox, Messrs., paper on Epilepsy, in Manchester Med. and Surg. Reports, vol. i., 763, 765
Maisonneuve, Recherches de sur l'Epilepsie, 773
Marshall Hall, Dr., on laryngeal spasm in Epilepsy (Memoirs on the Neck), 768
Niemeyer, F. von, Handbuch der Speciellen Pathologie, 763
Pritchard, Dr. J. C., on Diseases of the Nervous System, 773
Radcliffe, Dr., on Epilepsy and other Convulsive Affections, 773
Reynolds, Dr. J. R., on Epilepsy, 764, 767
Romberg's Manual of Nervous Diseases (Syd. Society), 770
Schroeder van der Kolk, on the Pathology, &c., of the Medulla Oblongata and Spinal Cord (Syd. Society), 773, 777
Tissot, Traité de l'Epilepsie, 770
Wenzel, Observations sur le Cervelet, 777

ERYSIPELAS, Article on, by J. Russell Reynolds, M.D., F.R.S., p. 321.

AUTHORS REFERRED TO.

Arnott, Mr., on suppuration in the veins in erysipelas, 326
Bastian, Dr., on minute embolisms in the brain and cord in erysipelas, 326
Busk, Mr., on pus in the pulmonary vessels in erysipelas, 326
Niemeyer, M., on inflammation of the lymphatics in erysipelas, 324
Ribes, M., on pus in the veins in erysipelas, 326

GLANDERS, Article on, by Arthur Gamgee, M.D., and John Gamgee, Esq., p. 182.

AUTHORS REFERRED TO.

Aristotle, on glanders in the ass, 182
Elliotson, Dr., on glanders in man, 188
Muscroft, Mr. T., on glanders in man, 186
Rayer, on the state of the lungs in glanders, 183
Schilling, on glanders in man, 186
Tardieu, M., on chronic farcy, 191
Travers, on the communication of glanders by the blood, 184; on glanders in man, 187
Trousseau, M., on the state of the lungs of the horse in glanders, 183
Vegetius, on glanders in the ass, 182

GONORRHŒAL RHEUMATISM, Article on, by Bernard Edward Brodhurst, F.R.C.S., p. 576.

AUTHORS REFERRED TO.

Brodie, Sir Benjamin, on the pathology of gonorrhœal rheumatism, 576
Cooper, Sir Astley, on a case of gonorrhœal rheumatism, 576
Monteggria, supposed description of gonorrhœal rheumatism by, 576
South, Mr., the recognition of gonorrhœal rheumatism by, 576
Swediaur, supposed description of gonorrhœal rheumatism by, 576

GOUT, Article on, by Alfred Baring Garrod, M.D., F.R.S., p. 512.

AUTHORS REFERRED TO.

Aëtius, mention of gout by, 512
Aretæus, mention of gout by, 512; on disease of the kidney in gout, 525
Barthez, M., on the pathology of gout, 533
Begbie, Dr., on white patches on the heart in gout, 519
Boerhaave, on the preference of gout for the great toe, 535
Ceeley, Mr., on deposits of urate of soda in the kidney in gout, 526
Celsus, mention of gout by, 512
Charcot, Dr., and Cornil, M., on gouty deposits, 525, 526
Chelius, Prof., on the effect of colchicum on the urine, 541
Christison, Dr., on the effect of colchicum on the urine, 541
Cœlus Aurelianus, mention of gout by, 512
Cruveilhier, on the pathology of gout, 533; on gouty deposits in bone, 525
Cullen, on the pathology of gout, 531
Demetrius Pepagomenos, mention of gout by, 512
Gairdner, Dr. W. T., on the pathology of gout, 532
Galen, mention of gout by, 512
Hippocrates, mention of gout by, 512
Holland, Sir Henry, on the pathology of gout, 532
Home, Sir Everard, on the pathology of gout, 532
Jones, Dr. Bence, on wines, 528
Maclagan, Dr. J. M., on the effect of colchicum on the urine, 541
Morgagni, on diseases of the kidney in gout, 525
Paulus Ægineta, mention of gout by, 512
Ranke, Dr., on the effect of quinine on the excretion of uric acid, 542
Ruef, Dr., on Baden-Baden waters in gout, 547
Scudamore, Sir C., on the pathology of gout, &c., 514, 527, 532
Seneca, mention of gout by, 512
Sydenham, on disease of the kidney in gout, 525
Todd, Dr., on gouty deposits in the kidney, 526
Trallianus, Alexander, mention of gout by, 512
Trousseau, M., on the pathology of gout, 533
Van Swieten, on the preference of gout for the great toe, 535
Wood, Dr., on cider as a predisposing cause of gout, 528

HOOPING-COUGH, Article on, by Edward Smith, M.D., F.R.S., p. 48.

AUTHORS REFERRED TO.

Badham, on the nature of hooping-cough, 49
Bird, Dr. Golding, on the treatment of hooping-cough, 56
Boerhaave, on the treatment of hooping-cough, 54
Broussais, on the stomach as the source of irritation in hooping-cough, 49
Chambon, on the stomach as the source of irritation in hooping-cough, 49
Copland, Dr., on the medulla oblongata as the seat of hooping-cough, 49
Cullen, on the nervous nature of hooping-cough, 49
Dawson, on the glottis as the seat of hooping-cough, 49
Desruelles, on the nature of hooping-cough, 49
Dewees, on the nature of hooping-cough, 49
Gibb, Dr., on the antiquity of hooping-cough, 48; on the nervous nature of hooping-cough, 49
Graves, Dr., on the treatment of hooping-cough, 55
Guersant, on the nature of hooping-cough, 49
Guibert, on the whole nervous system as the seat of hooping-cough, 49
Hippocrates, mention of hooping-cough by, 48
Hoffmann, on the vagus as the seat of hooping-cough, 49
Hufeland, on the vagus as the seat of hooping-cough, 49
Jahn, on the phrenic as the seat of hooping-cough, 49
Laennec, on the nature of hooping-cough, 49
Leroy, on the nervous nature of hooping-cough, 49
Löbel, on the nervous nature of hooping-cough, 49
Lobenstein, on the nervous nature of hooping-cough, 49
Millot, on the diaphragm as the seat of hooping-cough, 49
Müller, on the treatment of hooping-cough, 55
Paldame, on the lungs as the seat of hooping-cough, 49
Pearson, Dr., on the treatment of hooping-cough, 55
Pinel, on the nervous nature of hooping-cough, 49
Rees, Dr. Owen, on the treatment of hooping-cough, 55
Ringer, Dr. S., on the treatment of hooping-cough, 55
Roe, Dr. Hamilton, on the treatment of hooping-cough, 55
Todd, Dr., on the nervous nature of hooping-cough, 49
Trousseau, on the treatment of hooping-cough, 54
Watson, Dr. Eben, on the treatment of hooping-cough, 55

Watson, Sir Thomas, on the treatment of hooping-cough, 54
Watt, on the nature of hooping-cough, 49
Wendt, on the lungs as the seat of hooping-cough, 49
West, Dr. C., on the treatment of hooping-cough, 54
Williams, Dr., on the treatment of hooping-cough, 54
Willis, Dr., the earliest description of hooping-cough, 48

HYDROPHOBIA, Article on, by Arthur Gamgee, M.D., and John Gamgee, Esq., p. 192.

AUTHORS REFERRED TO.

Bardsley, Dr., on the antiquity of hydrophobia, 193
Celsus, description of hydrophobia by, 193, 198
Cullen, description of hydrophobia by, 194
Desault, on immersion for hydrophobia, 200
Dioscorides, mention of hydrophobia by, 193
Earle, Mr. H., on the frequency of hydrophobia, 195
Fothergill, Dr., description of hydrophobia by, 194
Galen, description of hydrophobia by, 193
Hippocrates, mention of hydrophobia by, 193
Mead, Dr., description of hydrophobia by, 194
Reynoult, M., on incubative period of hydrophobia, 196
Van Swieten, description of hydrophobia by, 194
Youatt, on the frequency of bites from rabid animals, 195

HYPOCHONDRIASIS, Article on, by Sir William Gull, Bart., M.D., D.C.L., F.R.S., &c., and Francis E. Anstie, M.D., F.R.C.P., &c., p. 623.

AUTHORS REFERRED TO.

Burton, on the Anatomy of Melancholy, 624, 626
Cullen, Dr., Clinical Lectures (1777), 624
Falret, Dr. J., de l'Hypochondrie (1822), 624
Flemying, Neuropathia, sive de Morb. Hypochond. et Hyster. (1744), 624
Galen, on Pathology of Hypochondriasis, 623
Georget, de la Physiologie du Syst. Nerv. (1819), 624
Griesinger, Dr. W., die Path. und Therap. der psych. Krankheiten, 624
Hippocrates, description of Hypochondriasis, 623
Leidesdorf, Dr. M., die Path. und Therap. der psych. Krankheiten, 624
Whytt, Dr. R., Observations on the Nature, &c., of the Disorders called Nervous, Hypochondriacal, &c. (1777), 624
Willis, T., on Hysteria and Hypochondriasis (1676), 624

HYSTERIA, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., p. 630.

AUTHORS REFERRED TO.

Briquet, M., Traité Clinique, &c., de l' Hystérie, 633
Carter, R. B., on the Pathology and Treatment of Hysteria, 641
Hare, Dr. C. J., on the Treatment of the Hysteric Paroxysm, 645
Hoveil, Medicine and Psychology, 641
Landouzy, Traité complet de l'Hystérie, 631, 638
Morell Mackenzie, on the Treatment of Hysterical Aphonia, 645, 646
Niemever, F. von, Handbuch der Speciellen Pathologie, 632, 639
Todd, Dr., on hysterical paralysis; Clinical Lectures, 638

[HYSTERO-EPILEPSY, Article on, by Henry Hartshorne, A.M., M.D., p. 649.]

INFLUENZA, Article on, by E. A. Parkes, M.D., F.R.S., p. 33.

AUTHORS REFERRED TO.

Arbuthnot, on the epidemic of influenza in 1732–33, 34
Baker, Sir George, on the epidemic of influenza in 1762, 34; on the causes of influenza, 40; on intermittent fever complicating influenza, 43

Biermer, on autochthonic development of influenza, 36
Blakiston, on lobelia in influenza, 46
Bryson, Dr., on the epidemic of influenza in 1837, 34
Diodorus Siculus, supposed mention of influenza by, 34
Falconer, on the epidemic of influenza in 1803, 34, 38
Fothergill, Dr., on the epidemic of influenza in 1775, 34; on heat of the skin in influenza, 42
Gluge, on the course taken by epidemics of influenza, 35
Graves, Dr., on the epidemic of influenza in 1837, 34; on the relation of influenza to other epidemic diseases, 39; on paralysis of the vagus in influenza, 42
Gray, on the epidemic of influenza in 1782, 34
Haygarth, on the epidemic of influenza in 1782, 34, 38
Heberden, on the epidemic of influenza in 1767, 34
Hertwig, on the non-inoculability of influenza, 38
Hingesten, on the epidemic of influenza in 1833, 34
Hippocrates, supposed mention of influenza by, 34
Huxham, on the epidemics of influenza in 1729 and 1743, 34; on delirium in influenza, 42
Laycock, Dr., on the epidemic of influenza in 1847, 34
Legendre, on plasters between the shoulders in influenza, 47
Peacock, Dr., on the epidemic of influenza in 1847, 34; on an herpetic eruption in influenza, 42
Pearson, Dr., on the epidemic of influenza in 1803, 34; on the causes of influenza, 37
Riverius, on the epidemic of influenza in 1557, 34
Rutty, Dr., on the epidemic of influenza in 1762, 34
Salius Diversus, on the heat of skin in influenza, 41
Schnurrer, on the influence of telluric emanations on influenza, 36
Schönbein, on the influence of ozone on influenza, 37; on warm baths in influenza, 45
Sennert, on the epidemic of influenza in 1508, 34
Short, Dr. T., on epidemics of influenza in 1510 and 1557, 34
Smith, Carmichael, on the epidemic of influenza in 1782, 34
Streeten, on the epidemic of influenza in 1837, 34
Sydenham, on the epidemic of influenza in 1675, 34
Tigri, on the state of the pharynx in influenza, 41
Vigla, on the blood in influenza, 43
Watson, Sir Thomas, on contagion in influenza, 38
Webster, Noah, on the course taken by epidemics of influenza, 35; on the influence of telluric emanations on influenza, 36
Whytt, on the epidemic of influenza in 1758, 34
Williams, Robert, on the course taken by the epidemic of influenza in 1762, 35
Willis, Dr., on the epidemic of influenza in 1658, 34

INSANITY, Article on, by Henry Maudsley, M.D., F.R.C.P., p. 584.

AUTHORS REFERRED TO.

Boyd, Dr., Vital Statistics (*Journal of Mental Science*, Jan. 1865), 618
Bucknill and Tuke, Drs., Manual of Psychological Medicine, 604, 608
Clouston, Dr. T. S., on Tuberculosis and Insanity (*Journal of Mental Science*, April, 1863), 590
Esquirol, Traité des Maladies Mentales, 587, 589
Griesinger, Dr. W., die Pathologie und Therapie der psychiscken Krankheiten, 586, 590, 591
Guislain, Traité sur l'Aliénation Mentale, 589, 595
Haslam, Dr. J., the Sound Mind, &c., 587
Leidesdorf, Dr. M., die Pathologie und Therapie der psychischen Krankheiten, 586, 605
Moreau, Dr. J., Psychologie Morbide, 588
Morel, Dr. B., Traité des Degénérescences physiques, intellectuelles et morales, 586, 604
Muguier, Dr. E., de la Folie consécutive aux Maladies Aigues, 591
Parchappe, Traité de la Folie, 589
Pinel, on Insanity, translated by Davis, 596
Pritchard, Dr. J. C., a Treatise on Insanity, &c., 596, 601
Schroeder van der Kolk, die Pathologie und Therapie der Geisteskrankheiten, 588, 590
Skae, Dr. D., on Rational Classification of Insanity, 586, 600
Solomon, Dr. E., on the Pathology of General Paresis, in *Journal of Mental Science*, Oct. 1862, 614
Wilks, Dr. S., on Atrophy of the Brain, in *Journal of Mental Science*, Oct. 1864, 603, 614

INTRODUCTION, by J. Russell Reynolds, M.D., F.R.S., p. 17.

LOCAL PARALYSIS FROM NERVE DISEASE, Article on, by J. Warburton Begbie, M.D., F.R.C.P.E., &c., p. 1048.

AUTHORS REFERRED TO.

Bell, Sir Charles, on the Nervous System, 1051
Graves, Clinical Lectures, 1048, 1051
Romberg, on Nervous Diseases, 1051
Sanders, W. R., paper on Facial Paralysis, in *Lancet*, 1865, 1052
Todd, Clinical Lectures on Paralysis, &c., 1049, 1050, 1052
Watson, Sir Thomas, Practice of Physic, 1053

LOCAL SPASMS, Article on, by J. Warburton Begbie, M.D., F.R.C.P.E., &c., p. 1055.

AUTHORS REFERRED TO.

Davine, on reflex spasms from worms (Traité des Entozoaires), 1056
Graves, Clinical Lectures, 1056, 1059
Hyde Salter, on Asthma and Bronchial Spasm, 1056
Laennec, on bronchial spasm (Traité de l'Auscultation), 1057
Romberg, on Nervous Diseases, 1055, 1058

MALARIAL FEVERS, Article on, by W. C. Maclean, M.D., p. 352.

AUTHORS REFERRED TO.

Aitken, Dr. W., on the diagnosis of intermittent fever, 358
Blair, Dr. David, on quinine in remittent fever, 370
Blane, Sir G., on ulceration of the stomach in the Walcheren fever, 358
Burton, on the treatment of intermittent fever, 358, 363
Casorati, on the effects of malaria, 354; on blood-letting in intermittent fever, 356; on the morbid anatomy of the stomach in intermittent fever, 358
Cotton, Major-General, on the effects of malaria, 354
Davy, Dr., on quinine in remittent fever, 370
Haldane, Dr. R., on bleeding in intermittent fever, 360
Jones, Dr., on the urine in intermittent fever, 358; in remittent fever, 367
Martin, Sir Ranald, on clothing after malarial fevers, 364; on scurvy complicating malarial fevers, 367
Morehead, Prof., on jaundice in remittent fever, 368; on displacement of the heart by enlarged spleen, 358
Parkes, Dr. E. A., on malaria, 352, 353; on the temperature in intermittent fevers, 357; on the urine in intermittent fever, 358, 362
Ringer, Dr. Sydney, on the temperature in intermittent fever, 357; on the urine in intermittent fever, 358, 362
Wunderlich, on the temperature in intermittent fever, 357

MEASLES, Article on, by Sydney Ringer, M.D., p. 106.

AUTHORS REFERRED TO.

Hare, Dr. C. J, on the resemblance between the rash of measles and syphilitic roseola, 113
Veit, Dr., on petechial eruption in measles, 108

(See also foot-note, p. 106.)

MENINGEAL HEMORRHAGE, AND ON ADVENTITIOUS PRODUCTS IN THE MENINGES, Article on, by J. Spence Ramskill, M.D., &c., p. 840.

AUTHORS REFERRED TO.

Abercrombie, on Diseases of the Brain, 840
Andral, Clinique Médicale, 841
Baillarger, du Siége de quelques Hémorrhagies des Méninges, 841
Cruveilhier, Anatomie Pathologique, 841, 844
Legendre, Recherches anatomo-pathologiques, &c., sur quelques Maladies de l'Enfance, 842
Louis, on fungus of the dura mater (in Mém. de l'Acad. de Chirurgie, 1774), 843

Menière, Anatomie Typographique, &c., 841
Prus, sur l'Apoplexie meningée (in Mém. de l'Acad. Roy. de Médecine), 841
Reynolds, J. R., Diagnosis of Diseases of the Brain, 842
Rostan, Recherches sur le Ramollissement du Cerveau, 841
Trousseau, Clinique Médicale, 843
Virchow, die Krankhaften Geschwülste, 841, 843

METALLIC TREMOR, Article on, by W. Rutherford Sanders, M.D., F.R.C.P., p. 801.

AUTHORS REFERRED TO.

Christison, on Mercurial Tremor (on Poisons), 802
Darwall, on Chronic Mercurial Poisoning, in Forbes' Cyclop. of Practical Medicine (1833), 801
De Haen, Ratio Medendi, 802, 803
Jussien, on the diseases affecting quicksilver miners (Mém. de l'Académie Roy. des Sciences, 1719), 802, 805
Mérat, Mémoire sur le Tremblement des Doreurs, 802
Ramazzini, de Morb. Artif., 801
Sauvages, on Tremor metallurgorum (Nosologia Methodica, 1768), 806
Tardieu, on the health of workers in mercury (Dict. d'Hygiène), 801, 803
Whitley, Sixth Report of the Medical Officer of the Privy Council, 1863, 801
For numerous other references, see footnotes, 801

MUSCULAR ANÆSTHESIA, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., p. 783.

AUTHORS REFERRED TO.

Radcliffe, Dr. C. B., on Locomotor Ataxy, 785
Topinard, de l'Ataxie Locomotrice, 784
Trousseau, article on Ataxie Locomotrice, in "Dictionnaire nouveau de Méd. et de Chir.," tome iii., 785

NEURALGIA, Article on, by Francis E. Anstie, M.D., F.R.C.P., &c., p. 1026.

AUTHORS REFERRED TO.

Beau, Traité des Névralgies (Arch. Gén. de Médecine, 1847), 1048
Brown-Séquard, on Phys. and Path. of the Nervous System, and Lectures in the *Lancet*, 1866, 1048
Griffin, W. and D., on the Functional Affections of the Spinal Cord, 1027, 1048
Handfield Jones, on Functional Nervous Disorders, 1029
Trousseau, Clinique Médicale, 1027, 1032
Valleix, Traité des Névralgies, 1027 *et seq.*
For additional references see list on page 1048

NEURITIS AND NEUROMA, Article on, by J. Warburton Begbie, M.D., F.R.C.P.E., &c., p. 1020.

AUTHORS REFERRED TO.

Beau, Traité des Névralgies (Archives Gén. de Médecine, 1847), 1021
Brown-Séquard, on Neuroma (Holmes' Surgery, vol. iii.), 1024
Garrod, on Gout and Rheumatic Gout, 1021
Hughes Bennett, Clinical Lectures on Medicine, 1024
Odier, Manuel de Médecine, 1022
Paget, on Surgical Pathology, 1022, 1024
Rokitansky, Pathological Anatomy (Syd. Society), 1020, 1024
Romberg, Manual of Nervous Diseases, 1021
Smith, R., on the Pathology, &c., of Neuroma, 1021
Valleix, Guide du Médecin Practicien, 1021
Wood, Practice of Medicine, 1021
And papers in Med.-Chir. Trans. of Edinburgh, &c.

PARALYSIS AGITANS, Article on, by W. Rutherford Sanders, M.D., F.R.C.P., &c., p. 718.

AUTHORS REFERRED TO.

Handfield Jones, Dr., on Functional Nervous Disorders, 730
Maclachlan, Dr., on the Diseases and Infirmities of Advanced Life, 723, 727
Marshall Hall, Dr., Diseases and Derangements of the Nervous System, 723
Parkinson, Dr., Essay on the Shaking Palsy (1817), 719
Remak, on Galvano-Thérapie, 727
Romberg, on Nervous Diseases (Syd. Society), 722, 730
Trousseau, Clinique Médicale, 721
See also list on page 730, &c.

PAROTITIS, Article on, by Sydney Ringer, M.D., p. 118.

AUTHORS REFERRED TO.

Graves, Dr., on treatment of parotitis, 121
Jeaffreson, Dr. H., on parotid bubo, 120

PLAGUE, Article on, by Gavin Milroy, M.D., p. 314.

AUTHORS REFERRED TO.

Bartoletti, on the relation of famine to the plague, 319
Brayer, Dr., on the state of the atmosphere during the plague, 319
Bulard, on the morbid anatomy of the plague, 316
Clot-Bey, on the duration of the plague, 315; on the morbid anatomy of the plague, 316
Heberden, on the diagnosis of the plague, 319
Hennen, Dr., on the state of the atmosphere during the plague, 319
Morehead, Dr., on the Pali plague, 318
Sydenham, on the plague of London, 319
Volney, on the effects of seasons on the plague, 319

PURPURA, Article on, by Thomas Hillier, M.D., Revised by Tilbury Fox, M.D., p. 460.

AUTHORS REFERRED TO.

Barthez and Rilliet, MM., on a peculiar case of purpura, 463
Bateman, on purpura senilis, 461
Behrend, on bark in purpura, 468
Dickinson, Dr., on meningeal apoplexy in purpura, 464
Fox, Dr. Wilson, on the morbid anatomy of a case of purpura, 464, 466
Frerichs, on the pathology of purpura, 466
Graves, Dr., on "exanthema hæmorrhagicum," 462
Habershon, Dr., on arsenic in purpura, 468; on the spleen in purpura, 464
Hardy, Dr., on larch bark in purpura, 468
Hebra, on "purpura papulosa," 463; on the capillaries in purpura, 466
Hunt, Mr., on arsenic in purpura, 468
Jeller, on bark in purpura, 468
Lingen, Dr., on "Irish purpuric disease," 462
Neligan, Dr., on turpentine in purpura, 467
Ogle, Dr., on purpura in heart disease, 461
Ollivier, M., on purpura urticans, 463
Parkes, Dr. E. A., on the blood in purpura, 465
Pize, M., on sesquichloride of iron in purpura, 467
Rontier, on the blood in purpura, 465
Simon, Mr., on the blood in purpura, 465
Virchow, on the changes in the small vessels in amyloid degeneration, 466
Werlhoff, on bark in purpura, 468
Zenker, on the state of the muscles in purpura, 464

PYÆMIA, Article on, by J. Syer Bristowe, M.D., p. 330.

AUTHORS REFERRED TO.

Arnott, Mr., on the cause of pyæmia, 339; on suppuration of the eyeball in pyæmia, 334
Budd, Dr. G., on hepatic abscess in dysentery, 342
Cruveilhier, on the effects of injection of pus, mercury, &c., into the veins, 336
Frerichs, on abscess of the liver in dysentery, 342; on the cause of jaundice in pyæmia, 342
Jones, Dr. Wharton, on the effects of obstruction of an artery in the frog's foot, 336

Kirkes, Dr., on embolism in heart disease, 337
Lee, Mr. Henry, on the effects of injection of pus, mercury, &c., into the veins, 336; on a pustular eruption in pyæmia, 345
Polli, Prof., on the use of the sulphites in pyæmia, 351
Sédillot, on the effects of injecting pus, mercury, &c., into the veins, 336; on pus in the blood in pyæmia, 340
Simon, Mr., on the temperature in pyæmia, 345
Virchow, on the cause of pyæmia, 340; on embolism in heart disease, 337
Wilks, Dr. Samuel, on suppuration of joints in pyæmia, 342; on pustular eruption in pyæmia, 345

RELAPSING FEVER, Article on, by J. Warburton Begbie, M.D., p. 269.

AUTHORS REFERRED TO.

Alison, Dr., on the epidemic of relapsing fever in 1843, 272
Anderson, Dr., on the treatment of ophthalmia in relapsing fever, 280
Barker, Dr., on the epidemic of relapsing fever in 1817, 271
Bateman, on the epidemic of relapsing fever in 1817, 271
Cheyne, Dr., on relapsing fever in Ireland, 271
Christison, Dr., on the relation between typhus and relapsing fever, 271
Cormack, Dr., on contagion in relapsing fever, 275; on the symptoms of relapsing fever, 277
Corrigan, Dr., on famine as a cause of relapsing fever, 276
Craigie, Dr., on contagion in relapsing fever, 274
Flint, Dr. A., on relapsing fever in America, 273
Gairdner, Dr. W. T., on the periodical occurrence of relapsing fever, 270
Henderson, Dr., on the pulse in relapsing fever, 277
Huxham, on relapses in fever, 271
Jenner, Sir William, on recurrence of relapsing fever, 275; on jaundice in relapsing fever, 278
Lind, Dr., on relapses in typhus, 270
Lyons, Dr., on relapsing fever in the Irish famine, 272; on relapsing fever in the army of the Crimea, 273
Mackenzie, Dr., on ophthalmia in relapsing fever, 279
Murchison, Dr. C., on the epidemic of relapsing fever in 1851, 270
O'Brien, Dr., on the epidemic of relapsing fever in 1826–27, 272
Ormerod, Dr., on miliaria in relapsing fever, 278
Rutty, Dr., on the epidemic of relapsing fever in 1741, 271
Spittal, Dr. R., on the mention of relapsing fever by Hippocrates, 270
Stokes, Dr., on famine as a cause of relapsing fever, 276
Strother, Dr. E., on relapses in fever, 270
Virchow, on contagion in relapsing fever, 274
Wardale, Dr., on contagion in relapsing fever, 274
Welsh, Dr. B., on the epidemic of relapsing fever in 1817, 271; on contagion in relapsing fever, 274; on symptoms of relapsing fever, 278; on blood-letting in relapsing fever, 274

RHEUMATISM, Article on, by A. B. Garrod, M.D., F.R.S., p. 458.

AUTHORS REFERRED TO.

Aitken, Dr. W., on the mortality from rheumatism, 567
Andral, on the analysis of the blood in rheumatism, 562
Baillie, Dr., on cardiac disease in rheumatism, 563
Baillon, M., inventor of the word "rheumatism," 559
Bashan, Dr., on nitre in rheumatism, 569, 572
Bouillaud, M., on cardiac disease in rheumatism, 563; on venesection in acute rheumatism, 568
Briquet, on quinine in rheumatism, 569
Brocklesbury, Dr., on salines in rheumatism, 569
Brodie, Sir Benjamin, on the separation of rheumatism from rheumatoid arthritis, 559
Chomel, M., confusion between gout and rheumatism by, 559; on the morbid anatomy of rheumatic joints, 564
Ciraud, Dr., on lemon-juice in rheumatism, 570
Cullen, definition of rheumatism by, 559; on the pathology of rheumatism, 566; on venesection in acute rheumatism, 567
Davis, Dr. Herbert, on blisters in rheumatism, 572
Dechilly, Dr., on blisters in rheumatism, 572
Dickinson, Dr., on the frequency of cardiac disease in rheumatism, 563
Dundas, Sir D., on cardiac affections in rheumatism, 563
Fordyce, on blood-letting in rheumatism, 568
Fothergill, Dr., on bark in rheumatism, 569
Fuller, Dr., on hereditary predisposition to rheumatism, 564; on morbid anatomy of

rheumatism, 564; on potash in the treatment of rheumatism, 569, 572
Gavarret, on analysis of blood in rheumatism, 562
Geudinn, M., on nitre in rheumatism, 569
Haller, on the blood in rheumatism, 562
Haygarth, Dr., on the influence of age and sex on rheumatism, 564; on quinine in rheumatism, 569
eberden, on the influence of age on rheumatism, 564
Hulse, Dr., on bark in rheumatism, 569
Macleod, Dr., on the frequency of cardiac disease in rheumatism, 563
Martin-Solon, M., on nitre in rheumatism, 569; on blisters in rheumatism, 572
Monneret, on quinine in rheumatism, 569
Morton, Dr., on bark in rheumatism, 569
Parkes, Dr., on the urine in rheumatism, 562, 563
Perkins, on lemon-juice in rheumatism, 570
Pitcairn, Dr., on cardiac affections in rheumatism, 563
Prout, Dr., on lactic acid in the blood in rheumatism, 566
Rees, Dr. G. O., on the natural duration of rheumatism under expectant treatment, 567; on lemon-juice in rheumatism, 570
Richardson, Dr. W. B., on injection of lactic acid into the peritoneal cavities of dogs, 566
Ringer, Dr. Sydney, on the temperature in rheumatism, 561
Scudamore, Sir C., on the pathology of rheumatism, 566
Sydenham, on the distinction of gout from rheumatism, 559; on venesection in acute rheumatism, 567
Tulloch, Sir A., on the influence of climate on rheumatism, 565
Vinet, on quinine in rheumatism, 569
Wells, Dr., on cardiac disease in rheumatism, 563
Willan, Dr., on blood-letting in rheumatism, 568
Wright, Dr., on potash in rheumatism, 569

RHEUMATOID ARTHRITIS, Article on, by Alfred Baring Garrod, M.D., F.R.S., p. 550.

AUTHORS REFERRED TO.

Aitken, Dr., on the supposed existence of a hybrid disease between gout and rheumatism, 554
Begbie, Dr. W., on "digitorum nodi," 552
Craigie, Dr., on the supposed existence of "rheumatic gout" as a hybrid disease, 555
Fuller, Dr., on the supposed existence of "rheumatic gout" as a hybrid disease, 555
Haygarth, on the diagnosis of rheumatoid arthritis from gout, 555
Heberden, on "digitorum nodi," 552
Trousseau, M., on rheumatoid arthritis in women, 554
Wells, Mr. Spencer, on the supposed existence of "rheumatic gout" as a hybrid disease, 555
Wood, Dr., on the supposed existence of "rheumatic gout" as a hybrid disease, 555

RICKETS, Article on, by W. Aitken, M.D., p. 472.

AUTHORS REFERRED TO.

Ancel, on tubercle associated with rickets, 485
Beneke, on oxalic acid in the blood as the cause of rickets, 487
Bordenave, on congenital syphilis, its relation to rickets, 475
Bouchut, on cod-liver oil in rickets, 496
Copland, Dr., on the age at which rickets commences, 475; on cod-liver oil in rickets, 496
Cumin, Dr. W., on the age at which rickets commences, 475; on rickets in animals, 474
Dunglinson, Dr., on the age at which rickets commences, 475
Easton, Dr., on a formula for combining nux vomica and the phosphates of iron and quinine, 496
Friedleben, on gluten in rickety bones, 491
Good, Mr. Mason, on the age at which rickets appears, 475
Guérin, on artificial production of rickets in animals, 474; on the age at which rickets commences, 475
Herring, on the hereditary nature of rickets, 473
Jenner, Sir William, on rickets, *see* Rickets, 473, 474, 490
Kölliker, on the structure of bone in rickets, 488
Küttner, on intermarriage as a cause of rickets, 473
Lonsdale, Mr., on improper food as a cause of rickets, 474
Marchand, on excess of lactic acid in the blood as a cause of rickets, 487
Meric, on hypertrophy of the brain in rickets, 483
Meyer, on intra-uterine rickets, 475; on the structure of bone in rickets, 487
Neumann, on acute hydrocephalus in rickets, 483
Parkes, Dr. E. A., on the urine in rickets, 484
Pinel, on rickets in the fœtus, 475
Portal, on acute hydrocephalus in rickets, 483
Rokitansky, on the structure of bone in rickets, 488

Schlonberger, on gluten in rickety bones, 491
Schmidt, on oxalic acid free in the blood as a cause of rickets, 487
Schönlein, on early marriages as a cause of rickets, 473
Shaw, Mr., on arrest of growth of bones in rickets, 493
Simon, Mr., on the absence of gelatine in rickety bones, 491
Solly, Mr., on the phosphates in the urine of rickets, 484
Stanley, on the bones in rickets, 485
Stiebel, on the general tenderness in rickets, 477
Trousseau, M., on want of suckling as a cause of rickets, 474
Ure, on oxalic acid free in the blood as a cause of rickets, 487
Virchow, on the structure of bone in rickets, 488
Vogel, on the connection between syphilis and rickets, 474
Weatherhead, on phosphoric acid in the blood as a cause of rickets, 487
Wiltshire, Dr., on softness of the cranial bones in rickets, 477; on hypertrophy of the brain in rickets, 483

ROSEOLA, Article on, by Hermann Beigel, M.D., p. 104.

AUTHORS REFERRED TO.

Bateman, on the varieties of roseola, 105
Rayer, on the varieties of roseola, 105
Willan, on the varieties of roseola, 105
Wilson, Mr. Erasmus, on the varieties of roseola, 105

[RÖTHELN, Article on, by Henry Hartshorne, A.M., M.D., p. 117.]

SCARLET FEVER, Article on, by S. J. Gee, M.D. Lond., p. 83.

AUTHORS REFERRED TO.

Armstrong, on ulceration of the throat in scarlet fever, 86; on the pyrexia in scarlet fever, 86
Barthez and Rilliet, MM., on the prognosis of scarlet fever, 95
Begbie, Dr. W., on albuminuria in scarlet fever, 91
Fenwick, on casts of the gastric tubuli in vomit of scarlet fever, 94
Graves, Dr., on diphtheria as a sequel of scarlet fever, 93
Heberden, on sloughing in scarlet fever, 86
Huxham, on sloughing in scarlet fever, 86
Ringer, Dr. S., on the temperature in scarlet fever, 86
Sydenham, on the relation of seasons to scarlet fever, 84
Trousseau, M., on diphtheria as a sequel of scarlet fever, 93; on the incubative period of scarlet fever, 84
Valleix, on coma in scarlet fever, 85; on duration of scarlet fever, 88
Watson, Sir Thomas, on contagion in scarlet fever, 84
Zenker, on changes in the muscular tissue in scarlatina, 94

SCORBUTUS, Article on, by Thomas Buzzard, M. D., p. 445.

AUTHORS REFERRED TO.

Becquerel and Rodier, MM., on the blood in scurvy, 455
Bird, Dr. G., on scurvy among the poor of London, 449; on chemosis in scurvy, 451
Boyd, Dr., on cider as an antiscorbutic, 459
Bryson, Dr., on nyctalopia in scurvy, 453
Budd, Dr., on history of scurvy, 446; on causes of scurvy, 449
Christison, Dr., on an outbreak of scurvy in Scotland, 446
Curran, Dr., on scurvy in Ireland, 446
Darby, Dr., on scurvy in the Confederate armies. 448
Garrod, Dr., his theory of scurvy, 455
Grant, Dr. J. O., on scurvy occurring in men fed on food salted with nitrate of potash, 456
Hammond, Dr., on scurvy in the American armies, 448
Haspel, M., on the state of the lungs in scurvy, 457
Himmelstiern, Dr., on the effusions in scurvy, 457
Joinville, le Sieur, on scurvy in the army of Louis IX. in Egypt, 449
Kane, Dr., on the antiscorbutic properties of walrus meat, 450
Lawson, Mr., on the affections of the eye in scurvy, 453
Leach, Mr. Harry, on syncope in scurvy, 452
Lind, Dr. James, on lime-juice in scurvy, 448
Lonsdale, Dr., on scurvy at Carlisle, 446
Maclean, Professor, on Bael fruit in scurvy, 458
Marrion, Dr., on preserved vegetables in scurvy, 458
Parkes, Dr. E. A., on the prevention of scurvy, 458
Ritchie, Dr., on an outbreak of scurvy in Scotland, 446
Shapter, Dr., on scurvy in Ireland, 446
Wells, Mr. Soelberg, on the affections of the eye in scurvy, 453
Whymper, Mr., on the antiscorbutic properties of seal-meat, 450

[SCROFULA, Article on, by Henry Hartshorne, A.M., M.D., p. 497.]

SIMPLE MENINGITIS, Article on, by J. Spence Ramskill, M.D., &c., p. 808.

AUTHORS REFERRED TO.

Abercrombie, Dr., on Diseases of the Brain, 811, 814
Andral, Clinique Médicale, 814
Blaud, Dr., on treatment of meningitis by compression of the carotids (Bibliothèque Méd., tome lxii.), 815
Fuller, Dr., on rheumatic meningitis (Rheumatism), 811
Guersant, art. Méningite, in Dict. de Médecine, 814
Maclachlan, on the Diseases and Infirmities of Advanced Life, 810
Parent-Duchâtelet et Martinet, de l'Arachnitis, 811
Rilliet et Barthez, on Diseases of Children, 814
Robin, on Syphilitic Meningitis, 812
Trousseau, on Cerebral Fever (Clinique Médicale), 808, 811
Valleix, Guide du Médecin Practicien, 815
Watson, Sir Thomas, Practice of Physic, 811, 812

SMALLPOX, Article on, by J. F. Marson, p. 127.

AUTHORS REFERRED TO.

Aitken, Dr., on the prevention of pitting in smallpox, 144
Bulkley, Dr., on the mortality in dark-skinned races from smallpox, 138
Ceely, Mr., on inoculation of the cow with human smallpox, 154; on inoculation of sheep-pox on the human subject, 128
Graves, Dr., on the prevention of pitting in smallpox, 144
Gregory, Dr., on the history of smallpox, 128; on inoculation for smallpox, 156; on smallpox pustules in the intestines, 146; on the treatment of smallpox, 140
Hedlund, M., on variola sine eruptione, 140
Higginbottom, Mr., on the prevention of pitting in smallpox, 144
Hunter, John, on the anatomical characters of a variolous pock, 147
Jenner, Edward, introduction of vaccination by, 149
Mead, on smallpox before birth, 132
Montague, Lady Mary Wortley, on inoculation for smallpox, 156
Moore, Dr., on the history of smallpox, 128
Petzholt, Dr., on smallpox pustules in the intestine, 146
Procopius, the earliest account of smallpox by, 128
Rhazes, description of smallpox by, in 910, 128
Simon, Dr. Gustav, on the anatomical characters of a variolous pock, 148
Simon, John, on the prevention of smallpox by vaccination, 156
Sydenham, on the treatment of smallpox, 141; on variola sine eruptione, 139
Thompson, Dr. John, on varioloid, 150
Van Swieten, on variola verrucosa or cornea, 132
Velpeau, M., on the prevention of pitting in smallpox, 144
Watson, Sir Thomas, on smallpox pustules in the intestine, 146; on the anatomy of smallpox pustules, 147
Wedl, Carl, M.D., on the anatomy of smallpox pustules, 149
Wilson, Erasmus, on the anatomical characters of the variolous pock, 147

SOFTENING OF THE BRAIN, Article on, by J. Russell Reynolds, M.D., F.R.S., and H. Charlton Bastian, M.D., F.R.S., p. 856

AUTHORS REFERRED TO.

Abercrombie, on Diseases of the Brain and Spinal Cord, 866
Andral, Clinique Médicale, 866
Bennett, Hughes, Cl nical Lectures, 868, 871
Bouillard, Traité de l'Encéphalite, 861, 866
Broca, sur le Siége de la Faculté du Langage articulé (Bullet. de la Soc. Anat. 1861), 861
Cruveilhier, Anat. Pathologique, 866
Durand-Fardel, Maladies des Vieillards, 866
Gluge, Comptes Rendus, 1837, &c., 867
Hughlings Jackson, on Loss of Speech (London Hospital Reports), 861, 867
Kirkes, on Cerebral Embolism, in Med.-Chir. Transactions, vol. xxxv., 866, 869
Laborde, Ramollissement et Congestion du Cerveau, 867, 872
Lallemand, Recherches anat.-path. sur l'Encéphalite, 865, 872
Lancereaux, de la Thromb. et de l'Emb. Céréb., 866, 869

Prevost and Cotard, Recherches sur le Ramollissement Cérébrale (Gaz. Méd. de Paris, 1866), 867, 868
Rokitansky, Pathol. Anat. (Syd. Society), 867
Rostan, Ramollissement du Cerveau, 867
Todd, Clinical Lectures on Paralysis, 860, 871
Trousseau, on Aphasia (Gaz. des Hôpitaux, 1864), 861
For numerous additional references, to Cohn, Proust, Virchow, Van der Kolk, and other well-known authors, see foot-notes at 866, 871, &c.

SOMNAMBULISM AND ITS ALLIED STATES, Articles on, by Thomas King Chambers, M.D., F.R.C.P., &c., p. 658.

AUTHORS REFERRED TO.

Abercrombie, Dr., on the Intellectual Powers, 660
Trousseau, Clinique Médicale, 660
Transactions of the Royal Society of Edinburgh, vol. ix. &c.

DISEASES OF THE SPINAL CORD, Articles on, by C. B. Radcliffe, M.D., F.R.C.P., &c., p. 942.

AUTHORS REFERRED TO.

Adams, W., on Infantile Paralysis, 1004, 1005
Barthez and Rilliet, on Infantile Paralysis, 1005
Bell, Sir Charles, on the Nervous System, 942
Briquet, on hysterical paraplegia, &c. (Traité Clinique, &c. de l'Hysterie), 994, 1000
Brodie, Sir Benjamin, on injuries of the spinal cord (Méd.-Chir. Trans. 1837), 945
Brown-Séquard, on Phys. and Path. of the Nervous System, 943, 959
Curling, on Tetanus, 971, 975
Duchenne, on Locomotor Ataxy, &c., 981, 999
Griffin, W. and D., on Functional Affections of the Spinal Cord, 991
Lockhart Clarke, on the pathology of locomotor ataxy (Med.-Chir. Trans. 1865), 942, 977
Marshall Hall, Diseases of the Nervous System, 943, 961
Ollivier, on Diseases of the Spinal Cord, 953, 959
Romberg, Manual of Nervous Diseases (Tabes Dorsalis), 980
Teale, on Neuralgic Diseases dependent on Irritation of the Spinal Marrow, 991, 995
Todd, on Nervous Diseases, 961, 980
Trousseau, Clinique Médicale, 984, 985
Watson, Sir Thomas, Practice of Physic, 976, 979

SUDAMINA AND MILIARIA, Article on, by Sydney Ringer, M.D., p. 122.

SUNSTROKE, Article on, by W. C. Maclean, M.D., &c., p. 661.

AUTHORS REFERRED TO.

Barclay, Dr., Natural History of Insolation, 663
Bassier, M., Dissertation sur la Calenture, 664
Boudin, M., Statistiques Médicales, 663, 664.
Ludwig, on the effect of elevation of temperature on the body (Handbuch der Physiologie), 665
Martin, Sir Ranald, on the Influence of Tropical Climate, &c., 662
Morehead, Dr., Clinical Researches on Diseases in India, 666
Obernier, Dr., experiments on the effects of high temperatures on the body, 665
Parkes, Dr., on Practical Hygiene, 663
Papers in Indian Annals of Medicine, &c.

SYPHILIS, Article on, by Jonathan Hutchinson, F.R.C.S., p. 423.

AUTHORS REFERRED TO.

Carmichael, on the nature of syphilitic poison, 423
Curling, on syphilitic orchitis, 434
Diday, M., on recurrence of syphilis, 427
Lee, Mr., on hereditary immunity from syphilis, 428; on calomel vapor baths in syphilis, 438
Paget, Mr. James, on syphilitic ulceration of the rectum, 430
Parker, Mr. Langstone, on calomel vapor baths in syphilis, 438
Wilks, Dr. Samuel, on syphilitic disease of the liver, 434

TORTICOLLIS, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., p. 1060.

AUTHORS REFERRED TO.

Brown-Séquard, Lectures in the *Lancet*, 1866, 1063

Romberg, Manual of Nervous Diseases (Syd. Society), 1060, 1062

TUBERCULAR MENINGITIS, Article on, by Samuel Jones Gee, M.D., F.R.C.P., &c., p. 817.

AUTHORS REFERRED TO.

Allbutt, Dr. C., paper on the diagnostic value of the ophthalmoscope (*Lancet*, 1868), and on Optic Neuritis (*Med. Times and Gazette*, 1868), 824, 825

Bastian, Dr. C., on perivascular sheaths in the brain, 834

Rilliet and Barthez, on Diseases of Children, 821, 825

Trousseau, Clinique Médicale, 821, 822

Virchow's Jahresbericht (1869), on tubercle in the choroid, 824

Whytt, Dr. R., on Hydrocephalus Internus (1768), 820

TYPHOID OR ENTERIC FEVER, Article on, by John Harley, M.D. Lond., p. 201.

AUTHORS REFERRED TO.

Boudin, M., on the relation of malaria to specific fevers, 231

Bretonneau, on contagion in typhoid fever, 238

Buchanan, Dr. G., on impure water as a cause of typhoid fever, 242

Budd, Dr. William, on contagion in typhoid fever, 238; on impure water as a cause of typhoid fever, 242

Chomel, on the diagnosis of typhoid fever, 233

Davis, Mr., on the Walcheren fever, 228, 229

De Claubry, on the distribution of typhoid fever, 234; on the spontaneous origin of typhoid fever, 240

Ebel, Dr., on an outbreak of typhoid fever at Stangerod, 242

Forget, M., on the blood in typhoid fever, 218

Gairdner, Dr. W. T., on the post-mortem appearances in cholera, 231

Galtier, C. P., on poisoning by poisonous mushrooms, 243

Grossheim, Dr., on the cause of typhoid fever, 238

Guy, Dr., on the health of night-men, 241

Jenner, Sir Willian, on the distinction between typhus and typhoid fevers, 245; on pleurisy in typhoid fever, 222; on the state of the liver in typhoid fever, 216

Kerner, Dr., on poisoning by putrid food, 243

Latham, P. M., M.D., on the outbreak of scurvy at Milbank Penitentiary, 231

Louis, on the causes of typhoid fever, 238; cases of typhoid fever reported by, 226; on the state of the liver in typhoid fever, 216; on the state of the lungs in typhoid fever, 221; on pleurisy in typhoid fever, 222

Mayo, Mr. C., on the camp fever in the army of the Potomac, 228

Moffat, Dr., on the effect of absence of ozone from the air, 238

Murchison, Dr., on perforation in typhoid, 208; on sewer emanations as a cause of typhoid, 241; on the state of the liver in typhoid, 216; on pleurisy in typhoid fever, 222

Parkes, Dr. E. A., on the stools in typhoid fever, 207

Piedvache, M., on the spontaneous origin of typhoid, 240

Pirogoff, on the post-mortem appearances of the intestines in cholera, 231

Schumann, Dr., on poisoning by putrid food, 243

Simon, J., on water as a cause of typhoid fever, 242

Stewart, Dr. A. P., on the distinctions between typhus and typhoid, 245

Trousseau, M., on nitrate of silver in typhoid fever, 248; on the blood in typhoid fever, 218

Tweedie, Dr., on nitrate of silver in typhoid fever, 248

Whitley, on the relation of malaria to typhoid fever, 228

Zenker, on the degeneration of muscular tissue in typhoid fever, 217

TYPHUS FEVER, Article on, by George Buchanan, M.D., p. 251.

AUTHORS REFERRED TO.

Murchison, Dr., on the spontaneous origin of typhus, 254
Parkes, Dr., on the urine in typhus, 260

VACCINATION, Article on, by Edward Cator Seaton, M.D., p. 158.

AUTHORS REFERRED TO.

Balfour, Dr., on the amount of smallpox in the army, 167
Ceely, Mr., on inoculation of cows with human smallpox, 177; on vaccination with lymph direct from the cow, 160
Cross, Mr., on the protective power of vaccination, 167
Cullerier, M., on the effects of using lymph from a syphilitic child, 179
Heim, Professor, on the influence of time on the protective power of vaccination, 174; on revaccination, 175
Jenner, Edward, introduction of vaccination by, 158; on the degeneration of vaccine lymph, 172
Jenner, Sir W., on the communication of other diseases by vaccination, 178
Kinnis, J., M.D., on the effects of vaccination on smallpox, 169
Marshall, Mr., on the protective power of vaccination, 167
Marston, Mr., on failures in vaccination, 165; on revaccination, 175
Pachiotti, on the outbreak of syphilis at Rivalta, 181
Paget, Mr., on the production of cutaneous eruptions by vaccination, 178; on the communication of syphilis by vaccination, 178
Sperino, on the outbreak of syphilis at Rivalta, 181
Taupin, M., on experiments with lymph from a syphilitic child, 179
West, Dr. C., on the communication of syphilis by vaccination, 178

VARICELLA, Article on, by Samuel Jones Gee, M.D., p. 124.

AUTHORS REFERRED TO.

Abercrombie, on the relation of varicella to cow-pox, 125
Bryce, on the eruption in varicella, 126
Cross, on the diagnosis of varicella, 126
Fuller, on the non-identity of varicella and variola, 124
Gregory, on the symptoms of varicella, 126
Heberden, on the non-identity of varicella and variola, 124
Trousseau, on inoculation of varicella, 124, 125

VERTIGO, Article on, by J. Spence Ramskill, M.D., &c., p. 690.

AUTHORS REFERRED TO.

Brown-Séquard, Dr., Physiology of the Nervous System, 694
Menière, Dr., on Vertigo from Diseases of the Ear, in Bulletin de l'Académie de Médecine, vol. xxvi., 694
Trousseau, Clinique Médicale, 694

WASTING PALSY, Article on, by William Roberts, M.D., F.R.C.P., p. 786.

AUTHORS REFERRED TO.

Aran, descripton of Wasting Palsy, 793
Bergmann, papers in *St. Petersburger Medicinische Zeitschrift*, 1864, 787, 791
Cruveilhier, on Progressive Muscular Atrophy, in Archives Générales, 1853, 791
Duchenne, Dr., de l'Electrisation Localisée, &c., 786, 798
Gull, Sir W., on the pathology of wasting palsy (Guy's Hospital Reports, series iii.), 792
Lockhart Clarke, on microscopic appearances of the spinal cord in wasting palsy (Beale's Archives, 1861), 787, 792

WRITER'S CRAMP, Article on, by J. Russell Reynolds, M.D., F.R.S., &c., p. 732.

AUTHORS REFERRED TO.

Brown-Séquard, Physiology and Pathology of the Central Nervous System, 735
Lockhart Clarke, on Locomotor Ataxy, 737
Solly, on Scrivener's Palsy (*Lancet*, January 1865), 734

YELLOW FEVER, Article on, by J. Denis Macdonald, M.D., F.R.S., p. 281.

AUTHORS REFERRED TO.

Aitken, Dr. W., on the treatment of yellow fever, 294
Blair, Dr., on the blood in yellow fever, 288; on black vomit, 290
Blane, Sir G., on the yellow tint of skin in yellow fever, 287
Bryson, Dr., on infection in yellow fever, 288
Buchanan, Dr. G., on the yellow fever at Swansea, 295
Cartwright, Dr., on the state of the semilunar ganglia in yellow fever, 291
Chassanoil, on urea in the blood in yellow fever, 289
Davy, Dr., on the blood in yellow fever, 288
Frost, Professor, on chlorate of potash in yellow fever, 294
Humboldt, on the effect of elevation on yellow fever, 284
Jackson, Dr., on the prognosis of yellow fever, 292
La Roche, Dr., on the symptoms of yellow fever, 285; on the varieties of yellow fever, 295; on the urine in yellow fever, 290
Maclean, Dr., on the diagnosis of yellow fever, 286
Mason, Dr. R. D., on contagion in yellow fever, 296
Pennel, Dr., on the morbid anatomy of yellow fever, 291, 292
Rogers, Professor, on the excess of salts in the blood in yellow fever, 288; on the composition of the black vomit, 289
Warren, on the cause of the yellow tint of the skin in yellow fever, 287

END OF VOLUME I.

6.G.77.
A System of medicine 1880
Countway Library
AIO3194
3 2044 045 116 472

6.G.77.
A System of medicine 1880
Countway Library
AIO3194
3 2044 045 116 472